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Anosognosia in Older People with Early-Stage Alzheimer's Disease

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Thesis Abstract

A large body of literature has suggested people with Alzheimer's Disease (AD) are particularly prone to experiencing impairments in insight. This poses significant implications for our understanding of AD, the mechanisms underlying self-awareness and the clinical management of such individuals.

The first paper of this thesis explores the literature in this field and discusses the tentative findings to date, together with the methodological difficulties associated with studying impaired insight. It goes on critically to explore current models of this phenomenon, with the clinical implications of these models considered in relation to the treatment of people with AD. The paper concludes that the impairments in insight exhibited by people with AD are heterogeneous and are likely to be best explained as representing a number of subtypes.

The second paper of this thesis describes a study that aimed to investigate Agnew and Morris's (1998) model of memory awareness, by exploring the dimensions of awareness exhibited by a sample of people in the early stages of AD. This study found that a significant proportion of people with early-stage AD were able to improve their awareness after exposure to memory tasks and that they were able to generalise this improvement from a task specific level to a global level of memory awareness. Furthermore, whilst this improvement was retained after a 20-minute delay period, there was evidence that with a longer delay this would have reset to pre-testing levels. Such findings are in keeping with Agnew and Morris's (1998) mnemonic anosognosia sub-type.

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Impaired Insight in People with Alzheimer's Disease

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Impaired Insight in People with Alzheimer's Disease

Abstract

Alzheimer's Disease (AD) typically results in a dementia characterised by significant cognitive dysfunction with prominent memory deficits, impairment of self-care abilities and disturbed mood and behaviour. However, despite these salient and debilitating symptoms, many people with AD are seemingly unaware of their deficits. This apparent impairment of awareness has been the focus of growing interest within the AD population, due to its implications for our knowledge of the progression of AD, the aetiological mechanisms underlying self-awareness and the psychological management of people with AD and their carers. However, whilst extensive research has been completed within this field, it has been prone to methodological difficulties and has produced variable and often contradictory findings. This has contributed to a prolonged debate within the literature as to the mechanisms underlying deficits in insight. Some authors have argued that the impairments in awareness are due to environmental contingencies; others that they are due to psychological defence mechanisms; whilst others still, have argued they are due to neurological damage. These arguments, and the evidence they draw upon, are discussed, together with their implications for treatment. Conclusions drawn from the limited empirical evidence, suggest that insight in AD is heterogeneous and is likely to be best understood via an integrated, multifactorial model. The author argues that identifying and distinguishing the sub-types of impaired awareness within this population, is imperative to the future clinical management of people with AD (and their carers).

Impaired Insight in People with Alzheimer's Disease

Introduction

Over recent years there has been a growing recognition that a disproportionately high number of people with Alzheimer's Disease (AD) suffer from impaired awareness of their symptoms. Such is the apparent commonality of a lack of insight amongst this diagnostic group, that some authors (e.g., DeBettignes, Mahurin & Pirozzolo, 1990; Mahendra, 1984) have gone so far as to say that it is a defining characteristic of the disorder. Empirical investigation of impaired insight within the AD population is likely to have important implications for our knowledge of the progression of AD, the cognitive systems underlying self-awareness and the psychological management of people with AD and their carers. This paper will explore the current difficulties in assessing insight, the research findings to date, and the theoretical models that have attempted to explain this phenomenon. The clinical implications of each model will also be discussed.

Alzheimer's Disease

AD is a form of progressive dementia caused by atrophy of the brain. Evidence suggests that the pathology involves a graded bilateral process, whereby medial temporal and hippocampus areas are affected early on, followed by frontal structures and latterly parietal and occipital areas (Albert, 1996; Casanova et al., 1993; Morrison et al., 1990; Nagy, 1996). It is the most common cause of dementia within Western populations and is marked by a gradual onset and progressive decline of cognitive skills, including memory, executive functioning, attention, language, and perception (Zec, 1993). Individuals with AD may also experience

functional limitations and a range of behavioural and psychiatric symptomatology such as depression, delusions and hallucinations (e.g., Mega, Cummings, Fiorella & Gornbein, 1996). Figures vary between studies, but some estimates suggest that it constitutes as much as 55% of dementia cases and has a higher incidence amongst females (Jorm, Korten and Henderson, 1987). Whilst some individuals develop this condition when they are as young as 40 years of age, the disease is most common in persons over the age of 65 years (Baker, Jordan, Barclay & Schoenberg, 1993).

Insight

Insight is notoriously difficult to define, with definitions varying greatly within the literature. Foley (1992) defined it as "the capacity to discern the true nature of the situation, or, as applied to dementia, the recognition of fact, degree and implications of one's own illness" (pp.37). Barco, Crosson, Bolesta, Werts and Strout (1991) recommended that insight be considered pathological when "individuals possess a level of awareness lower than the accepted norm and functioning in daily life becomes disturbed" (pp. 129). However, it remains contentious as to what constitutes the accepted norm.

The terms "lack of insight" and "impaired awareness" have largely been used within the psychiatric field to refer to a lack of knowledge of one's mental illness, usually psychosis (David, 1990), while the term "anosognosia" has been used within the neurological field to describe a failure to acknowledge a particular neurological deficit. This latter term was originally used by Babinski (1914) to describe an absence of awareness in a hemiplegic client, but it has since been used to describe a lack of insight in a number of other disorders, such as Korsakoff's

syndrome, Pick's disease and AD (e.g., McGlynn & Schacter, 1989; Zangwill, 1966). As a result, the term is now used more generally to include a lack of insight into the existence of physical, neurological or cognitive impairments (Schacter, 1990). Due to the similarities in the definitions of the terms anosognosia, impaired awareness and lack of insight, many authors have used the terms synonymously. For the purpose of this paper these terms will also be used interchangeably.

Insight within the AD population

Whilst the problems defining insight and the inconsistencies between studies have made it difficult to establish the exact prevalence rate of impaired awareness within the AD population, estimates have generally been high and have ranged from 20% (Migliorelli et al., 1995) to as high as 80% (Sevush & Leve, 1993). Although one study did identify a greater incidence of impaired awareness amongst females with AD (Migliorelli et al., 1995), most studies have failed to find a gender difference, or a correlation with other demographic variables, such as age of onset, education, or duration of the disease (Feher Mahurin, Inbody, Crook, & Pirozzolo, 1991; Vasterling, Seltzer, Foss & Vanderbrook, 1995). Furthermore, a number of studies have indicated that people with AD have significantly greater impairment of insight than individuals with multi-infarct dementia (Sultzer, Levin, Mahler, High, & Commings, 1992), Parkinson's disease (Danielczyk, 1993), or vascular dementia (Wagner & Crushman, 1994). This has led to speculation that a lack of insight constitutes an important diagnostic characteristic of AD. Although, it should be noted that studies have not been conclusive, with one study finding no significant difference between these groups (Verhey, Ponds, Rozandaal & Jolles, 1995).

However, this study can be criticised for solely using people in the very early stages of dementia, where the differentiation between dementias is likely to be less clear.

The level of insight has important implications for an individual's quality of life. In particular, impaired insight is likely to reduce the person's ability to function safely and independently, and increase the level of burden on their carers (Seltzer, Vasterling, Yoder & Thompson, 1997; Trudel, Tryon, & Purdum, 1998). People who are unaware of their limitations tend to choose activities beyond their capabilities and do not recognise when they need help. For example, Hunt, Morris, Edwards and Wilson (1993) found that individuals with mild dementia, who failed a standard driving assessment, had little awareness of their poor performance. Indeed, Bergman, Proctor and Prudham (1979) found that retained insight distinguished people with dementia who were able to live independently within the community from those who were hospital based.

In addition, insight is likely to have a significant influence on the clinical management of an individual. A person who does not recognise their functional difficulties is less likely to be motivated to undergo treatment (Fleming, Strong & Ashton, 1998). In the traumatic brain injury literature, awareness has been found to be related to attainment of rehabilitation goals (Prigatano & Wong, 1999), adherence to medication (Cramer, 1992) and employment outcome (Sherer et al., 1998). Similar findings have also been found in the AD field. For example, Koltai, Welsh-Bohmer, and Schmechel (2001) found that higher levels of awareness of memory difficulties were associated with greater gains from participation in a memory therapy group.

Finally, lack of insight presents significant legal implications regarding whether a person with AD has capacity to make decisions about their future financial and care needs, as well as, give informed consent to treatment and research. Wilber (2001) stated that legal provisions for people with dementia should be tailored to the individual differences in areas of capacity. However, impaired insight is likely to impinge on this. It is, therefore, important for professionals to have a good understanding of the boundaries of a person's insight (Mullen, Howard, David & Levy; 1996). If they do not achieve this, they risk either leaving the person in danger of harm or unnecessarily restricting their fundamental liberties and their sense of personhood (Kitwood, 1997). All of these factors emphasise the importance of gaining a better understanding of the characteristics of impaired insight within this population.

Assessment of insight

The lack of clear definition of what constitutes insight and, more specifically, impaired insight, has hindered the development of appropriate and validated assessment measures. As a result of the complexity of this issue a number of assessment measures have been used in the literature. Each of these measures has advantages and drawbacks, but all suffer from a lack of sound validation of their psychometric properties.

Clinician ratings

The clinician uses a scale to make an overall rating of the individual's level of awareness, based on questions and observations made during interviews with the person and their carer(s). This technique has been used in a number of studies, with Verhey, Rozendaal, Ponds and Jolles' (1993) criteria most commonly used (e.g., Allen & Killick, 1998; Mullen, et al., 1996). This criterion uses a 4-point rating scale, where the individual's awareness is rated as either "adequate" (i.e., the person adequately and spontaneously relates their impairments), "mildly disturbed" (i.e., mild gaps in awareness), "moderately disturbed" (i.e., vague knowledge of impairments), or "severely disturbed" (i.e., complete denial of impairments).

However, such measures have been criticised for presenting a subjective concept of awareness that is dependent on the clinician's view of what constitutes adequate awareness (Clare, 2001). Such measures do not take into account the social, contextual implications of a clinical interview, whereby the person may be influenced by a social pressure to present themselves in a positive way to an unfamiliar person (Malec, Machulda & Moessner, 1997). Indeed, Weinstein, Friedland and Wagner (1994) found some individuals denied problems in a clinical interview, but freely admitted to them in other contexts. Furthermore, clinician ratings can be criticised for only giving a fixed assessment of the individual's insight for global levels of functioning, thus limiting their validity (Clare, Wilson, Carter, Roth, & Hodges, 2002; Verhey et al., 1993). In addition, there is no standard format used for clinician ratings, with measures varying greatly in the adjectives used to define them (Mullen et al., 1996) and the number of categories in the scale. For example, some studies have used 4-point scales (e.g. Verhey et al., 1993), and

others have used 6-point scales (Sultzer et al., 1992). All these factors are, therefore, likely to affect the validity and reliability of such a measure, as well as the accuracy of the conclusions drawn from them.

Discrepancy measures

Researchers have attempted to overcome some of the problems presented by clinician ratings by using discrepancy measures to assess awareness. Two types of discrepancy measures have been reported in the literature to date.

Patient-carer discrepancy measures

The client and carer complete parallel rating scales regarding the client's difficulties. The discrepancy between the ratings is then calculated and taken as a measure of the client's awareness (e.g., Migliorelli et al., 1995; Vasterling, Seltzer & Watrous, 1997). If there is a discrepancy between the client and carer ratings, then the client is judged to lack awareness. The advantage of this method is that it can be geared to the awareness of abilities in specific domains. It is also believed that because the carer has the opportunity to view the client's behaviour over a number of situations, they can offer a broader view of the client's abilities than a clinician is able to.

Whilst investigations of the reliability of this technique have been encouraging (Feher et al., 1991; Green Goldstein, Sirockman, & Green, 1993), its validity has been more contentious. Such measures are based on the assumption that carers give an accurate picture of the client's abilities, while the client is prone to over-

estimates of their ability. However, whilst there is some evidence to support the validity of this assumption (e.g., Feher et al., 1991), a number of authors have not supported this view (e.g., Bucks, 1998; Hart, Giovannetti, Montgomery, & Schwartz, 1998; Mullen et al., 1996). In particular, Green and colleagues (1993) found no significant relationship between carer ratings of recent memory and the client's performance on a verbal learning test, leading them to doubt the validity of using carer ratings. Furthermore, whilst McGlynn and Kaszniak (1991) found carers were more accurate than clients at estimating the client's performance, they found carers still made considerable over and underestimates of their relative's abilities. Indeed, both the client's and the carer's judgements are likely to be influenced by contextual and personality factors. It is possible that if carers are suffering from carer burden they may be prone to underestimate the client's ability level (Mangone et al., 1991; Sohlberg, Mateer, Oenkman, Glang & Todis, 1998), or, if they are having difficulty coping with their relative's diagnosis, they may deny the client is having any difficulties (Derouesne et al, 1999). Similarly, the client may underestimate their abilities if they are depressed or prone to low self-esteem (Michon, Deweer, Pillon, Agrid & Dubois 1994). Furthermore, it is likely to be difficult for both the carer and the client to make concrete judgements about abilities, which, due to the nature of AD, are likely to vary over time. All of these factors are likely to affect the validity of such a measure.

Patient-performance discrepancy measures

Recently, researchers have applied experimental methods used to evaluate paradigms of metamemory to obtain a more objective measure of self-awareness (James & Moulin, 2002; Moulin, Perfect & Jones 2000; Toglia & Kirk, 2000). Such

methods involve asking the client to predict their performance on a test, before they complete it. A greater discrepancy between the client's prediction and their actual performance, indicates a greater impairment of awareness. However, studies using this one test approach have found that the correlation between participant rating and performance has been weak, even for the general population (Dixon, 1989). Evidence suggests that this may be attributed to the fact that people, when faced with a novel task as is often the case with such measures, tend to "hedge their bets" and rate their performance at the mid-point. It is only with the opportunity to monitor their performance that they alter their predictions to more accurate ones (Connor, Dunlosky, & Hertzog, 1997; Koss, Patterson, Ownby, Stuckey & Whitehouse, 1993). In order to overcome this problem, Moulin and associates (2000) proposed an updated version of this measure, in which participants were asked to predict their performance over a number of trials, with their degree of insight determined by the changes in the accuracy of their predictions.

However, even this updated approach is not without criticism. By solely assessing insight though changes in participants' predictions of their performance, it is difficult to establish whether awareness is at explicit or implicit levels, i.e., are the participants aware of why they are changing their ratings? Furthermore, this approach only looks at awareness for a specific task carried out in a clinical setting, which may not be relevant to the client's insight into their performance on "real life" tasks. Indeed, it cannot be assumed that the client is rating themselves on the same factors as those measured by the tests (Trosset & Kaszniak, 1996). Perhaps the only way to minimise these difficulties is to closely match the rating scales to the task (Larrabee, West & Crook, 1991).

Questionnaire measures

Sevush (1999) recently developed a standardised scale of awareness for memory impairments, which attempts to overcome the problems presented by previous measures, by using absolute scores rather than subjective discrepancy scores. The Awareness of Memory Impairment Scale (AMIS) was constructed from items characteristic of early-stage AD and consists of six questions about the participant's memory. The client is required to answer "Yes" or "No" to the questions to indicate whether they have that particular difficulty. Denial of any of the complaints is taken as an indication of impaired insight; the more characteristics denied, the more the person's insight is considered impaired. Whilst this questionnaire remains in its early stages of development, it has been found to have good validity and reliability ratings in preliminary research (Sevush, 1999).

However, this measure is not without criticism. In particular, it seems overly simplistic to reduce awareness of memory functioning into just six questions, given the accepted variability of AD. In addition, a self-report style questionnaire, such as this, can only be used with clients with AD who are not aphasic, and so is unlikely to be appropriate for use with individuals in the severe stages of AD. It may also present difficulties for some people with AD whose accurate self-appraisal is likely to be dependent on the social context, wording and specificity of the items (Hart et al., 1998). However, despite these difficulties such a measure is promising for use in assessing global awareness within the memory domain.

Comparison studies

Currently research comparing the different awareness measures is limited and has produced conflicting findings. Emmerson & Bucks (1997) conducted a comparison study comparing Verhey and colleagues'(1993) 4-point clinician rating scale, a patient/carer discrepancy measure of Activity of Daily Living skills (ADL) and AD clients' ratings on dimensions of mood, memory ability and independence. They found no significant difference between the measures in predicting awareness and concluded they were measuring the same thing. However, in a study of brain injured clients, Sherer and colleagues (1998) found that a patient-carer discrepancy awareness measure produced significantly different results to a patient-clinician discrepancy measure. Furthermore, McGlynn and Kaszniak (1991) found a patient-carer discrepancy measure of awareness and a patient-performance discrepancy measure also produced inconsistent results. They suggested that questions requiring clients to make abstract judgements about their disability, require a different level of awareness than that required in making predictions about their current ability to perform concrete tasks. Certainly, it is possible that these measures are tapping different aspects of awareness, with one tapping a more global awareness level and the other tapping task specific awareness (Cotrell, 1997). Alternatively, it is possible that this difference in abstract and concrete tests may result from deficits in executive functioning and reflect impaired reasoning as opposed to impaired insight per se. Theoretically these two processes could be distinguished if a person with impaired insight exhibited unimpaired performance on tests of reasoning. A small number of such cases (e.g., Migliorelli et al., 1995; Reed et al., 1993) have been reported in the literature, but further investigation is warranted in order to fully explore this difficulty in awareness research.

Therefore, further research is required to evaluate these measures. However, it seems likely that comparisons between studies using different measures and different awareness levels/domains are problematic.

In summary, all of the measures discussed have methodological limitations and require further validation. Furthermore, they all have a high loading on expressive and receptive language skills, with which some people with AD may have difficulty. This may limit the types of clients who can be assessed using this method and, thus, the generalisability of the findings. There remains a need for a well validated and consistently used measure of insight, with the lack of one representing a substantial weakness in the research that has been carried out to date. At present, a combination of methods to define insight may be the most prudent approach, given the limitations of individual measures.

Current research evidence

Studies into impaired insight in the AD population have been instrumental in investigating the dimensions of self-awareness and potential aetiological correlates. Given the methodological problems discussed above, it is perhaps unsurprising that research currently remains in its infancy. However, some tentative findings have been reported and these will be discussed in the following sections.

Dimensions of anosognosia

There is significant evidence to suggest that insight is not a unitary "all or nothing" phenomenon, but varies across a number of levels.

Degree

Neary and colleagues (1986) found that different subgroups of individuals with AD exhibited different degrees of awareness of their impairments. Whilst some individuals were keenly aware of their impairments and openly acknowledged them, others denied them completely and showed a complete lack of insight into them. Others, still, underestimated the severity of their impairments, whilst acknowledging they had difficulties; whilst others again displayed overt signs of anxiety when asked to complete difficult tasks, yet denied any problems, suggesting they had some degree of insight. Such evidence suggests that the degree of insight found within the AD population varies between individuals and is likely to be on a continuum.

Domain

Research into the occurrence of impaired insight in disorders, such as hemiplegia and traumatic brain injury, has suggested that it does not merely represent a global deficit of awareness, but can also be domain specific (e.g., Bisiach & Geminiani, 1991; Mazzioni et al., 1997;). For example, individuals with hemiplegia have been found to lack awareness of their motor abilities, but be aware of their functioning in cognitive domains (Von Hagen, & Ives, 1937). Domain dissociations have also been found within the AD population. For example, Vasterling and colleagues (1995) found that individuals with AD were less aware of their higher-order deficits

(e.g., memory and self care), than emotional disturbances and general health. Similarly, Kotler Cope and Camp (1995) found that severely impaired people with AD had reasonably preserved awareness of psychiatric and behavioural problems (e.g., agitation, need for routine, depression and disorientation) compared to their awareness of cognitive problems (e.g., language disorder, higher cognitive deficits, memory disorder and apraxia). Furthermore, Ott et al. (1996) found that in people with AD, awareness of memory functioning was more disrupted than awareness of ADL functioning. In addition, interestingly, people with AD have been shown to retain awareness of other peoples' functioning, while lacking insight for their own (Reisburg, Gordon, McCarthy & Ferris, 1985; Schacter, 1990). Such evidence suggests that people with AD exhibit domain specific impairments in awareness.

Over time

Neundorfer (1997) described how carers often reported windows of clarity in their relatives' insight. Unfortunately, whilst temporal variability of awareness within the individual has been well documented in AD case reports, empirical investigation is lacking. An exception to this, however, is a recent study of meta-memory by James and Moulin (2002) in which they researched individuals' with AD ability to predict how many words they could recall from a word list, across trials. They were able to show that some participants demonstrated improved accuracy in their predictions over trials, and suggested that this indicated that their insight changed with exposure to their impairments.

The variability of insight, both between individuals and over time, has important implications for the methodologies we use to assess impaired insight. Current

research has largely failed to take into account the temporal variability of insight. Measuring awareness at one time interval, is unlikely to give an accurate picture of the average level of awareness and may represent an over- or under-estimate (Bucks, 1998). Furthermore, studies have varied in the specifity of awareness. Some studies have focused on awareness of global functioning, whilst others have looked at specific domains, such as memory and ADL skills; others still, have looked at on-task awareness; while others again have looked at abstract knowledge of functioning. It is possible that each of these areas of awareness is differently affected in people with AD and so the comparison between such studies may be inappropriate.

Association with severity of AD

A number of studies have found a correlation between insight and severity of AD (e.g., Migliorelli et al., 1995; McGlynn & Kaszniak, 1991; Zanetti et al., 1999). Reisberg and colleagues (1985) found that individuals with advanced AD ($n = 25$) were significantly less aware of their cognitive deficits than people with mild dementia ($n = 5$), who in turn, were less aware than healthy older adults with age related memory loss ($n = 10$). Although this was a small study, similar findings have been found in larger studies. For example, McDaniel, Edland, and Heyman (1995) looked at this relationship in a sample of 650 participants, using a clinician rating of awareness. They followed up the participants after a two-year delay period and found those participants with greater cognitive impairments were significantly more likely to suffer from impaired insight at both time points. Sevush (1999) also replicated this finding using the AMIS and found a significant relationship between awareness of memory deficits and severity of AD. These findings suggest that

insight for memory functioning is more likely to be retained in the early stages of AD, and then decrease as the disorder progresses.

However, these studies have been criticised as the strength of association between AD severity and insight has tended to be at best modest. Indeed, whilst McDaniel and colleagues (1995) reported a significant relationship between insight and the severity of AD, they failed to comment on their finding that 57% of participants had not declined in their level of insight during the delay period and 10% actually showed improvement. Furthermore, some studies have found no significant relationship between insight and the severity of AD (e.g., Michon et al., 1994; Reed, Jagust & Coulter,1993). Whilst these inconsistencies may be due to methodological issues, the variability in findings is consistent with clinical observations that some individuals with only mild AD lack insight into their deficits, whereas some people with moderate dementia retain awareness of their deficits (Derouesne et al., 1999; Feher et al., 1991). Thus, although it is common for awareness to be progressively lost as AD becomes more severe, severity of AD, can at best, only partially account for the variability of impaired insight in AD.

Association with depression

Despite difficulties in assessing depression in individuals with AD, a number of studies have suggested that it is particularly common within this disorder (e.g., Burke, Rubin, Morris & Berg, 1988), with prevalence rates largely falling within the 10% to 50% range (Brodaty & Luscombe, 1996; Burns, 1991; Parks, Zec & Wilson, 1993). This has led to speculation regarding the relationship between insight and depression. Intuitively, it would seem likely that reactive depression

might be more common in individuals who retain insight into their difficulties, with depression trailing off as the degree of insight decreases. Indeed, a number of studies have supported this hypothesis (e.g., Harwood, Sultzer, & Wheatley, 2000; Seltzer, Vasterling, Hale & Khurana, 1995; Smith, Henderson, McCleary, Murdock & Buckwater, 2000). In particular, McGlone and associates (1990) reported a significant positive relationship between scores on a self-report memory impairment scale and the Geriatric Depression Scale. However, in contrast, Freidenberg, Huber, and Dreskin (1990) found the opposite relationship, in that more self-aware people with AD tended to be less depressed. Furthermore, other studies have found no significant relationship between depression and insight within this population (e.g., Lopez, Becker, Somsak, Dew, & DeKosky, 1994; Ott, et al., 1996; Reed, et al., 1993) Thus, the relationship between depression and anosognosia remains controversial.

These differences in findings may reflect the methodological variations between studies. In particular, studies have differed depending on whether they have evaluated clinical depression (Ott et al., 1996) or dysthymia (Starkstein et al., 1997). Harwood and colleagues (2000) reported that research that used observer-based scales (e.g., Hamilton Depression Rating Scale, Cornell Scale for Depression), which include somatic or neurovegative symptoms of depression, have all reported negative findings. On the other hand, studies focusing on the self-reported subjective expression of depression (e.g. dysthymia) have reported positive findings, suggesting depressive symptoms have been associated with greater insight. Whilst it could be argued this represents a dissociation between dysthymia and major depression, with dysthymia being proposed as a reaction to perceived

cognitive decline and major depression being an independent disorder (Migliorelli et al., 1995); it could also be argued that the difference is due to the assessment method used in these two types of studies (Ott et al., 1996). The self-report scales used in subjective expression of depression studies, require insight to identify depressive symptoms, whereas the studies using the observer-based scales do not. Indeed, Mullen and associates (1996) have argued that many depressed individuals show a tendency to underrate their own memory performance, which may appear to be a sign of greater retention of insight in a group of people with AD, but may in reality represent a confounding effect of the overlap between symptoms of the two disorders. Certainly, one of the major criticisms of such studies is the general lack of depression scales that have been validated for use with the AD population (Cummings, Ross, Absher, Gornbein & Hadjiaghai, 1995).

However, whilst these methodological difficulties are likely to cloud the significance of any relationship between depression and insight in people with AD, clinical reports do add support to the variability of the empirical evidence. Such reports have suggested that some individuals with severe dementia are depressed (Verhey, et al., 1993) and other individuals, who have retained their insight, are not depressed (e.g., Emmerson & Bucks, 1997). Therefore, it would seem that, whilst a relationship between depression and insight is likely to exist, the nature of this relationship is unclear and does not fully account for the variability in awareness found within this population.

Association with neurological functioning

Perception

Early research into anosognosia for motor deficits in hemiplegia, found a strong association between impaired insight and lesions in the inferior (right) parietal regions of the brain (McGlynn & Schacter, 1989; Bisiach, Vallar, Perani, Papagno, & Berti, 1986). This has led a number of researchers to investigate the association between right parietal functioning and self-awareness in people with AD (Lopez et al., 1994; Michon et al., 1994; Reed et al., 1993). However, whilst some neuropsychological studies within the AD population have found correlations between insight and performance on tests of visual construction and spatial skills (e.g., Reed et al., 1993); the evidence has been far from conclusive, with a number of studies failing to replicate these findings (Lopez et al., 1994; Michon et al., 1994). Furthermore, visual construction tests are unlikely to represent pure parietal functioning. Indeed, Reed and colleagues (1993) acknowledged that the frontal lobes are also likely to be implicated in visuo-constructional ability and that awareness may involve a parietofrontal pathway, connecting frontal, parietal and limbic structures. The relationship between the inferior parietal lobe and insight therefore remains contentious. Even research investigating this relationship within the field of hemiplegia, where this relationship has been found to be strongest, has produced mixed results, with not all individuals with right hemisphere damage exhibiting impaired insight and some individuals with no obvious parietal damage appearing unaware of their deficits (McGlynn & Kaszniak, 1991).

Executive dysfunction

The Central Executive System (CES), believed to be associated with the frontal lobes, is responsible for self-monitoring, working memory, cognitive resource allocation and set shifting (Baddeley, 1992; Stuss & Benson, 1986). Such cognitive functions could, debatably, be instrumental in achieving insight into one's functioning. This has led to suggestions of an association between insight and CES functioning (Stuss & Benson, 1986). Impaired insight has commonly been reported in disorders that are known to result from deficits in the frontal lobe areas, e.g., Korsakoff's syndrome (Jarho, 1973), Pick's disease (Neary, et al., 1990) and Huntington's disease (McGlynn & Kaszniak, 1991). Furthermore, people with discrete localised damage to the frontal regions, following penetrative brain injury (e.g., Jahro, 1973), have been found to lack awareness of their memory difficulties, whilst people with amnesia without frontal signs, have been reported as being aware of their deficits (e.g., Kaushall, Zetin & Squire, 1981).

Research into the association between insight in clients with AD and CES functioning has shown some replication of these findings, with evidence suggesting that poor performance on tests of frontal lobe functioning is more predictive of impaired insight than overall severity of cognitive impairment (e.g., Lopez et al., 1994; Ott et al., 1996; Zanetti et al., 1999). For example, Michon and associates (1994) found a disproportionately strong relationship between a patient-carer discrepancy measure of insight for memory functioning and AD participants' performance on a number of tests of executive functioning. Further correlations with other neuropsychological functions were found to be insignificant. Importantly they also found that the relationship between insight and frontal lobe functioning

was independent of the severity of cognitive impairment as measured by the Mini Mental State Examination (Folstein, Folstein, & McHugh, 1975).

However, other studies have not replicated these findings, finding no association between executive functioning and insight, or finding equally strong associations with other cognitive domains, such as memory (Migliorelli et al., 1995; Reed et al., 1993). Indeed, a recent study by Clare (2001) found no relationship between AD participants' performance on tasks thought sensitive to frontal lobe functioning and participants' awareness ratings as measured by clinician interviews, patient/carer discrepancies, or prediction/task performance discrepancies. And whilst Lopez and associates (1994) found a significant correlation between AD clients' performance on tests of frontal lobe functioning, a review of the participants' CT scans did not suggest that those participants with more severely impaired insight had greater atrophy of the frontal lobes. These studies suggest it is unclear whether the presence of dysexecutive symptoms reflects increased frontal lobe dysfunction, or simply a greater distribution of AD pathology. Research into this area is greatly hindered by methodological problems, with no consistency between the studies on the test of CES used. Perhaps more significantly, like many tests, current CES tests can be criticised for not being "pure" tests as they commonly tap into perceptual and memory functioning as well as executive skills.

Memory

Given the significant role memory impairment plays in the characterisation of AD symptomatology, it is, perhaps, unsurprising that the relationship between memory functioning and insight has been the focus of much research in the literature. A

number of studies have found a positive correlation between self-awareness and degree of memory impairment (Feher et al., 1991; Ott et al., 1996; Reed et al., 1993). For example, Feher and colleagues (1991) found that a carer-patient discrepancy measure of insight for memory functioning was significantly correlated with memory impairment on subtests of the Wechsler Memory Scale (Wechsler, 1945), and that this relationship was stronger than the correlation between insight and overall severity of cognitive deficits. Such findings have been argued to suggest that impaired insight in people with AD is due to them forgetting they forget (Mangone et al., 1991).

However, contrary to these findings, other studies have not demonstrated a relationship between memory and insight (Michon et al., 1994; Seltzer, Vasterling, Mathias & Brennan, 2001). Verhey and colleagues (1993) found that many of the memory impaired clients in their sample were aware of their deficits. Furthermore, studies of pure amnesic syndromes of temporal lobe origin, with no frontal lobe pathology, have not exhibited impaired insight into their difficulties (e.g., Byer & Crowley, 1980). In addition, those studies that have found a significant relationship have varied in the type of memory they found to be associated with insight. Some studies have only identified a relationship between insight and recall memory (Feher et al., 1991; Ott et al., 1996), whilst for others it has been with recognition memory (Reed et al., 1993). Others still, have found a relationship for immediate memory only (Feher et al., 1991), whilst others, have found it only for delayed memory (Ott et al., 1996). Whilst it is possible these differences are due to inconsistencies in the measures used, it seems unlikely that there is a straightforward causative relationship between memory loss and impaired insight in

people with AD. However, the possibility remains that memory impairment, plus other factors, may interact to account for the unawareness of deficits seen in people with AD.

Thus, whilst neurological damage to the inferior parietal lobe, frontal lobe and limbic system have all been implicated as possible causes of impaired insight in AD, the research evidence as to their causative role has not been conclusive. This may be due to the diffuse nature of damage exhibited in clients with AD making the relationship between neurological lesions and impaired insight difficult to prove. Furthermore, current research findings are largely based on neuropsychological tests, which can be criticised for having questionable reflection on the areas of the brain involved in awareness (Mullen et al., 1996).

Summary

Insight in AD does not appear to be a unitary phenomenon, with it varying in presentation both between and within individuals in its degree, domain specificity and stability over time. With regard to aetiological correlates, research has suggested that awareness of impairments in AD may be related to global cognitive decline, right hemisphere dysfunction, frontal dysfunction, memory impairment and depression. No one factor has been found to be responsible across all studies. The variability of the findings may be a consequence of methodological inconsistencies and assessment difficulties or, as is more likely, the heterogeneous nature of insight.

Theoretical review

Over the years, researchers have attempted to develop theories to understand the complex concept of insight. Such theories can largely be divided into three types; social environmental, psychological defence and neuropsychological. Although some of them were developed from research into other disorders, such as hemiplegia and traumatic brain injury, for the purpose of this paper they will be applied to our understanding of insight in AD.

Social environment perspective

Jacobs (1993) and Kilhlstrom and Tobias (1991) suggested that the main cause of impaired insight was a lack of information being provided about the individual's level of functioning. They argued that for a person to be aware of their impairments they needed to experience the incapacity. However, people with AD are often protected from experiencing their illness by their family and medical professionals. They may not be told their diagnosis, or may have their environment controlled in such a way as to prevent them from encountering tasks they will find difficult or distressing. This results in the person's insight being externally impaired by the constraints of their social environment.

Treatment implications

Advocates of this model emphasise the value of a carer psychoeducational approach to treatment. Such interventions would actively involve the carer and assist them in creating an environment for the client that allowed them to function at an optimal level of independence for their abilities, whilst still keeping them out of physical

danger and preventing significant damage to their self-esteem (DeBettignes et al., 1990). Once the individual with AD became more aware of their capabilities, psychological support and compensatory skill training could then be offered to help them adapt to their illness and reduce the amount of burden on the carer.

Critique

Although it is possible that some people's unawareness of their deficits can be attributed to social environmental issues, research evidence does not suggest that this is the main, or sole explanation, for impaired insight in clients with AD. Indeed, research has suggested that when the majority of clients with AD have been asked to comment on their performance after carrying out a task, they have been found to be less aware than healthy controls (e.g., McGlynn & Kaszniak, 1991). Furthermore, one would expect that as individuals with AD become more severely cognitively impaired it would be harder for carers to hide this from the client, which, logically, would lead to improvements in their insight into their difficulties. However, as discussed previously, evidence suggests insight generally becomes worse as the client becomes more cognitively impaired.

Psychological defence perspective

Some authors have suggested that the loss of one's abilities through illness represents a threat to one's sense of self and that impaired insight plays a functional role in protecting against this (e.g., Clare, 2001;Weinstein & Kahn, 1955). The onset of AD is, arguably, a significant threat to self both physically, as there is currently no cure, and socially, as the person becomes more dependent on others as

their abilities deteriorate. Thus, people may deny their deficits (consciously or subconsciously) because awareness would lead them to face a reality that is both threatening and unpleasant (Cocchini, 1996; De Vreese, Neri, Fioravanti, Belloi, & Zanetti, 2001). This view is supported by research that has found an inverse relationship between severity of AD and insight, as well as research that has found a positive correlation between depressive symptoms and insight (e.g., Seltzer, et al., 1995; Smith et al., 2000). Intrapsychic theorists have interpreted these findings as evidence that the individual uses stronger psychological defences as they attempt to cope with the threat of worsening cognitive abilities, with those who do not use such coping mechanisms being more likely to suffer depressive symptoms (e.g., Weinstein & Kahn, 1955). Further support for this theory comes from a study by Weinstein and associates (1994). They investigated premorbid personality traits associated with denial in a sample of people with mild/moderate AD and found that those with higher ratings of denial personality traits had less insight.

The advantage of this theory is that it can explain the non-unitary presentation of impaired insight in the AD population. Kitwood (1997) suggested that premorbid coping styles continue to be used by people with dementia as a means of coping with the threat of mental impairment, with coping styles varying between denial, dependency, repetitive behaviour, planning and accepting offered help. Thus, insight may depend on the individual's method of dealing with psychological stress and the perception of stress. Indeed, Clare (2002), in a study of 12 people in the early stages of AD, found that coping strategies described by the participants fell along a continuum that ranged from self-protective responses (e.g. denial), where the person strives to maintain a prior sense of self, to integration responses, which

allow for adjustment of the self concept. Furthermore, the domain specificity of impaired insight may be attributable to a selective denial of the impairments the individual finds most threatening to their sense of self. Fleming and Strong (1999) found that, in a sample of adults with traumatic brain injury, the three most frequently over-estimated ADLs were managing finances, driving a car and recognising if they had upset someone. These tasks are key areas of personal control, independence and self-esteem.

Treatment implications

Intrapsychic theorists warn against the potentially psychologically damaging effect of using confrontational approaches that emphasise the client's impaired abilities. Instead, they stress the importance of understanding the subjective experience of AD for the individual, taking time to gain an understanding of what the individual's personality and life was like before the illness, what they valued, what they enjoyed and how they coped with previous stresses (Toglia & Kirk, 2000). Clinical management of the illness can then be addressed through the therapeutic management of psychologically motivated defences (Lewis, 1991), such that the person is helped to find ways to maintain hope and strengthen their sense of self, whilst also facilitating adjustment and adaptation to the changes that dementia brings. Again, work with the carer may be helpful in such cases, particularly where denial is so strong that no direct psychological input is tolerated.

Critique

This theory has been criticised for a number of reasons. Firstly, it is overly simplistic in its attempt to explain the high incidence of impaired insight in AD as

solely due to psychological defence mechanisms. Indeed, it does not adequately explain why impaired insight is more common in AD than in other progressive dementias, or why impaired insight has been connected with disorders associated with discrete areas of brain damage. Furthermore, given evidence to suggest there is a trend between severity of AD and impaired insight, logically it seems unlikely that more cognitively impaired individuals would be able to implement a coping strategy more effectively than those who are less impaired (Seltzer et al., 1995). Finally, this theory is difficult to evaluate objectively due to the problems in assessing psychological defence mechanisms in people with cognitive deficits and the fact that most studies have used retrospective measures of pre-morbid personality traits. As a result, it has received little empirical evaluation. Indeed, the most recent study that sought to evaluate this in a sample of clients with "non-frontal" dementia (Seiffer & Clare, 2003), found that pre-morbid negative attitudes towards emotional expression, and the use of avoidant strategies in managing the experience of dementia, were not related to unawareness. Furthermore, whilst the study did find a significant relationship between participants' pre-morbid conscientiousness and unawareness, this was not significant when disease-related variables and emotional factors were controlled for. Thus, it seems unlikely that the use of denial as a coping strategy is the main cause of impaired insight in AD. However, it is possible that it interacts with other aetiological factors to create additional challenges for treatment.

Neuropsychological perspectives

Focal lesion theories.

It is well recognised that lesions in discrete areas of the brain often lead to predictable disturbances in cognitive function. A number of theories associating impaired awareness with damage to specific areas of the brain have been recorded in the literature. Three main areas have been put forward as possible centres for awareness.

1.) Right Parietal Lobe

As the right parietal lobe is believed to be associated with directing attention to extra-personal space, some authors (e.g., Giancino & Cicerone, 1998; Crichley, 1953; Roth, 1949) have proposed that lesions to this area lead to the representation of the body image being disconnected from awareness, with the result that the person fails to recognise their body has altered. However, whilst this theory goes some way to explaining the anosognosia for physical impairments presented by clients with hemiplegia, it does not readily account for the difficulties experienced by people with AD, in whom the impairment is largely for non-physical abilities, such as memory. Indeed, as discussed earlier, the research evidence within this population has shown only weak evidence of the role of the parietal lobes. Thus, whilst it is possible that the parietal lobes play some role in self-awareness, it seems unlikely that this theory fully accounts for the insight impairments exhibited within the AD population.

2.) Frontal lobes

As previously discussed, a large body of research has associated impaired insight for cognitive abilities with frontal lobe damage. The frontal lobes have been associated with executive functions, such as self-monitoring, and this has led Stuss and Benson (1986) to propose that impaired insight is a deficit of self-monitoring, such that changes in abilities are not detected and continue to be reported at their pre-morbid level. However, whilst there is a strong association between frontal lobe damage and impaired insight, it does not account for the reports of impaired awareness in people with no apparent brain damage, or with posterior lesions only.

3.) Limbic system

Zec (1993) and Moulin and associates (2000) have both drawn on the research into memory functioning and anosognosia in people with AD (e.g., Migliorelli et al., 1995) and have suggested that impaired insight represents a memory impairment, whereby damage to the limbic pathways prevents the encoding of an enduring record of their experience. This theory, in particular, is helpful in explaining the occurrence of moments of apparent clarity in insight. It argues that a person with impaired insight is able to acknowledge their deficits when exposed to them, but that this information is not permanently encoded into their memory. However, this theory has not been supported by studies of clients with pure amnesic syndromes who, despite damage to their mesial temporal lobes and severe memory impairments, generally have retained insight. Furthermore, a number of AD studies have failed to find an association between memory functioning and awareness, suggesting that memory may have a maintaining role, as opposed to a linear causative role, in awareness (Agnew and Morris, 1998).

Overall, the focal lesion theories do not adequately explain the variations found within the research literature, particularly regarding domain specificity (McGlynn & Schacter, 1989). However, the strong correlations between specific areas of anatomic brain damage and impaired insight in a number of studies have convinced many researchers that such damage plays a significant, but not an exclusive role in impaired insight.

Conscious awareness system model.

McGlynn and Schacter (1989) proposed a model of awareness that attempted to integrate the focal lesion theories and account for the domain specific variability of awareness deficits. They proposed that there was a central monitoring system, the "Conscious Awareness System" (CAS), which had a neural basis within the parietal lobes and was of principal importance in bringing a person's experiences of their functioning into awareness. They suggested that the CAS receives outputs from various domain specific modules within the brain. The CAS is also proposed to have an output link to the Central Executive System (CES) located in the frontal lobes, which is concerned with initiation, organisation and monitoring of the more complex sequences of cognitive outputs, such as memory, problem solving, and social behaviour. They postulated that impaired insight resulted from damage to either of these two systems. If damage occurred to the CAS, itself, impaired insight of all neuropsychological deficits would result. However, if there was a disconnection of the CAS from a particular neuropsychological input module, then impaired awareness for a specific impairment, such as motor deficits, would occur.

On the other hand, if damage occurred at the CES level, then an impaired awareness for more complex deficits (e.g. memory) would be presented.

However, this model can be criticised for a number of reasons. Firstly, whilst it does account for the domain specific variability of impaired insight, it fails to account for the temporal variability often reported within the literature. Furthermore, it does not fully explain the reports of relatively preserved insight in some people with severe dementia who, it could be argued, are likely to have greater damage to their frontal and parietal lobes. In addition, this model has been criticised for its lack of process detail, being described by Agnew and Morris (1998) as a "skeleton model" (pp.14). Indeed, it gives no details of the mechanisms responsible for metacognitive output or the awareness of specific domains of functioning. Furthermore, it fails to address how memory impairments may contribute to impairments of insight in domains other than memory.

Domain Specific Model.

Agnew and Morris (1998) proposed a new model of awareness for memory functioning that develops McGlynn and Schacter's (1989) model and attempts to overcome some of its limitations. They suggested that our knowledge of our abilities is constantly being revised in light of new incoming information. In keeping with McGlynn and Schacter's (1989) model, they proposed the individual, initially, consciously experiences the memory event via the CAS. However, they suggested that the information is also relayed to a mnemonic comparator (Cm) located within the CES, where the information is compared with the individual's existing knowledge of their memory functioning held in a Personal Knowledge

Base (PKB) within semantic memory. If a mismatch is detected, then the PKB is updated and the contents of the PKB are then fed into the CAS, resulting in the individual becoming consciously aware of the discrepancies, thus, making them able to change their behaviour and expectations accordingly.

Agnew and Morris (1998) felt that three distinct types of insight impairment could be caused by specific deficits in this system and that this could account for the heterogeneity of anosognosia for memory impairment exhibited by individuals with AD. These were: mnemonic anosognosia, which involves damage to the pathway between the Cm and PKB and results in a temporally variable type of anosognosia, whereby the person is immediately aware of any memory failure when it occurs, but are unable to permanently update their PKB, resulting in them forgetting they forget; executive anosognosia, which involves damage to the Cm and results in a static domain specific anosognosia; and primary anosognosia, which involves damage to the CAS and results in a global form of anosognosia.

Unfortunately, to date, there has been little research evaluating this model of awareness. However, it does provide a useful framework for future research.

Treatment implications

Neuropsychological models of awareness, such as the focal lesion models, have suggested that, due to the stability of organic damage, individuals with impaired awareness are unable to improve their insight. Authors adhering to this view have suggested that external compensation strategies are the rehabilitation intervention of choice for use with such clients (e.g., Regnier & Pynoos, 1992). Such strategies

usually involve the responsibility for managing the memory deficit being taken over by another person, for example, the carer. Such techniques might include modification of the environment and the use of prompts and cues, e.g., laying out the clothes to be worn (Regnier & Pynoos, 1992).

However, this approach has been criticised for taking away the client's independence and sense of personhood (Kitwood, 1997). Furthermore, more recent neuropsychological models, such as Agnew & Morris (1998), have suggested that, whilst some people with AD are unable to improve their level of insight due to their organic damage, others may, with recent and repeated exposure to their deficits, be able to change their behaviour at either an explicit or implicit level. Indeed, evidence does suggest that some people with AD are able to achieve new learning following such exposure (Moulin et al., 2000; Sohlberg et al., 1998). In particular, Butters, Glisky and Schacter (1993) found that for some individuals with memory impairments it was possible to demonstrate transference of self-awareness through the use of repetition and over-learning techniques and that this effect was increased when the stimulus material was meaningful to the participant. Such strategies may include video feedback, small group discussions, education regarding deficits (Deaton, 1986), reality testing through engagement in real life activities (Cicerone, 1989) and "planned failure" experiences (Marme & Skord, 1993).

However, little research has been completed to evaluate these rehabilitation techniques, especially for use within the AD population. The benefit of their use with this client group, therefore, remains largely speculative.

Critique

Overall, none of the neuropsychological models fully accounts for impaired insight in AD. In particular, they can be criticised for ignoring the effects of psychological coping strategies and environmental influences. However, the strong correlations between specific areas of anatomic brain damage and impaired insight in a number of studies, has convinced most researchers that such damage plays a significant, but not an exclusive role in impaired insight. Agnew and Morris's (1998) model of memory awareness provides a promising framework for understanding organically impaired insight within this population, but requires further validation.

Towards an integrated approach - Multifactorial Model

Neither psychological, social environmental, or neuropsychological models fully explain the relationship between impaired awareness and AD. Multifactorial models have attempted to combine the research evidence used to support these arguments into an integrated model of awareness (e.g., Clare, in press; Toglia & Kirk, 2000).

Of particular interest, Clare (in press) has recently developed a biopsychosocial model as a means of understanding the construction of awareness in the early-stages of AD. This model attempts to integrate the neurological, social and psychological models discussed previously. The major premise of this model is that awareness is a complex and fluid phenomenon that is primarily influenced by the person's psychological sense of self. Clare (in press) argues that the onset of AD should be viewed as a potential threat to the sense of self, with the degree of threat determined by the interaction between biological (e.g., neurological damage), psychological (e.g., premorbid coping style) and social factors (e.g., social context). Each factor

has the potential to affect self-awareness, both independently and in conjunction with the other factors. Clare (in press) suggested this results in the non-unitary characteristics of impaired insight observed in the literature.

Treatment

Authors who support the multifactoral view of awareness have emphasised the importance of completing a thorough assessment to establish the degree and type of impaired awareness the client is exhibiting (Mullen, et al.,1996) and then matching those treatment approaches discussed earlier with the type of awareness impairment. There is a danger that if interventions are not adequately matched to the awareness type, they may actually be harmful or ineffective for the client (Fleming et al., 1998). Unfortunately, little research has been completed to evaluate the use of different rehabilitation techniques with specific impaired awareness types. Furthermore, the benefit of individual treatments for use in the AD population has received little empirical evaluation and treatment difficulties are accentuated by the current difficulties in discriminating organic impaired awareness from psychological defence and social influences.

Critique

The biopsychosocial model has been developed only recently and as yet has not been formally published. As a result, it has yet to be empirically evaluated. Furthermore, it draws on models that have themselves received little explicit empirical evaluation. In addition, it suggests that awareness is primarily a psychological phenomenon, to which neurological and social factors contribute. However, it can be argued that this is not supported by evidence that impaired

awareness is more common in AD than other dementias (e.g., vascular dementia), or the strong associations found with specific areas of neurological damage. In fact, this evidence would suggest that neurological factors have at least an equal influence on awareness.

General conclusion

Impaired insight is a complex problem that is commonly found in people with AD. As such, it has significant implications for our understanding of AD and the treatment of people who suffer from it. Current research has been severely hampered by a lack of clear definition of insight, methodological difficulties in assessment, a lack of consistent experimental design and the use of retrospective studies. However, despite these difficulties a number of tentative findings have been made. Current AD evidence suggests that insight is not a unitary concept, but varies temporally, in degree and in the domains affected. Furthermore, a number of studies have found it to be associated with various factors, including memory, perception, executive functioning, depression and severity of AD. These findings have, however, not been found universally across the literature, with no one factor appearing to have a linear causative role in the development of impaired insight. Hence, these findings suggest that impaired insight is a heterogeneous phenomenon.

Given these findings, self-awareness within the AD population appears to be best understood within the context of a multifactorial model, in which a number of factors determine the degree of insight. As discussed, Clare's (in press) biopsychosocial model has sought to combine the perspectives of previous models

and argues that a mixture of psychological defence, social environmental and neurological factors determines awareness in the AD population. Whilst, this model goes some way in creating a useful structure by which to understand awareness, it is currently speculative. Furthermore, it can be argued that this model over emphasises the role of psychological defence in causing impaired insight in AD. Current evidence would suggest that neurological factors have at least an equal influence on awareness. Indeed, Agnew and Morris's (1998) model of memory awareness provides a useful, although again unvalidated, model of the subtypes of organic insight impairment. Clearly, future research is needed further to define the dimensions and subtypes of awareness within this field.

Clinical implications and future research

Deficits in awareness can have significant implications for a person's quality of life and the burden experienced by carers. Furthermore, it is known severely to restrict rehabilitation outcome, by interfering with treatment motivation, engagement in therapy and the use of compensatory strategies (Clare, 2002; Koltai, et al., 2001). However, this is not to say that people with impaired insight are unable to benefit from rehabilitation techniques, or that they are unable to increase their awareness via psychological intervention. The apparent heterogeneous nature of awareness, particularly the temporal variability, may suggest that some people are able to adapt their insight and may benefit from individualised rehabilitation approaches dependent on their awareness type. Knowledge of the factors that influence self-awareness is, therefore, likely to help health professionals to establish which clients would benefit from specific rehabilitation programmes. The sub-types of impaired awareness, proposed by Agnew and Morris's (1998) and Clare's (in press) models,

provide some tentative guidance on this. It can be hypothesised from the models, that for some types of impaired insight (e.g., mnemonic and social environmental anosognosia), an appropriate intervention strategy may be to increase the client's level of awareness by using exposure and repeated learning techniques; for others (e.g., primary anosognosia and psychological defence) it may be better to facilitate compensatory behavioural changes, without specifically addressing their awareness of their deficit; and for others still (e.g., executive functioning), it may be best to work with the carers supportively, to address expectations of the client and enhance their behavioural management skills. Finally, family intervention is likely to be useful for all types of impaired awareness as a means of reducing carer burden.

However, before the issue of rehabilitation can be adequately tackled, research identifying the sub-types of awareness within this population is required, together with investigations into whether individuals with any of the sub-types are able to acquire and maintain improvements in awareness; whether this is acquired at an implicit or explicit level; and whether improvements in awareness can be generalised to situations outside of therapy (Cotrell, 1997).

The majority of past research has viewed insight as an unitary and global concept and, as a result, has failed adequately to investigate the dimensions of its variability. Future research should, therefore, attempt to identify sub-types of impaired awareness by assessing insight at a number of levels, including global, domain specific, implicit, explicit, "on-task" insight and self-knowledge. In addition, research that attempts directly to investigate the validity of the components of these multifactorial models is required to advance our understanding of this phenomenon.

Without such studies it is unlikely that our understanding of impaired insight within the AD population and its potential treatment implications will be advanced. Indeed, only once the characteristics of the individual sub-types of awareness have been established, can we identify the most appropriate rehabilitation techniques to treat this disorder.

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Empirical Paper:

Mnemonic Anosognosia in people in the Early-Stages of Alzheimer's disease

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Mnemonic Anosognosia in people in the Early-Stages of Alzheimer's disease

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Mnemonic Anosognosia in People in the Early Stages of Alzheimer's Disease

Abstract

Agnew and Morris' (1998) model of awareness for memory functioning has attempted to account for the variance of anosognosia exhibited within the Alzheimer's Disease (AD) population. To date, this model has lacked direct empirical evaluation. However, 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. This study investigated this by exploring the dimensions of awareness exhibited by such individuals. Eighteen older adults with early-stage AD and 18 healthy older adults were recruited. Their awareness was monitored across a number of dimensions by using a patient-performance measure of implicit and explicit "task specific" awareness; the Awareness of Memory Impairment Scale (AMIS) of global memory awareness; and a patient-informant discrepancy measure adapted from the AMIS. The stability of participants' awareness was measured across three word recall trials and after a 20-minute delay. Results suggested that, whilst the participants with early-stage 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 a 20-minute delay period. However, there was evidence to suggest that a longer delay period may have resulted in the resetting of their awareness levels. These findings were largely in keeping with the profile of mnemonic anosognosia. The implications of these findings for clinical management and future research are discussed.

Mnemonic Anosognosia in People in the Early Stages of Alzheimer's Disease

Introduction

Anosognosia is a term used within the neurological field to describe an organically derived absence of insight into physical, neurological or cognitive impairments (McGlynn & Schacter, 1989; Schacter, 1990). Whilst it has been associated with a number of disorders (McGlynn & Schacter, 1989), anosognosia for memory functioning is a particularly common feature of the clinical presentation of people with Alzheimer's Disease (AD), with a prevalence rate estimated to be between 20-80% (Migliorelli et al., 1995; Sevush & Leve, 1993). As a result, it poses significant implications for the clinical management and quality of life of people with AD (Cramer, 1992; Cottell, 1997; Fleming, Strong & Ashton, 1998; Koltai, Welsh-Bohmer & Schmechel, 2001) and their carers (DeBettignes, et al., 1990; Seltzer, Vasterling, Yoder & Thompson, 1997; Trudel, Tryon & Purdum, 1998). If a person is unaware of their impairments they are less likely to adopt strategies to compensate for them, placing themselves at increased risk of harm and, in turn, putting a greater burden on their carers to manage their difficulties externally. However, despite these significant clinical implications, little is currently known about the characteristics of anosognosia within this population, its relationship with the stages of AD, or its underlying mechanisms.

Phenomenology of anosognosia

Our understanding of anosognosia, within the AD population, has been particularly hampered by its complexity of presentation. Recent evidence suggests that anosognosia does not constitute an "all or nothing" unitary phenomenon, but instead varies, both within and between individuals, across a number of dimensions. In particular, it has been found to vary in degree (Bisiach, Vallar, Perani, Papagno, & Berti, 1986; Neary, et al., 1986), the level of awareness involved (e.g., explicit or implicit - De Vreese, Neri, Fioravanti, Belloi & Zanetti, 2001), the domain affected (Vasterling, Seltzer, Foss, & Vanderbrook, 1995), and its temporal stability (Neundorfer, 1997). However, despite such evidence, past studies have tended to assess it as if it were a unitary and static phenomenon, with studies varying greatly in the dimensions of awareness they have assessed. This has made comparisons between studies problematic and has hampered the development of a well-validated model of this phenomenon.

Association with the stage of AD

The evidence for the association of anosognosia with the progression of AD is also currently contentious. Whilst a number of studies have found a significant positive correlation between the degree of anosognosia and the severity of AD (e.g., McGlynn & Kaszniak, 1991; Migliorelli et al., 1995; Sevush, 1999; Zanetti et al., 1999), others have failed to find such a relationship (Michon, Deweer, Pillon, Agrid, & Dubois, 1994; Reed, Jagust & Coulter, 1993). This lack of a consistent association is in keeping with clinical case reports. Whilst most reports have tended to support the correlation between degree of anosognosia and severity of AD, there have been a number of cases reported where people with mild AD have lacked

insight and individuals with more moderate AD have retained full awareness of their deficits (Derouesne et al., 1999; Feher, Mahurin, Inbody, Crook, & Pirozzolo, 1991). This would suggest that although it is common for awareness to be lost progressively as the disease becomes more severe, the relationship is not linear. Instead, it may represent the influence of sub-types of anosognosia, with individuals differing in their vulnerability to specific types, depending on the stage in their disease process and the area of the brain affected (Agnew & Morris, 1998; Lopez, Becker, Somak, Dew, & DeKosky, 1994; Zanetti et al., 1999;). Unfortunately, this theory has, to date, lacked formal empirical evaluation.

Underlying mechanisms

Although it can be argued that psychological and social factors play a role in the presentation of impaired insight (Clare, in press), the disproportionately high incidence of anosognosia in the AD population compared to other dementias (Wagner & Crushman, 1994), suggests cognitive/neurological factors may play a primary role in its development and maintenance. The development of an understanding of these factors has particularly important implications for our knowledge of the progression of AD, the cognitive mechanisms involved in awareness and the clinical management of people with AD.

Attempts to define these mechanisms have focused on the neuropathological correlates of anosognosia for memory difficulties exhibited by clients with AD. In such studies, anosognosia has been widely correlated with right hemisphere parietal damage (e.g., Reed et al., 1993), memory impairment (e.g., Cocchoni, Beschin & Della Sala, 2002) and dysexecutive syndrome (e.g., Reed et al., 1993; Schacter,

1991). However, results have been markedly discrepant, with none of these factors being found to correlate consistently with anosognosia across all studies and all participants. This inconsistency in findings can only partly be accounted for by methodological differences between studies, suggesting that anosognosia is heterogeneous, with a wide variation in presentation within the AD population.

Currently little is known about the cognitive/neurological subtypes of anosognosia found within the AD population. However, Agnew and Morris (1998) have attempted to construct a cognitive framework to explain the apparent heterogeneous nature of anosognosia presented by people with AD. They proposed a model of a memory awareness system and suggested that impairments in this system lead to specific subtypes of anosognosia. Their model postulates that memory awareness is achieved by information from episodic memory being passed simultaneously to the Conscious Awareness System (CAS), located in the parietal lobes, and to the Mnemonic Comparator (Cm), located in the central executive system. The CAS enables the person to consciously experience the event, whilst the Cm compares the individual's current memory performance with their pre-existing knowledge of their state of memory functioning, held within the Personal Knowledge System (PKS) of semantic memory. The person becomes initially aware of any mismatch in this information by the link between the Cm, episodic memory and Cognitive Awareness System (CAS). However, an enduring awareness of the change in memory function is only achieved by the Cm updating the PKS in semantic memory.

Agnew and Morris (1998) suggested that impairments in this system could result in three distinct types of anosognosia within the AD population:

1. *Mnemonic anosognosia.*

A deficit in the pathway between the Cm and semantic memory results in the person being unable to update their PKS. Thus, they show initial insight into their memory functioning after completing the task, but are unable to create an enduring awareness of their memory functioning. However, due to the intact pathway between the Cm and implicit memory, they may exhibit subconscious alterations in their emotional/behavioural reactions, whilst continuing to deny their memory impairments.

2. *Executive anosognosia.*

A deficit in the Cm results in the person being unable to compare their memory performance with their PKS, leading to a complete lack of awareness of their memory deficit. As no information is sent to the implicit memory, the person will not alter their behaviour even at a subconscious level. Although, theoretically it is possible that the person could still obtain knowledge of their performance via a link between their episodic and semantic memory, this knowledge would not have a value judgement component. According to Agnew and Morris's (1998) model, this would not constitute awareness as the person would not be aware that their performance had changed in relation to their own performance or in comparison to others'.

3. *Primary anosognosia.*

An impairment of the cognitive awareness system results in unawareness of the state of functioning in all domains, not just memory. However, implicit learning is retained.

Whilst there has been little direct research evaluating this model, there is tentative evidence to suggest that mnemonic anosognosia may be particularly common in people with early-stage AD. This would be in keeping with our knowledge that medial temporal lobe and hippocampus pathology is associated with the early stages of AD and that memory (particularly new learning and retrieval of recently learnt information) is one of the first cognitive functions to become impaired in clients with AD (e.g., Baddeley, Bressi, Della Sala, Logie, & Spinner, 1991; Braak & Braak, 1991; Nagy et al., 1999). As the pathology spreads to include frontal and parietal structures one would theoretically expect executive and primary anosognosia subtypes to be come more apparent in the latter stages of AD .

Mnemonic anosognosia is of particular clinical interest as, theoretically, it may prove amenable to techniques designed to improve awareness through repeated exposure, thus facilitating rehabilitation outcome (Schlund, 1999).

In particular, initial evidence for this type of anosognosia comes from a study by Moulin, Perfect and Jones (2000). They compared the ability of an early-stage AD group and an Older Adult Control (OAC) group, to predict their performance before and after encoding lists of to-be-remembered words. They found that, while there were differences in the AD and OAC groups' accuracy for their pre-study

predictions, both groups were equally accurate after encoding. They argued that this suggested that people with early-stage AD do have initial awareness when faced with their performance on memory tasks, but that they do not achieve an enduring record of their experience. Thus, this provides tentative evidence that people with early-stage AD may be particularly prone to difficulties in modifying their self-evaluations, because they have difficulty retaining information about the discrepancy between current and past performance, "they forget they forget". These findings are in keeping with Agnew and Morris's (1998) mnemonic anosognosia.

However, as Moulin and colleagues (2000) were not evaluating Agnew and Morris' (1998) model directly, their study does not provide conclusive evidence for this theory. Firstly, their study only evaluated implicit memory, as they did not explicitly ask participants if they performed as they expected. This does not fully distinguish between mnemonic anosognosia and primary anosognosia. Secondly, they did not evaluate whether participants with AD were able to generalise their task-specific awareness to their global awareness of memory functioning, which one would expect if the participants were suffering from mnemonic anosognosia. Finally, it would have been useful if they had investigated whether any improvement in awareness diminished over time. Such a finding would have significant implications for future rehabilitation approaches, as well as the evaluation of Agnew and Morris' (1998) model.

The present study was a development of Moulin and colleagues' (2000) experimental method. The aim of the study was to investigate the existence of mnemonic anosognosia within a group of people within the early stages of AD.

This was achieved by exploring whether participants with AD were able to improve their implicit and explicit awareness of their performance on a memory task following task exposure; whether this was accompanied by a change in their global memory functioning awareness; and how stable any change in awareness was over time.

Given that memory has been found to be one of the first cognitive functions affected by AD (e.g., Baddeley et al., 1991; Braak & Braak, 1991; Nagy et al., 1999;), it was anticipated that people with early-stage AD would be less accurate than OACs in predicting their performance on a word recall task. In addition, it seemed likely that the individuals with AD would become more accurate in predicting their performance after exposure to their memory ability on the recall task, as indicated by the findings of Moulin and colleagues (2000). Furthermore, in keeping with Agnew and Morris' (1998) model, which suggested individuals with mnemonic anosognosia would have explicit awareness of their memory impairment when reminded of it, it was expected that any changes in the accuracy of participants with AD prediction of their performance would be correlated with explicit judgements of their ability. Similarly, it could be predicted that the participants with AD would also show an initial improvement in a measure of global memory awareness following exposure to memory testing. Finally, it was hypothesised that any changes in global awareness and prediction accuracy exhibited by the participants with AD, would not be retained after a delay period.

Method

Design

The study involved a mixed research design incorporating an early Alzheimer's Disease (AD) group and a healthy Older Adult Control (OAC) group. Between group comparisons of recall, predicted recall, judgement of accuracy of predictions, global awareness ratings and executive functioning were made, as well as within group comparisons on repeated measures of accuracy of predictions and global awareness ratings. The independent variables under investigation were cognitive impairment and time at testing of memory awareness. The dependent variables were accuracy of memory performance predictions, judgement of accuracy of predictions and global awareness ratings.

Participants

In total, there were 36 participants in the study. An early-stage AD group consisted of 18 participants recruited from a hospital-based memory clinic who had received a diagnosis of probable AD according to NINCDS-ADRDA (McKhann, Drachman, Folstein, Price & Stadlan, 1984) and DSM-IV (American Psychiatric Association, 1994) criteria. The diagnosis process involved client and carer interviews and extensive cognitive, medical, neurological, psychiatric and laboratory assessments. As the study was focusing on older adults in the early-stages of AD, only those individuals with a Mini Mental State Examination (MMSE) score of 18 or above (Folstein, Folstein & McHugh, 1975) and who were 60 years old or over were included in the research.

The OAC group also comprised 18 participants who were recruited from the friends and relatives of the early AD participants. All the OAC participants had a MMSE score of over 24. The OAC group was recruited selectively so as to maximise consistency between it and the AD group in terms of the demographic variables of gender, age, pre-morbid IQ and years in education.

Due to possible confounding effects of depression, participants were excluded if they scored at, or above, the cut off score of 7 on the Short Geriatric Depression Scale (S-GDS: Sheikh & Yesavage, 1986). This scale has been validated for use with individuals with mild to moderate dementia (Katz, 1998). Furthermore, participants were excluded if they were suffering from any other medical or neurological condition likely to interfere with their cognition, or were on medication likely to affect cognitive performance. Those who had uncorrected hearing or sight problems were also excluded from the study.

Recruitment

Potential early-stage AD group participants were identified by a search of the clinical notes at the memory clinic (Appendix 1). The consultants of patients that appeared to meet the study criteria were then approached about the person's suitability for research. Those participants considered suitable were then approached via an introductory letter from their consultant at the memory clinic (Appendix 2), enclosing an information sheet regarding the research (Appendix 3). This was then followed up by a telephone call from the author. If the person expressed an interest in taking part, an appointment was made to visit them at their home. During this visit they were informed of their rights as a research participant

and were asked to sign a consent form (Appendix 4). Where necessary, a friend or relative of the participant with AD was also approached, given an information sheet about the research (Appendix 5) and asked to sign an assent form (Appendix 6).

OAC participants were recruited from the relatives and friends of the early-stage AD participants. They were initially approached by the AD participant and followed up by a telephone call from the author. An information sheet about the research was provided (Appendix 7). If they expressed an interest in taking part in the research, an appointment was made and consent sought.

Twenty-five people declined to take part in the study. In cases where an explanation was given, the main reasons were: being too busy; already taking part in a research project: and, a concern that they would find participation distressing. There were no drop-outs from the research, but one AD participant was excluded due to their self-report of possible depression on the S-GDS and another participant with AD was excluded due to significant eyesight problems.

Materials

Three word lists (Appendix 8), each consisting of 10 words, were constructed for the study. The lists were matched on word length (List 1 vs List 2, t [9] = -.25, p = .81; List 1 vs List 3, t [9] = -.34, p = .74; List 2 vs List 3, t [9] = -.19, p = .85) and ease of recall (List 1 vs List 2, t [9] = .83, p = .42; List 1 vs List 3, t [9] = .47, p = .65; List 2 vs List 3, t [9] = -.35, p = .73), as specified by Rubin and Friendly (1986). Words were presented individually on card and were written in 2cm high black letters on a white background. In addition, a visual prompt was used for the

judgement of performance scale, which consisted of a scale printed in bold print on a piece of A4 paper (Appendix 8).

Measures

Measures of awareness of memory functioning.

i) Awareness of Memory Impairment Scale (AMIS; Sevush, 1999).

The AMIS is a simple six-item scale asking individuals to give "yes" or "no" answers to questions about the presence of memory deficits (Appendix 8). The items are based on factors that are characteristic of AD and so any denial of a factor is considered to be attributable to a lack of awareness for that memory factor. The AMIS generates absolute scores and does not rely on informant comparisons. The test has been assessed to have good test-retest reliability ($r = .91$) after a 30 minute delay period and good construct validity (Sevush, 1999).

ii) Awareness of Memory Impairment Scale - Informant version (AMIS-I; Sevush, 1999).

Although no informant version is strictly needed for the administration of the AMIS, it was felt that incorporating an informant version into the study would provide a further measure of awareness for the AD group. The six-item scale was therefore adapted into an informant version (Appendix 9).

iii) Pre and post-study awareness task (Moulin et al., 2000)

This method of assessing awareness involves asking participants to predict their performance on a subsequent word recall task, at pre- and post-studying stages of the trial. Participants' insight is then established by comparing their predicted

performance to their actual performance. A greater deficit between the two scores suggests a greater impairment of memory awareness. The advantage of this method is that it takes into account the fluid nature of insight and can assist in establishing the participant's level of insight given an opportunity to monitor the task demands.

(iv) Judgement of performance scale (devised for the study)

This consisted of a 5-point scale (Appendix 10). Participants were asked to rate their performance on the scale by rating how well they thought they performed in relation to their prediction. Responses were then coded as 2 for "much worse than expected", 1 for "worse than expected, 0 for "as expected", -1 for "better than expected", -2 for "much better than expected".

National Adult Reading Test (NART: Nelson & Wilson, 1991)

The NART is an oral, single word, reading test consisting of 50 words that violate graphemephoneme correspondence rules. Performance on this test correlates highly with Full Scale IQ as measured by the Wechsler Adult Intelligence Scale – Revised (Wechsler, 1981) and is therefore used to predict premorbid IQ. The NART has been shown to be robust in the face of most neurological and psychiatric illness and has been widely used to estimate pre-morbid intelligence. It has good validity and reliability (Nelson & Wilson, 1991).

The Executive Clock Drawing Task (CLOX; Royall, 1995).

The CLOX is a clock drawing task that has been specifically designed to assess executive impairment and discriminate it from non-executive impairment. It has been used widely with individuals with dementia and has good validity and

reliability (Royall, Cordes & Polk, 1998). The CLOX is divided into two parts. The first part involves assessing the participants' performance in a novel and ambitious situation by asking them to draw a clock showing the time 1.45. The second part of the test assesses visuospatial skills and involves the participant copying a clock drawn by the researcher. Individuals with dysexecutive functioning are expected to perform better on the second task than the first task.

Procedure

In most cases participants were visited at their homes by the researcher, with the exception of two cases who were seen, on their request, at the Memory Clinic. To ensure consistency the researcher used a script to administer the research procedure (Appendix 11).

The AMIS was administered by the researcher to establish a pre-testing rating of global awareness for memory functioning. Participants were then informed that there would be a 10-word free recall test and asked to predict how many of the words they would recall. Participants were then shown the words one at a time, for a duration of 2 seconds each and asked to read them aloud. After exposure to the words, participants were, again, asked to predict the number of words they would be able to recall. They were asked not to count up the words they could currently recall, but intuitively to predict their level of recall[1]. Participants were then immediately asked to recall the words. Afterwards, participants were requested to rate their performance on the five-point judgement of performance scale, as a means

[1] In contrast to Moulin et al. (2000) study, no additional distracter task was used, as it was felt that asking participants to predict their performance after studying would itself act as a distracter task, and a second distracter task would detract from the study's ecological validity.

of assessing their explicit awareness. The wordlist task was then repeated with two subsequent lists, with the wordlist presentation order counterbalanced across participants.

Following completion of the third wordlist task, the researcher re-administered the AMIS to assess for any change in participants' levels of global memory awareness. This was followed by a 20-minute delay period, during which time the participants completed the National Adult Reading Test (NART), as a means of estimating their pre-morbid level of intelligence (Nelson & Willison, 1991) and answered informal socio-demographic questions. Any additional time was filled with conversation with the researcher, which was directed away from the topic of memory functioning and consisted of such topics as holidays, family or other personal interests.

At the end of the delay period the researcher re-administered the AMIS and then asked the participant to predict how many words they would recall if they were given a subsequent word recall task. This was designed to establish whether participants changed their insight into their memory ability after a period in which they were not concentrating on memory tasks. However, no subsequent test was given. The MMSE and CLOX tasks were then completed, followed by participant debriefing. In addition, the carers of participants with AD were asked to complete informant versions of the AMIS. These were either completed during the visit to the AD participant's home or returned by post at a later date if the informant was not present.

Due to the vulnerability of cognitively impaired individuals, care was taken in monitoring the participants' verbal and non-verbal signs of distress throughout the data collection stage (Berghmans & Termeulen, 1995).

Data Analysis

Statistical analysis was conducted using SPSS 11.0 for Windows (2001). In order to assess the suitability of the data for parametric statistical analysis, goodness of fit tests were conducted to ensure that data met the assumptions of normal distribution. The majority of the variables were normally distributed, with the exception of the OAC group's List 1 pre-study predictions and List 2 and 3 judgement of performance scores. Non-parametric tests (Spearman's rank correlations and Wilcoxson signed rank tests) were used for analysis incorporating these variables. The rest of the data were analysed using independent t-tests and analyses of variance. Analysis at the level of .05 was used throughout. As with similar studies, in order to analyse the accuracy of predictions on the wordlist tasks, absolute and actual accuracy scores were calculated from the discrepancy between participants' predictions and their actual recall on each list.

Results

Demographic Data

There were 18 participants within the AD group and OAC group; consisting of 6 men and 12 women in each. Their descriptive details are shown in Table 1.

Insert Table 1 Here

The two groups did not significantly differ in their Age, t (34) = 1.49, p =.15; Years of Education, t (34) = .46, p =.65; level of depressive symptomatology (S-GDS), t (34) = 1.24, p =.22; or predicted IQ (NART), t (34) = -.79, p =.43.

Implicit awareness – word list trials

Figure 1 shows the mean pre-study prediction, post-study prediction and the actual recall over the three wordlist trials for the AD and OAC groups.

Insert Figure 1 Here

Recall.

As would be expected, a repeated measures ANOVA of List (1 / 2 / 3) by Group (AD / OAC) for the number of words recalled, indicated that there was a significant difference in the recall of the two groups, F (1,34) = 37.51, p = .00, with the AD group performing significantly worse. There was also a significant effect of List in the amount of words recalled, F (2, 68) = 5.96, p =.00, and a significant interaction between List and Group, F (2,67) = 6.45, p =.00. However, a simple main effect analysis indicated that the effect of List was only significant for the OAC group, F (1,17) = 11.09, p =.00, and paired sample t-tests (alpha adjusted for multiple comparisons) suggested that this was due to an initial decline in the mean number of words recalled from the first list trial to the following trials (List1 vs List2, t [17] = 3.62, p =.00; List2 vs List3, t [17] = 0.53, p =.60; List1 vs List3, t [17] = 4.78, p =.00). This can clearly be seen in Figure 1. As the lists were counterbalanced to avoid ordering effects, this finding is likely to represent motivational effects caused by the repetition and simplicity of this task for the OAC group.

Accuracy of predictions.

Two absolute accuracy scores were produced per list, one for pre-list prediction and one for post-list prediction (Figure 2).

Insert Figure 2 Here

A 3-way repeated measures ANOVA of Group (AD / OAC), by Prediction Type (pre-list / post-list), by List (1 / 2 / 3), was calculated based on the absolute accuracy scores. As expected, this revealed a significant main effect of Group, F (1,34) = 11.16, p =.00, suggesting that the participants with AD were less accurate overall. There was also a significant main effect of List, F (2,68) = 5.26, p =.01. Further analysis using tests of within-subjects contracts suggested that participants were significantly less accurate in list 1 than list 2, F (1, 34) = 10.13, p = .00, and list 3, F (1, 34) = 7.42, p = .01; with no significant difference in accuracy between the list 2 and list 3 trials, F (1, 34) = .26, p = .61. The List by Group interaction was not significant, F (2,68) = .34, p =.72, which suggested that both groups exhibited this pattern of performance across lists.

In addition, there was a significant main effect of Prediction Type, F (1,34) = 8.13, p =.01, and examination of Figure 2 indicates that overall the participants made less accurate pre-study predictions than post-study predictions. Furthermore, the Prediction Type by Group interaction was significant, F (1, 34) = 4.48, p =.04. This was the result of a significant effect of prediction type in the AD group, F (1,17) = 8.34, p =.01, but not in the OAC group, F (1,17) = .52, p =.48. This suggests that the participants with AD significantly revised their predictions after studying the words, whereas the OAC group, being more accurate, did not. Simple main effects were also used to explore the relationship between Group and Prediction Type on the accuracy of predictions over the wordlist trials. These suggested that there was a significant difference between the groups in the accuracy of both their pre-study predictions, F (1,34) =14.92, p =.00, and their post-study predictions, F (1,34) = 7.23, p =.01, with AD participants remaining less accurate over time.

In addition, the List by Prediction Type interaction approached significance, F (2,68) = 3.12, p = .07, sphericity not assumed. Overall this analysis indicated that participants became more accurate in their predictions, both pre and post-study, over the three lists and this was true for both groups, as evidenced by the failure to find a significant 3-way (Group by Prediction Type by List) interaction, F (2,68) = 2.39, p =.10. Overall, this analysis suggests that AD participants were acting in a similar way to the OAC participants, but at a less efficient level, requiring them to make greater revisions of their predictions after study.

However, the difficulty with using absolute scores is that any group difference in the direction of prediction accuracy might be obscured. Given that it could not readily be assumed that over and under predictions were theoretically equivalent forms of impaired awareness, a further analysis of the actual accuracy scores was also completed. (Figure 3).

Insert Figure 3 Here

A 3-way repeated measures ANOVA of Group (AD/OAC) by Prediction Type (pre-list / post List) by List (1 /2 /3) was calculated based on the actual discrepancy scores. Consistent with the ANOVA performed on the absolute accuracy scores, there was a significant main effect of Group, F (2, 68) = 15.11, p =.00, and Prediction Type, F (2, 68) = 4.23, p =.048, but a non significant List by Prediction Type interaction, F (2, 68) = 1.94, p =.16, sphericity not assumed. These results

support the finding that the AD group was less accurate overall and that both groups became more accurate after being given the opportunity to study the words.

However, in contrast to the previous absolute analysis, a significant interaction was found for the List by Group interaction, $F(2, 68) = 5.65, p = .01$. Further analysis of this interaction using within subjects contrasts suggested that the AD group's and OAC group's accuracy of predictions was different over the three lists, with a significant difference in their performance between lists 1 and 2, $F(1, 34) = 8.79, p = .01$; and lists 1 and 3, $F(1, 34) = 7.33, p = .01$; but no significant difference in the groups' performance between lists 2 and 3, $F(1, 34) = .03, p = .82$. In addition, contrary to the absolute analysis, there was a significant 3-way interaction of List by Prediction Type by Group, $F(1.55, 52.60) = 6.88, p = .00$. Within subjects contrast analysis suggested there was a significant difference in the two groups' pre- and post-study accuracies between lists 1 and 2, $F(1, 34) = 7.30, p = .01$; and lists 1 and 3, $F(1, 34) = 9.43, p = .00$, but no significant difference in the groups' pre- and post-study accuracies between lists 2 and 3, $F(1, 34) = .70, p = .41$. This implies the AD group's and OAC group's accuracy of predictions were different over the three lists and before and after studying the words. Participants with AD tended to over-estimate their ability, whilst OAC participants tended to under-estimate their ability prior to studying the words in the first trial.

Furthermore, there was found to be no significant main effect of List during this analysis, $F(2, 68) = .10, p = .91$. This is likely to be due to the effect of averaging between the AD participants' mean positive accuracy score and the OAC participants' negative accuracy score for the first list.

Delay.

Three Wilcoxon Signed Rank tests were calculated to assess the prediction that after a delay period the AD group's accuracy of their predictions would decrease to a level consistent with their pre-testing prediction. A significant difference was found between the List 1 pre-study prediction and the List 3 pre-study prediction, z (N = 18) = -2.17, p = .03. This suggested that the participants' predictions were significantly revised after exposure to the memory tasks. A borderline significant trend was found for the difference between the List 1 pre-study prediction and the Delayed List prediction, z (N = 18) = -1.94, p = .05. Finally, there was no significant difference between the List 3 pre-study prediction and the Delayed List prediction, z (N = 18) = -1.66, p = .10. Thus, whilst the AD participants' predictions after a delay period exhibited a moderate decline in accuracy, they did not reset to their level prior to task exposure, instead they remained consistent with the predictions made after exposure to the tasks.

For the OAC group, as anticipated, no significant differences were found either between List 1 and 3 pre-study predictions, z (N = 18) = -1.90, p = .06; List 1 pre-study and delay predictions, z (N = 18) = -1.90, p = .06; or List 3 pre-study predictions and delay predictions, z (N = 18) = .00, p = 1.00.

Explicit Awareness – Judgement Scale

In order to assess the hypothesis that there would be a positive relationship between the AD group's accuracy in predicting the number of words they would recall and their explicit awareness of their performance, the participants' explicit judgements

of the accuracy of their predictions were analysed in comparison to the actual accuracy of their predictions. Figure 4 depicts the mean actual accuracy scores for participants grouped by the explicit judgement they made for each list (i.e., "I did much better than I expected"......"I did much worse than I expected").

Insert Figure 4 Here

There was a significant correlation between the AD group's explicit judgement ratings and their actual accuracy scores, r_s (52) = .55, p = .00.

As would be expected, the OAC group also exhibited a significant correlation between their explicit judgement ratings and the actual accuracy score, r_s (52) = .70, p = .00.

Given the AMIS test-retest reliability of .91 and a population standard deviation of 1.81, a change in AMIS score of 2 produced a reliable change index score of 2.61, which was significant at the .05 level according to the method described by Jacobs and Truax (1991). Based on this calculation, further analysis of the relationship between the accuracy of predictions and participants' explicit awareness was conducted by dividing the AD group into those who revised their AMIS ratings after exposure to memory testing (AMIS Change group = a discrepancy between pre- and post-testing AMIS scores of 2 or more), and those who did not (AMIS No Change group = a discrepancy between pre- and post-testing AMIS scores of 0-1). The correlation between the explicit judgement of their prediction accuracy and

their actual prediction accuracy was then explored for each group. This suggested that both groups exhibited a significant correlation between these two factors, but that this relationship accounted for far greater variance within the AMIS Change group, r_s (25, n =9) = .73, p = .00, than the AMIS No Change group, r_s (25, n = 9) = .52, p = .01. However, this did not constitute a significant difference in the strength of the correlations between the two groups, Z = .61, n^1 = 9, n^2 = 9, p>.05. This lack of significant result may have been due to the small sample size.

Global Awareness - AMIS

Figure 5 shows the AMIS scores recorded by informants (AD only) and by participants, prior to and post testing, on the lists and after the delay period.

Insert Figure 5 Here

A series of paired t-tests were conducted in order to investigate the hypothesis that the AD group increased in global awareness after exposure to memory trials, but that this was not retained after a delay. A comparison of the pre-test and post-test AMIS scores suggested they were significantly different, t (17) = -2.67, p = .02. However, there was no significant difference between the pre-test and the delay scores, t (17) = -.89, p = .39, or between the post-test and the delay scores, t (17) = 1.29, p = .22. No significant differences were found between the pre-test, post-test and delay AMIS ratings for the OAC group. Thus, this suggests that the AD participants appeared to revise their AMIS ratings after exposure to memory testing.

However, although there was a decline in their AMIS ratings after exposure to memory testing this did not return to pre-testing levels, suggesting that, consistent with the prediction accuracy data, the AD participants retained much of the awareness of their difficulties gained by the exposure to testing after a brief delay.

Insert Figure 6 Here

A descriptive analysis of the individual questions of the AMIS (see Figure 6) suggested that there was some variation in the revision of responses to individual questions. The AD group showed less awareness for questions 2 and 4 overall, which related to remembering names and word finding difficulties. While question 5 (Do you forget where you have put something?), showed the least variation over trials. Question 3 (Is your memory as good as ever?) was the only question for which the percentage of the AD group who rated it as a problem, after exposure to the memory tasks, was equivalent to the ratings of informants. This rating, however, returned to the pre-exposure to memory tasks level, after the delay.

Informant-patient discrepancies

AMIS informant-patient discrepancies were used to investigate the hypothesis that AD participants with retained global awareness of their memory ability, would be more accurate in predicting their performance on a specific memory task than those with impaired global awareness. In order to assess this, the AD group was divided into a Globally Aware group and an Impaired Global Awareness group. Participants were considered globally aware if their pre-testing AMIS score was consistent with

their informant's ratings of their memory (i.e., within the range of plus or minus 1), and globally unaware if these score were different (i.e., discrepant by plus or minus 2, or more). A comparison of the two groups indicated that they were significantly different in the accuracy of their predictions, t (52) = 2.05, p = .046, with the Globally Aware group (n = 7) significantly more accurate than the Impaired Global Awareness group (n = 11).

Executive functioning

Executive Control Function (ECF) scores for the AMIS Change and AMIS No Change groups were compared to determine whether executive functioning differentiated the groups. Despite apparent differences in means (AMIS Change: *M* = 3.00, *SD* = 2.55, range = -1-11; AMIS No Change: *M* = 4.89, *SD* = 3.98, range = 0-7), no significant difference between the groups was found when analysed using an independent t-test, *t* (16) = -1.21, *p* =.245.

However, it is possible that the AMIS No Change group also incorporated AD participants with retained awareness who, logically, would not change their global awareness ratings with exposure to memory tasks and may not have executive impairments. The large standard deviation and range of ECF scores within this group may evidence this. Unfortunately, due to the small sample size, it was not possible to exclude those participants with retained insight from the analysis.

Further Analysis of Awareness Groups

It was felt that a simple AMIS change/no change division was not adequate to account for the heterogeneous nature of awareness in this sample. Therefore, further

analysis of the data was completed by sub-dividing both the AMIS Change and AMIS No Change groups based on whether their pre-testing AMIS ratings differed from their informant's ratings. It was envisaged that by splitting the data in this way, it allowed for analysis of sub-groups of awareness types, differentiating those who were fully aware and as a result did not change their AMIS ratings after exposure to memory tasks, from those who were unaware of their impairments initially and did not become more aware after testing. Four groups were therefore formed: a Fully Aware group, who exhibited initial awareness (consistent informant-patient pre-testing AMIS ratings) and did not change their AMIS scores from pre- to post-testing; a Variable Awareness group, who exhibited initial awareness (discrepant informant-patient AMIS scores), but also exhibited improved awareness following testing (discrepant pre- and post-testing AMIS scores); a group consistent with mnemonic anosognosia, who lacked initial awareness (discrepant informant-patient pre-testing AMIS scores), but did show improved awareness with exposure to memory testing (discrepant pre- and post-testing AMIS scores); and, finally, a group who exhibited a consistent form of anosognosia, who did not show initial awareness (discrepant informant-patient AMIS scores), nor any improvement in global awareness following memory testing (consistent pre- to post-testing AMIS scores).

Insert Table 2 Here

As the numbers in the awareness sub-groups were small, only descriptive analysis was carried out. Table 2 shows the mean and range of ECF scores for each awareness group. The Consistent Anosognosia group exhibited the highest ECF

scores, suggesting they were more prone to impairments in executive functioning than the other sub-types. However, it should be noted that the standard deviation and range for this group's ECF scores was large, indicating a high degree of variability. The Variable Awareness group also had a relatively high mean ECF score, but with a much lower standard deviation than the Consistent Anosognosia group. The Mnemonic Anosognosia group exhibited the lowest mean ECF score out of the four groups, indicating lower levels of executive functioning impairment. Interestingly, the mean ECF score for this group was lower than the Fully Aware group's ECF score.

Discussion

This study investigated Agnew and Morris' (1998) model of mnemonic anosognosia by exploring the dimensions of impaired insight exhibited in a sample of people with early-stage AD. Four main dimensions of awareness were evaluated: implicit awareness; explicit "task specific" awareness; explicit awareness; and stability of awareness over time.

Implicit awareness

Consistent with previous research (e.g., Green, Goldstein, Sirockman & Green, 1993; McGlynn & Kaszniak, 1991; Ott et al., 1996), those participants with early-stage AD were less accurate than OAC participants at predicting their performance on a memory task. Indeed, when the actual accuracy scores were examined, the OAC showed an initial tendency to under-estimate their ability, whilst the AD participants tended to over-estimate their ability. Even so, in keeping with Moulin and colleagues' (2000) study, participants with AD were able significantly to revise their predictions after task exposure. However, in contrast to Moulin and associates' (2000) findings, participants with AD did not revise their post-study accuracy of predictions to a level consistent with the OAC participants. Furthermore, although the improvements in awareness were retained for a third memory task, the AD participants did not exhibit continued gains in awareness. Thus, these findings suggested that the participants with AD exhibited improvements in their implicit memory awareness following task exposure, but that continued exposure to their memory impairments did not result in a level of awareness consistent with the OAC participants. It is unclear why the present study did not fully replicate the findings

of Moulin and associates (2000). However, it is possible that this reflected Moulin and colleagues' (2000) use of an additional distracter task between the encoding and recall aspects of the task. This was not included in the present study, because it was felt that the post prediction task was an adequate distracter task and that an additional distracter task would detract from the ecological validity of the research. As such, it is possible that the inclusion of a second distracter task in Moulin and colleagues' (2000) study measured a different aspect of metacognition. Further research would be beneficial in exploring this difference in results.

Explicit "task specific" awareness

As anticipated, both groups exhibited a significant correlation between the implicit measure of awareness (the accuracy of predictions) and the explicit judgement of their accuracy of prediction. This suggested the participants with AD were able to adjust their awareness of memory functioning, both at implicit and explicit levels. Furthermore, when the AD group was divided according to whether they revised their AMIS rating after exposure to memory testing, the group that revised their ratings exhibited a stronger correlation between the implicit and explicit awareness measures, which was consistent with the strength of the correlation found for the OAC group. However, the difference in the group's strength of correlation did not reach clinical significance. It is likely that this was due to the small sample size and the inclusion of fully aware participants in the group that did not revise their AMIS ratings.

Overall, these findings are in keeping with Agnew and Morris' (1998) model of mnemonic anosognosia and differentiates the possible mnemonic anosognosia, exhibited by this group, from primary anosognosia (Agnew & Morris, 1998).

Explicit global awareness

Consistent with previous research (e.g., Ott et al., 1996; Vasterling et al., 1995), participants with AD significantly under-reported their global memory problems. However, in keeping with their task specific awareness performance, participants with AD exhibited a significant improvement in their global awareness ratings post-testing. This suggested that AD participants were able to generalise these improvements in task-specific explicit awareness to a more general understanding of their memory functioning. Indeed, this was further substantiated by informant-patient discrepancy assessments of global awareness. Those participants with AD, whose pre-testing AMIS ratings differed from their informant's, were significantly less accurate at predicting their performance on the memory task than those who gave AMIS ratings consistent with those of their informant. Such findings are in keeping with Neundorfer's (1997) observations that anosognosia fluctuates over time.

Interestingly, there was also tentative evidence to suggest that the memory task exposure resulted in different degrees of revision for individual items on the AMIS, with questions referring to general memory ability being more sensitive to change than more specific memory domain questions. This is an interesting finding, which requires further investigation. Such a finding suggests that, while exposure to specific memory tasks may help improve awareness of general memory functioning,

people with early-stage AD may find it difficult to apply this knowledge to their performance on other specific memory tasks.

Stability over time

Although participants with AD exhibited a decline in both their accuracy of predictions and their ratings on the AMIS after a 20-minute delay period, these declines were not significant and did not return to pre-testing levels. This suggested that participants with AD retained much of their improvements in task-specific and global memory awareness after a brief delay. Therefore, these findings do not provide full support for Agnew and Morris' (1998) model of mnemonic anosognosia, where one would have expected insight to reset after a delay period, due to the inability of the individual to update their PKS in their long-term semantic memory. However, given the downward trend in task-specific and global memory awareness following the delay period, a longer delay period may have resulted in further decline in the accuracy of the delay predictions and its return to pre-testing levels. Furthermore, despite moving on to non-memory tasks and avoiding discussions on the subject of memory during the delay period, it is possible that, because the participants did not leave the testing situation, the environment continued to cue the person about their memory impairments. This may have affected the speed with which awareness returned to pre-testing levels (Tulving, 1983).

Executive functioning

Although not a main aim of the research, the relationship between executive functioning and the AD participants' ability to shift their global awareness was

explored. Executive functioning, alone, could not adequately account for the differences between the participants who changed their global memory awareness ratings post-testing and those who did not. However, it is possible that this was due to participants with full awareness being included in the "no change" group, thus obscuring any relationship.

Further analysis of the data suggested that there were four types of impaired awareness within the AD group: a Fully Aware group, who were initially aware and, as a result, did not change their AMIS ratings post memory testing; a Consistent Anosognosia group, who lacked initial awareness and did not improve their awareness after memory testing; a Mnemonic Anosognosia group, who lacked initial awareness, but who improved their awareness following exposure to memory testing; and a Variable Awareness group, who were initially considered aware, but exhibited further shift in their awareness following exposure to memory testing. Unfortunately, due to the small sample size it was not possible to evaluate these statistically. However, there was tentative evidence to suggest that the Consistent Anosognosia group had the greatest impairments in executive functioning. Whilst this would be in keeping with executive anosognosia, it should be noted that the range of ECF scores for this group was large, which may suggest that this group incorporated a number of awareness types. Indeed, both executive and primary anosognosia would, theoretically, show profiles in keeping with this subgroup, yet have markedly different performance on tests of executive functioning. In further support of Agnew and Morris' (1998) model, those who fitted the profile of mnemonic anosognosia had the least evidence of frontal impairment out of the four sub-groups, which, interestingly, was less than the group considered fully aware.

However, the Variable Awareness group was more difficult to account for as it exhibited relatively high scores on the test of frontal impairment, despite showing a shift in awareness scores post memory testing. As such, this group is harder to explain using Agnew and Morris' (1998) model of anosognosia sub-types. It is possible that the executive functioning measure was tapping into an executive skill (planning) that was not related to the type of executive functioning performed by the Cm (self-monitoring) and this led to the recorded high ECF scores in the presence of a shift in AMIS scores post testing. However, it is also possible that whilst this subtype involved damage to the Cm, so that a comparison between current and past functioning was not possible, knowledge about one's performance was able to pass directly from the episodic to semantic memory. Such a link is not accounted for by Agnew and Morris's (1998) model.

Although this provisional analysis provides some interesting tentative findings, further research, with a larger sample size, is needed to evaluate these subtypes more robustly.

Summary and theoretical implications

Overall, the present study supports previous findings that awareness is a non-unitary phenomenon that varies both between and within individuals (e.g., Derouesne et al., 1999; James & Moulin, 2002; McGlynn & Kaszniak, 1991). The results indicated that a significant proportion of people with early-stage AD were able to improve their awareness following exposure to their memory impairments. This improvement in awareness was evident at implicit, explicit task-specific and explicit global levels, and suggested that a significant proportion of early-stage AD

participants were able to demonstrate a transition from task-specific to general awareness of memory functioning. Whilst this improvement in memory awareness was retained after a 20-minute delay period, there was evidence to suggest that with a longer delay period the participants with AD would have shown further decline in their awareness, which may have resulted in the resetting of their awareness to its original level. Such findings are, on the whole, consistent with Agnew and Morris' (1998) model of mnemonic anosognosia, which stipulates that people with damage to the connection between the CES and PKS would not be able to update their long-term personal knowledge of their memory functioning, resulting in only a temporary improvement in awareness. This was observed in a significant proportion of the early-stage AD participants in this study and was further supported by evidence that the sub-group of participants, who fitted this profile, had the lowest executive impairment scores.

However, this is not to say that mnemonic anosognosia is the only type of anosognosia found within the early-stage of AD. Certainly, in keeping with previous research (e.g., Derouesne et al., 1999; De Vreese et al., 2001), there was evidence to suggest that some participants with AD had retained their awareness, whereas others had a relatively dense anosognosia that was resistant to revision following exposure to their memory impairments. Further investigation of these sub-types is needed to clarify the dynamics of awareness found within this population. The different sub-types may, in particular, explain the inconsistent findings in the research regarding the area of neurological damage associated with anosognosia, with some types being due to damage to the pathway between memory and the CES, and others associated with frontal, or right hemisphere parietal

damage. However, Agnew and Morris's model of memory awareness does not fully account for all of the possible subtypes found within this study. In particular it did not fully account for the variable anosognosia type, which may be indicative of a link between the episodic and semantic memory processes in the memory awareness system. In addition, it is possible that the deficits in insight reflect damage to more than one process within the memory awareness system. Certainly, given the diffuse nature of pathology found within the AD population; it is likely to be difficult to find pure damage to key awareness areas (e.g., memory, central executive and parietal). The differentiation of executive and primary anosognosia is likely to be particularly problematic as damage to the frontal and parietal lobes is likely to occur simultaneously and in conjunction with temporal lobe changes, in moderate to severe cases of AD. Recruiting samples of participants with dissociatively different pathologies would be advantageous in investigating these subtypes more robustly, but given the small sample sizes within this study, it was not possible to make any firm conclusions about the possible subtypes identified. However, they do provide some interesting challenges for further investigation.

Of additional interest, was the finding that the OAC participants exhibited initial under-estimates of their performance, whereas the AD participants (including those considered to be fully aware of their memory functioning) tended to over-estimate their performance. Whilst this is consistent with the findings reported in Ott and colleagues' (1996) study, this relationship has largely been overlooked in the research literature due to the use of absolute score analysis (e.g., Moulin et al., 2000; Clare 2002). As such, it may represent the role of social influences on the participant's self-reports, with the OAC individuals not wishing to appear overly

confident and the AD participants not wishing to admit to their impairments in front of a stranger. Thus, this is an important finding that is likely to have significant implications for our understanding of the biopsychosocial mechanisms underlying awareness and warrants further investigation.

Clinical implications

In the past it has been assumed that if a person has anosognosia, they are unlikely to benefit from psychological treatment to improve their memory functioning and psychological adjustment to AD. However, the findings from the current study suggest a distinct group of people with early-stage AD are able to improve their self-awareness following exposure to their memory impairments and that this is retained after a delay period. Although it is currently unclear how long such individuals are able to maintain this improved level of insight, such findings have important implications for the clinical management of people with AD and their carers. It is possible that by the provision of external feedback concerning the quality of their memory functioning, such clients may be helped to develop more accurate perceptions of their abilities and, as a result, obtain greater benefit from subsequent treatment (Green et al., 1993). Indeed, this has been supported by recent reports of clients with anosognosia who exhibit greater use of memory management strategies after structured exposure to their memory impairments (Schund, 1999; Koltai et al., 2001; Sohlberg, Mateer, Penkman, Glang & Todis, 1998). However, care should be taken in implementing such strategies, as in some cases impaired awareness may be providing a psychological defence against the stress of having AD (Clare, in press). Individualised assessments of awareness are likely to be important in guiding individual treatment approaches, as is in keeping

with the person-centred dementia care approach (Kitwood, 1997; Crosson et al., 1989).

Furthermore, there is increasing emphasis on the involvement of patients in the decision-making process and to keep them informed about decisions being taken on their behalf (Gove & George, 2001). If a person lacks awareness of their impairments it is likely to significantly affect their ability to do this (Becker & Kahana, 1993). However, if a person is able to improve their awareness, even if it is only for a limited period of time, it may provide clinicians and legal professionals with a window of opportunity in which it is possible to gain informed consent for legal and medical procedures. Indeed, according to the British Medical Association and Law Society (BMA/ Law Society, 1995) two of the key factors in determining a person's ability to give informed consent are their ability to understand, in broad terms, what the consequences are of not receiving the proposed treatment, and their ability to retain the information for long enough to make an informed decision. People with mnemonic anosognosia may be able to achieve this, given adequate preliminary input first to increase their awareness. Such knowledge can help maintain a person's control over their own life choices for as long as possible and counteract the infantilisation of people with AD.

Future research

This study highlights a number of useful areas for future research. In particular, it remains unclear as to how long improvements in awareness are maintained before returning to their pre-exposure setting. Research regarding this would have important implications both for the theoretical understanding of mnemonic

anosognosia, and the clinical management of clients with this type of difficulty. In addition, research is needed to further clarify the other sub-types of anosognosia found within the AD population and their relationship with cognitive factors, such as central executive functioning. Once this has been achieved, it will be important to establish whether these sub-types are common across other cognitive domains, such as awareness for activities of daily living.

Future studies will need to incorporate measures assessing different dimensions of awareness e.g., implicit, explicit, task specific, and global awareness. A significant number of past studies have not done this and, as a result, are likely to have been tapping into different anosognosia subtypes. This may well explain the high variance in past findings. Furthermore, prospective research may be particularly useful in determining the association of different subtypes of awareness with different stages in the progression of AD. Finally, there has been little exploration of the use of different rehabilitation approaches with the sub-types of anosognosia. This is likely to be imperative to the treatment of such clients and should be a high priority for future research.

A notable problem of all research into anosognosia is selection bias. It is possible that those with more severe anosognosia decline to take part in research because they are completely unaware of their memory problems and therefore do not see the relevance in taking part. This is a difficult issue to overcome without affecting the person's ability to give informed consent, which clearly raises significant ethical issues. Furthermore, the common usage of acetylcholinesterase inhibitors to stem the rate of cognitive decline may have implications for the clarity of findings.

Although it has not been formally assessed, clinical observations have suggested that the use of such medication can increase insight in some people (Bucks, 2003; personal communication). However, the refusal, or delay of these drugs for the purpose of research has significant ethical implications and the window of opportunity between diagnosis and commencement of medication is becoming smaller as these drugs become more openly available. These factors, therefore, present significant challenges for the study of insight in AD.

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Table 1: Demographic data for participants.

	AD Group *N*=18			OAC *N*=18		
	M	(*SD*)	Range	*M*	(*SD*)	Range
Age	77.7	(1.4)	66-88	74.2	(1.9)	61-89
Years of education	12.2	(0.9)	7-19	11.7	(0.7)	8-21
NART premorbid IQ	116.3	(1.5)	102-125	117.7	(1.0),	110-127
MMSE	24.4	(0.8)	18-30	28.5	(0.3)	26-30
S-GDS	2.1	(2.0)	0-6	1.4	(1.5)	0-5

Note: NART = National Adult Reading Test

MMSE = Mini Mental State Examination

S-GDS = Short Version of the Geriatric Depression Scale

Table 2: ECF scores for the four global awareness sub-groups.

	ECF		
	M	*(SD)*	Range
Aware $n = 4$	4.2	(3.20)	2 - 9
Variable Awareness $n = 3$	5.0	(2.00)	3 - 7
Consistent Anosognosia $n = 5$	5.4	(4.83)	-1 - 11
Mnemonic Anosognosia $n = 6$	2.0	(2.28)	0 - 6

Note: Higher ECF scores indicate greater impairments in executive control functioning.

Figure 1: Mean number of words predicted or recalled during the pre-study, post-study and recall stages of the three trials and after the delay.

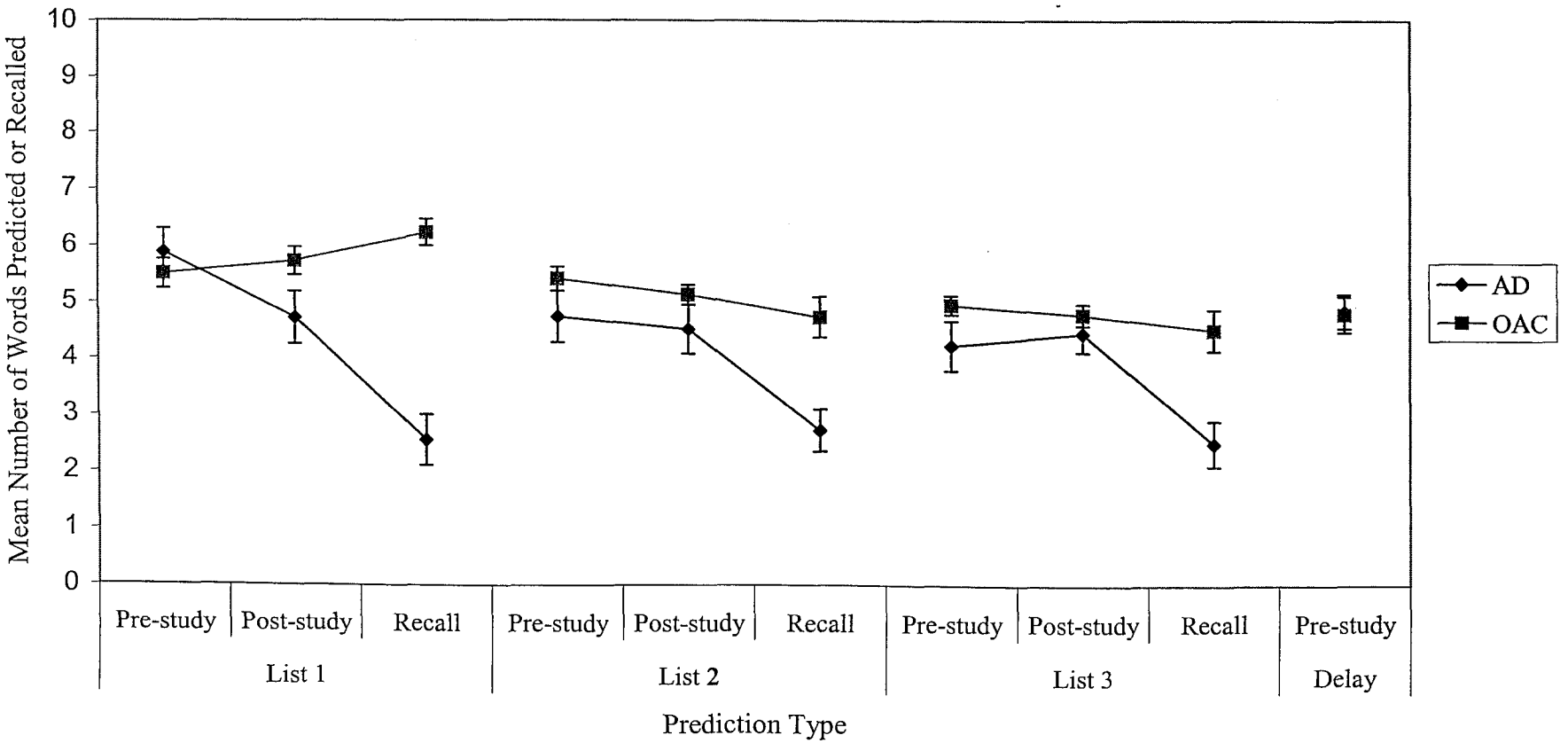
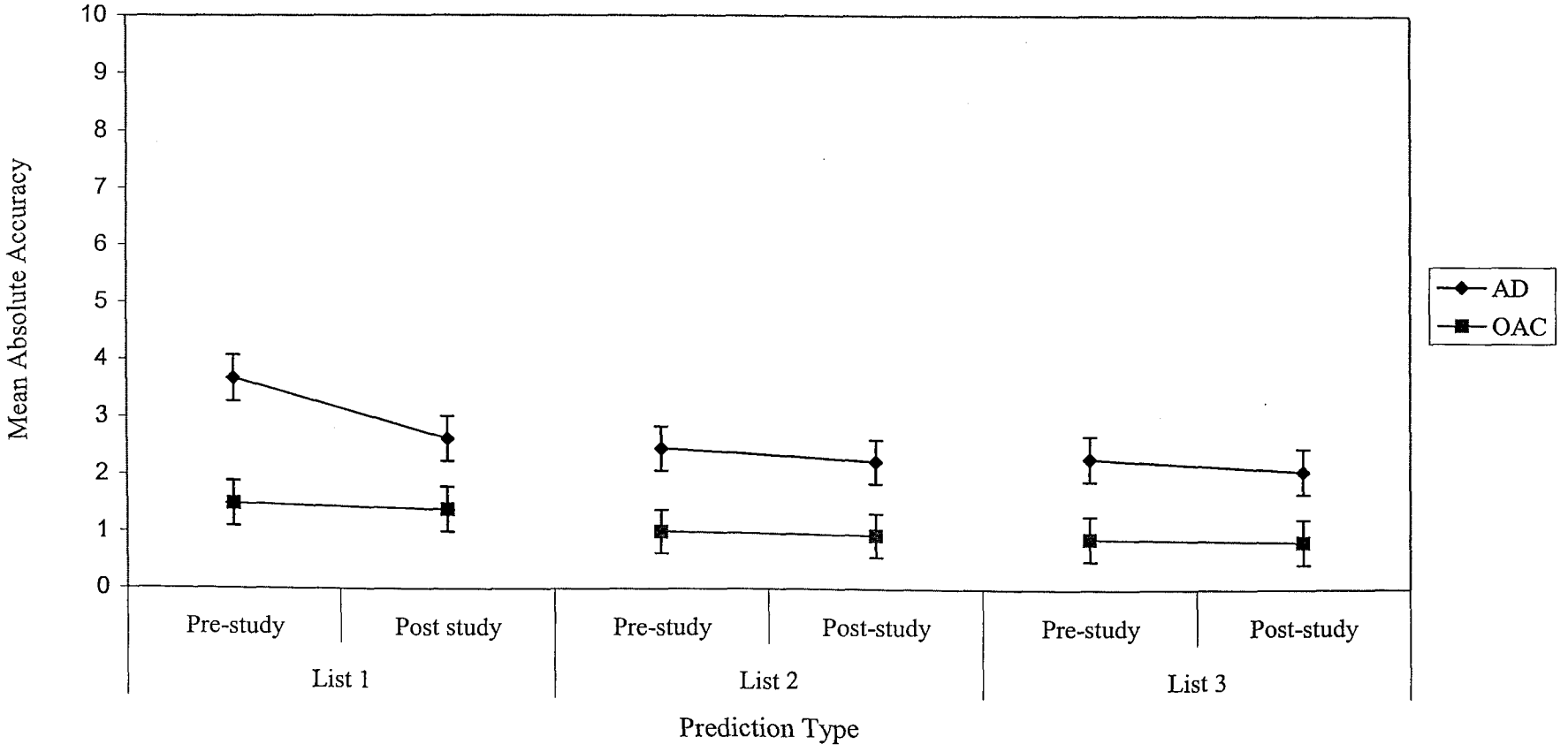
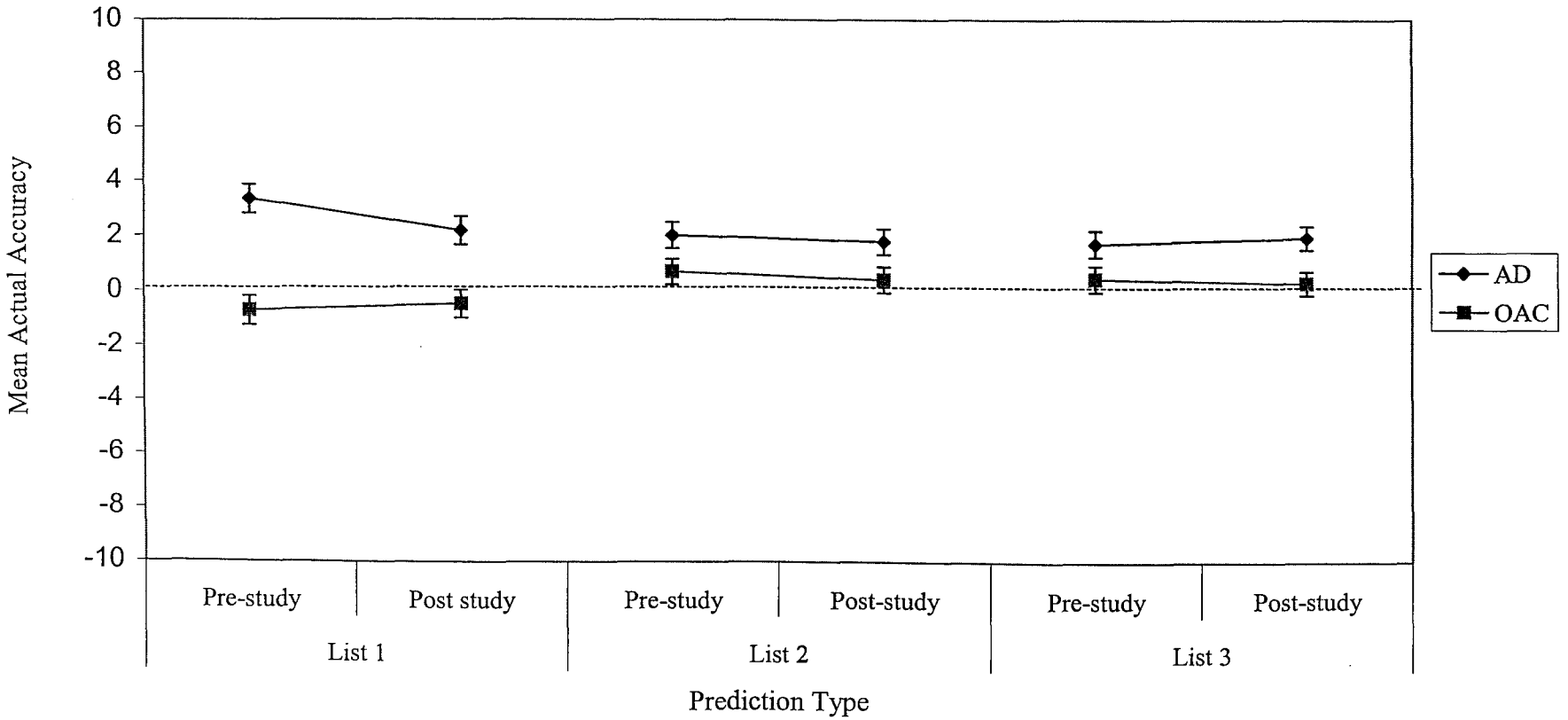


Figure 2: Mean absolute accuracy of pre- and post-study predictions over the three trials.



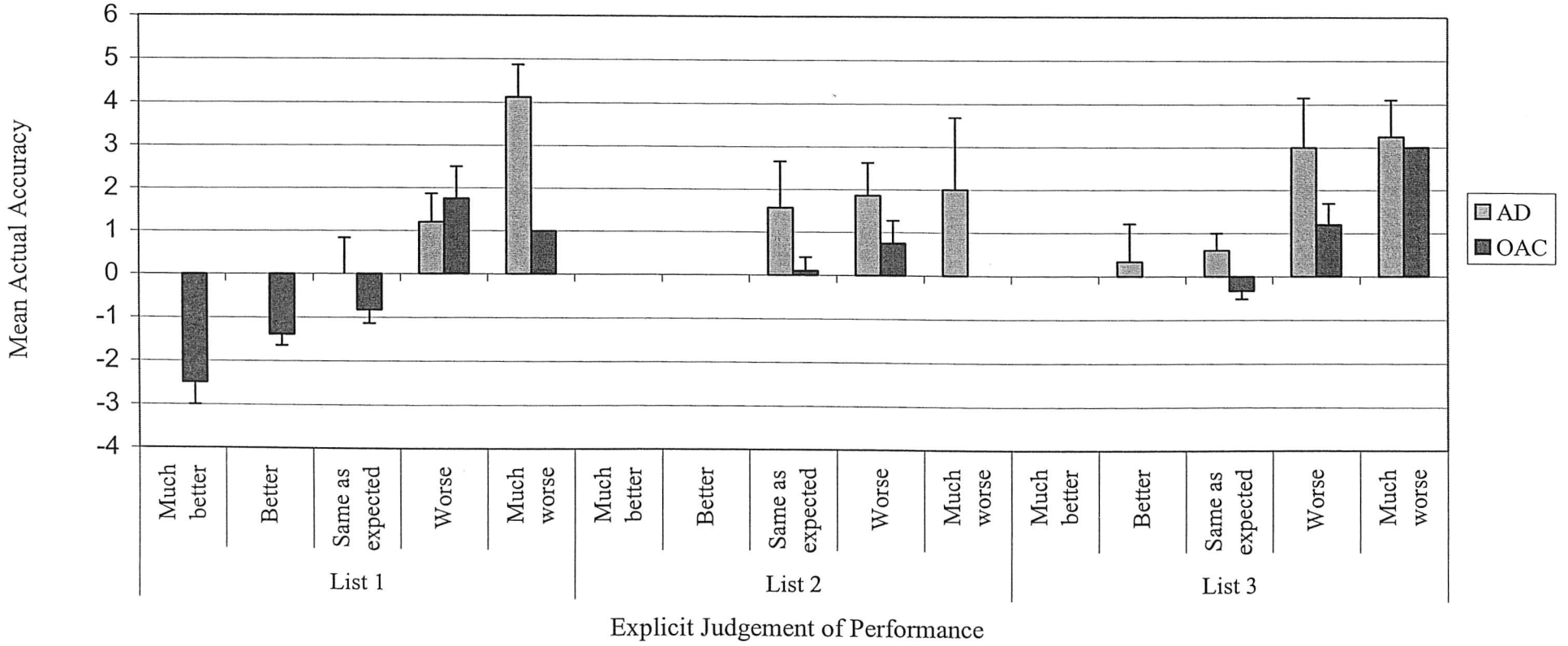
Note: Absolute Accuracy = the number of words discrepant between the prediction and the actual recall
Lower scores indicate more accurate performance.

Figure 3: Mean actual accuracy of pre- and post-study predictions over the three trials.



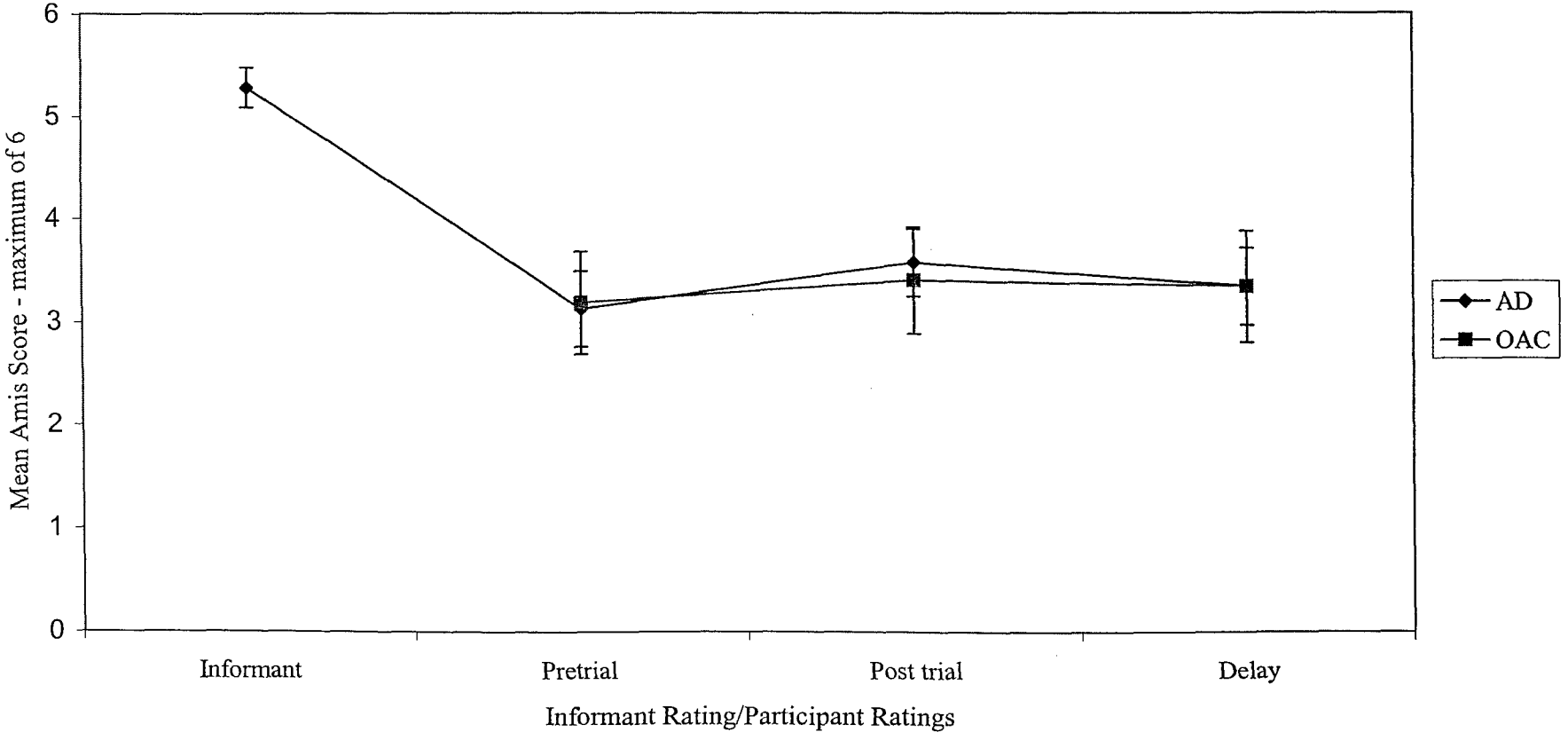
Note: Actual accuracy = the number of words over- or under-estimated in comparison to the actual recall Positive scores represent over-predictions and negative scores represent under-predictions

Figure 4: Participants' mean actual accuracy scores compared to their explicit judgements of their performance.



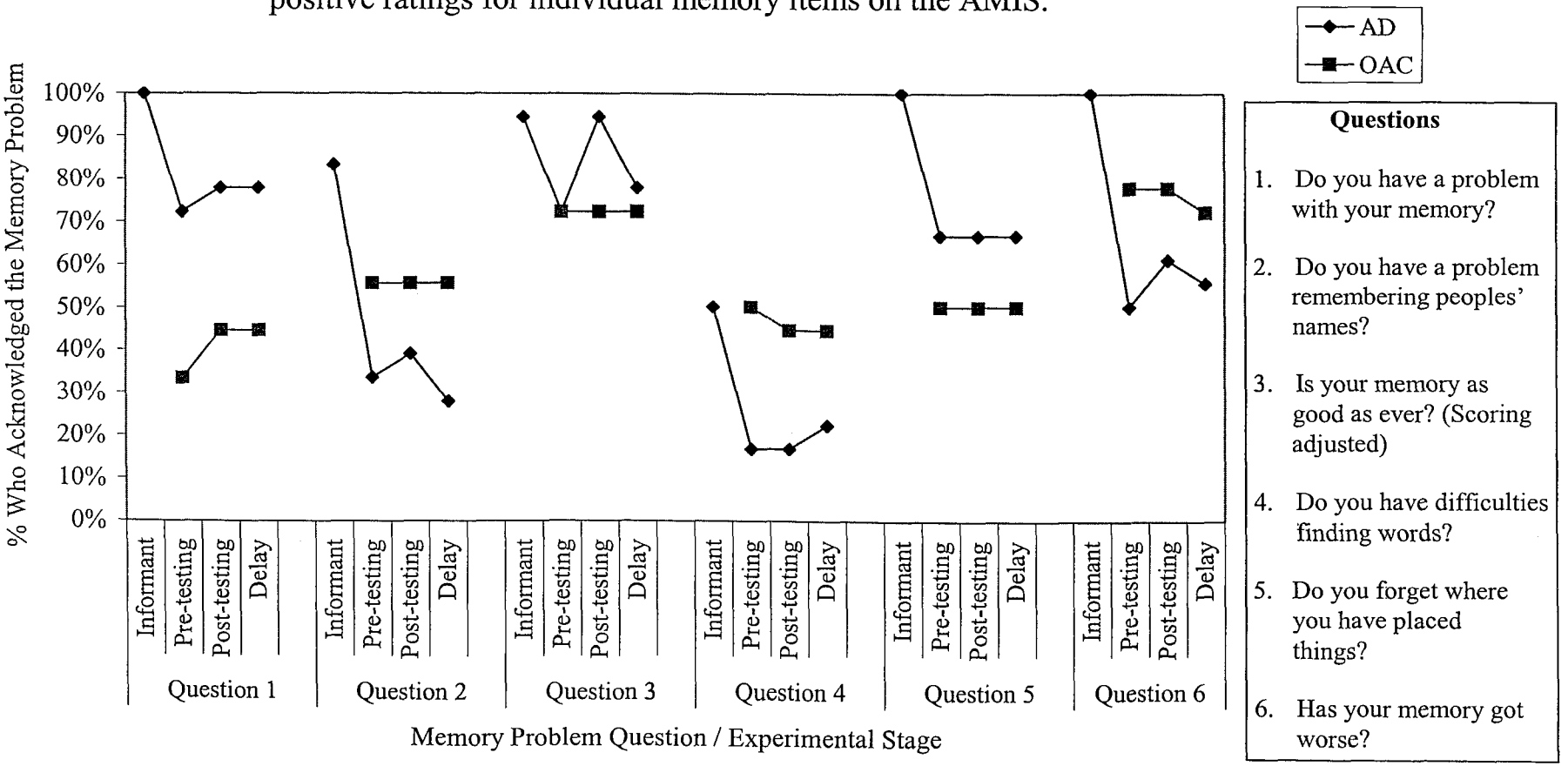
Note: Actual accuracy = the number of words over- or under-estimated in comparison to the actual recall
Positive scores represent over-predictions and negative scores represent under-predictions

Figure 5: Mean total AMIS scores for informants (AD only) and for participants (AD and OAC) at pre-testing, post testing and after a delay.



Note: Higher scores for participants indicate greater awareness of global memory functioning

Figure 6: Percentage of informants (AD only) and participants (AD and OAC) that reported positive ratings for individual memory items on the AMIS.



Appendices

Appendix 1: Flow diagram illustrating the recruitment of participants with AD

Appendix 2: Introductory letter from the consultant

Appendix 3: AD participant information sheet

Appendix 4: Participant consent form

Appendix 5: Informant information sheet

Appendix 6: Relative/friend assent form

Appendix 7: Control information sheet

Appendix 8: Word lists

Appendix 9: Awareness of Memory Impairment Scale (AMIS)

Appendix 10: AMIS – Informant adaptation

Appendix 11: Judgement of performance scale

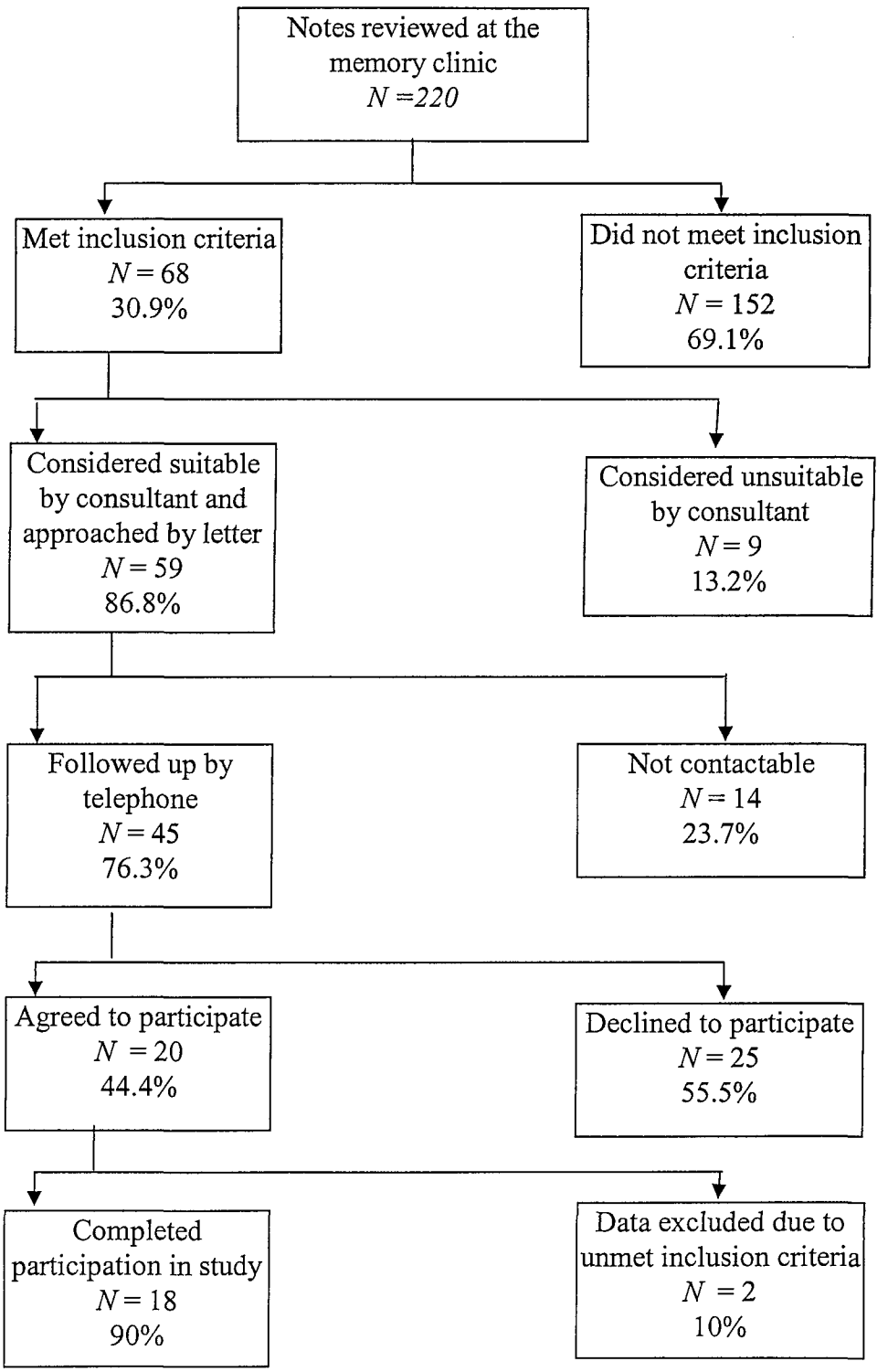
Appendix 12: Experimental script

Appendix 13: Psychology Sub-Committee, Southampton & S.W. Hants, Joint Ethics Committee communications

- Conditional approval letter
- Response to conditional approval letter
- Full approval letter

Appendix 14: Journal submission criteria

Appendix 1: Flow diagram illustrating the recruitment of participants with AD



Appendix 2: Introductory letter from consultant

West Hampshire NHS
NHS Trust

Memory Assessment and Research Centre
Moorgreen Hospital
Botley Road
West End
Southampton
SO30 3JB

Tel: 023 8047 5216
Fax: 023 8046 3022
E-mail: thorn5216@aol.com

Dear

Can you help us study peoples' experiences of memory problems?

Eleanor Ansell, a Trainee Clinical Psychologist, from the University of Southampton, is undertaking a project with us, talking to people who have experienced mild memory problems. She particularly wants to understand your experience of your memory problem and how it influences your life. I have enclosed an information sheet about the study and will be interested to know if you would like to take part. Whether you say yes or no, will not influence your care in any way and if you agree, you are at liberty to change your mind at any time. The results of the study will be kept confidential. Eleanor will give you a call in the next few weeks to see if you are agreeable and arrange a convenient time to visit you, if you are. Her telephone number at the University Clinical Psychology Department is 023 8059 5321, if you have any queries.

Thank you for taking the time to read this letter and I hope you will be agreeable and find the project interesting.

Kind regards

Yours sincerely

Dr David Wilkinson
Consultant in Old Age Psychiatry

Appendix 3: Patient information sheet

Version 1: 04/02/02

West Hampshire NHS Trust

Thornhill Unit
Moorgreen Hospital
Botley Road
West End
Southampton
SO30 3JB

Information Sheet (Patient)

Project: Experiences of memory problems

My name is Eleanor Ansell and I am a Trainee Clinical Psychologist, based at the University of Southampton. I am carrying out a research project to find out about memory problems and peoples' experiences of them. I am writing to ask you to take part in the study, but before you decide please take time to read the following information carefully and discuss it with others, if you wish. Please contact me if there is anything that is unclear or if you would like more information.

What is the purpose of the study?

This study is trying to find out about peoples' memory and experiences of memory problems.

Why have I been chosen?

You have been chosen because this study is interested in the memory functioning and experiences of people who report memory difficulties, such as yourself.

What will happen if I take part?

You will complete questionnaires about your experience of your memory difficulties and five short memory measures, like the ones you usually have whilst attending the Memory Clinic. It should take about 1 hour and 15 minutes to complete (including a break). I would visit you at your home or, if you preferred, I could see you at the Memory Clinic. Each of the questionnaires and measures comes with instructions and I will be there while you complete them, so that you can ask me about anything that is unclear.

What are the possible risks of taking part?
Very occasionally, people find the tasks more difficult than they had expected and this can be off-putting for them. I do not expect people to perform perfectly and I am happy for you to guess if you are not sure of the answer.

Will I benefit from taking part?
You may or may not receive any direct benefit from taking part in the study. However, information obtained during the course of the study may help us to gain a better understanding of peoples' memory difficulties. I hope that by knowing more about what it is like to have memory problems, we will be able to develop better ways of helping people in a similar position.

Do I have to take part?
It is entirely your choice to take part in the study. If you choose to take part, but then change your mind, you can stop **at anytime.** If you decide to stop, you don't have to give a reason. This will not affect any medical care you are getting now or in the future.

Will my taking part in the study be kept confidential?
All information collected during the course of the research will be kept strictly confidential. The results of this study will be marked with a number and **not** your name or address.

What will happen to the results of the study?
A report of the study will be written and you can ask to receive a summary of the results.

Who is organising and funding the research?
The project is being conducted as part of my training for the Doctorate Programme in Clinical Psychology at the University of Southampton.

Who has reviewed the study?
The Local Research Ethics Committee for the South West has reviewed the study jointly with the University of Southampton Research Ethics Committee.

If you have any questions about your rights as a participant in this research or you feel you have been placed at risk, you may contact Katherine Smith, Ethical Committee Secretary, Psychology Department, University of Southampton. Tel: 023 8059 4041.

How can I find out more?
If you have any questions about the project or you wish to request a summary please contact: **Eleanor Ansell, Doctorate Programme in Clinical Psychology, Shackleton Building (44), University of Southampton, Highfield, Southampton SO17 1BJ. Tel: 023 8059 5321**

Thank you for reading this information sheet. I hope that you will decide to take part in my project and that you will find it interesting.

Appendix 4: Participant consent form.

Version 1: 04/02/02

Consent Form - Participant

Project: Experiences of Memory Problems

Eleanor Ansell
Trainee Clinical Psychologist
Doctorate Programme in Clinical Psychology
Shackleton Building (44)
University of Southampton
Highfield
Southampton SO17 1BJ

Tel: 023 8059 5321

Please initial box

1. I confirm that I have read and understand the information sheet dated 04/02/02 (version 1) for the above study ☐

2. I understand that my participation is voluntary ☐

3. I understand that I am free to withdraw at anytime without my medical care or legal rights being affected ☐

4. I agree to take part in the above study ☐

Name of participant _____ Date_____ Signature_____

Researcher_____ Date_____ Signature_____

Appendix 5: Informant information sheet

Version 1: 04/02/02

West Hampshire NHS Trust

Thornhill Unit
Moorgreen Hospital
Botley Road
West End
Southampton
SO30 3JB

Information Sheet - Informant

Project: Experiences of memory problems

I am a Trainee Clinical Psychologist, based at the University of Southampton. I am carrying out a research project investigating memory problems and peoples' experiences of them in, collaboration with my colleagues at the Memory Clinic and at the University of Southampton.

I have written to your relative / friend to ask them to take part in the study. Before they decide it is important they understand why the research is being done and have the opportunity to discuss it with others. Please take time to read the following information carefully and discuss it with your relative/friend if you wish. Please contact me if there is anything that is not clear or if you would like more information.

Why am I being asked to agree to this study?

Because your relative/friend may have difficulty remembering/concentrating on all the details of this study, we are asking you to confirm that they would be happy to take part and you are happy for them to do so. We recognise that no one can decide for another individual. However, we feel that it is a good idea to ask the opinion not only of the person themselves, but also of someone who knows them well, so as to be absolutely sure that we have not misunderstood their decision. Even if both you and your relative/friend decide to go ahead with the study, if at any stage your relative/friend starts to show that they are unhappy with what we are asking him/her to do, we will

stop what we are doing and, if necessary, will stop the study altogether.

What is the purpose of the study?
This study is trying to find out about peoples' memory and experiences of memory problems. Your relative/ friend has been asked to take part as they have reported memory difficulties and we are interested in their experiences of these difficulties.

What will it involve?
Should they decide to take part in the study, your relative/friend will be asked to complete questionnaires about their experiences of memory difficulties and five short memory measures, like the ones they usually have whilst attending the Memory Clinic. It should take about 1 hour and 15 minutes to complete (including a break). I would visit them at their home or if they preferred I could see them at the Memory Clinic. Each of the questionnaires and measures comes with instructions and I will be there while they complete them, so that they can ask me about anything that is unclear.

What are the possible risks of taking part?
Very occasionally, people find the tasks more difficult than they had expected and this can be off-putting for them. I have informed your relative/friend that I do not expect people to perform perfectly and am happy for them to guess if you are not sure of the answer.

Will they benefit from taking part?
Your relative/friend may or may not receive any direct benefit from taking part in the study; some people enjoy taking part in research. However, information obtained during the course of the study may help us to gain a better understanding of peoples' memory difficulties. I hope that by knowing more about what it is like to have memory problems, we will be able to develop betters ways of helping people in the future.

Do they have to take part?
It is entirely their choice to take part in the study. If they choose to take part, but then change their mind, they can stop **at anytime.** If they decide to stop, they don't have to give a reason. This will not affect any medical care they are getting now or in the future.

Will their taking part in the study be kept confidential?
All information collected during the course of the research will be kept strictly confidential. The results of this study will be marked with a number and **not** your relatives/friends name or address.

What will happen to the results of the study?
A report of the study will be written. A summary of the results will be made available on request.

Who is organising and funding the research?
The project is being conducted as part of my training for the Doctoral Programme in Clinical Psychology at the University of Southampton.

Who has reviewed the study?
The Local Research Ethics Committee for the South West has reviewed the study jointly with the University of Southampton Research Ethics Committee.

If you have any questions about your rights as a participant in this research or you feel you have been placed at risk, you may contact Katherine Smith, Ethical Committee Secretary, Psychology Department, University of Southampton. Tel: 023 8059 4041.

How can I find out more?
If you have any questions about the project or you wish to request a summary please contact me at: **Eleanor Ansell, Doctorate Programme in Clinical Psychology, Shackleton**

Building (44), University of Southampton, Highfield, Southampton SO17 1BJ. Tel: 023 8059 5321

Thank you for reading this information sheet.

Appendix 6: Relative / friend assent form

Version 1: 04/02/02

Assent Form - Relative/friend

Project: Experiences of Memory Problems

Eleanor Ansell
Trainee Clinical Psychologist
Doctorate Programme in Clinical Psychology
Shackleton Building (44)
University of Southampton
Highfield
Southampton SO17 1BJ

Tel: 023 8059 5321

Please initial box

1. I confirm that I have read and understand the information sheet dated 04/02/02 (version 1) for the above study ☐
2. I understand that my relative/friend's participation is voluntary ☐
3. I understand that my relative/friend is free to withdraw at any time without his/her medical care or legal rights being affected ☐
4. I am happy for my relative/friend to take part in the above study ☐

Name of patient _____

Name of relative _____ Date_____ Signature_____

Researcher_____ Date_____ Signature_____

Appendix 7: Control information sheet

Version 1: 04/02/02

West Hampshire NHS
NHS Trust

Thornhill Unit
Moorgreen Hospital
Botley Road
West End
Southampton
SO30 3JB

Information Sheet (Control)

Project: Experiences of memory problems

My name is Eleanor Ansell and I am a Trainee Clinical Psychologist, based at the University of Southampton. I am carrying out a research project to find out about peoples' experiences of their memory functioning. I am writing to ask you to take part in the study, but before you decide please take time to read the following information carefully and discuss it with others, if you wish. Please contact me if there is anything that is unclear or if you would like more information.

What is the purpose of the study?
This study is trying to find out about peoples' memory and experiences of memory problems.

Why Have I been chosen?
You have been chosen because I need a sample of people without memory problems with whom I can compare people with memory problems.

What will happen if I take part?
You will complete questionnaires about your experience of your memory functioning and five short memory measures. It should take about 1 hour and 15 minutes to complete (including a break). I would visit you at your home or if you preferred I could see you at Moorgreen Hospital, Southampton. Each of the questionnaires and measures comes with instructions and I will be there while you complete them, so that you can ask me about anything that is unclear.

What are the possible risks of taking part?
Very occasionally, people find the tasks more difficult than they had expected and this can be off-putting for them. I do not expect people to perform perfectly and I am happy for you to guess if you are unsure of the answer.

Will I benefit from taking part?
You may or may not receive any direct benefit from taking part in the study. However, information obtained during the course of the study may help us to gain a better understanding of peoples' awareness of their memory functioning. I hope that this will improve our knowledge of what it is like for people with memory problems and, as a result, aid the development of better ways of helping them.

Do I have to take part?
It is entirely your choice to take part in the study. If you choose to take part, but then change your mind, you can stop **at anytime.** If you decide to stop, you don't have to give a reason. This will not affect any medical care you or your relatives are getting now or in the future.

Will my taking part in the study be kept confidential?
All information collected during the course of the research will be kept strictly confidential. The results of this study will be marked with a number and **not** your name or address.

What will happen to the results of the study?
A report of the study will be written. A summary of the results will be made available on request.

Who is organising and funding the research?
The project is being conducted as part of my training for the Doctorate Programme in Clinical Psychology at the University of Southampton.

Who has reviewed the study?
The Local Research Ethics Committee for the South West has reviewed the study jointly with the University of Southampton Research Ethics Committee.

If you have any questions about your rights as a participant in this research or you feel you have been placed at risk, you may contact Katherine Smith, Ethical Committee Secretary, Psychology Department, University of Southampton. Tel: 023 8059 4041.

How can I find out more?
If you have any questions about the project or you wish to request a summary please contact: **Eleanor Ansell, Doctorate Programme in Clinical Psychology, Shackleton Building (44), University of Southampton, Highfield, Southampton SO17 1BJ. Tel: 023 8059 5321**

Thank you for reading this information sheet. I hope that you will decide to take part in my project and that you will find it interesting.

Appendix 8: Word lists

List 1	List 2	List 3
Pepper	Butter	Coast
Doll	Market	Arm
Table	Shore	Lip
Letter	Chair	Earth
Fox	Queen	Student
Apple	Lemon	Cabin
Window	Pole	Horse
Ticket	Pipe	Doctor
Corner	Grass	Valley
Rock	Jury	Engine

Appendix 9: Awareness of Memory Impairment Scale (AMIS)

AMIS (Sevush, 1999)

I am going to ask to six questions about your memory and I would like you to simply answer "YES" or "NO" to the questions.

1. Do you have a problem with your memory?	Yes	No
2. Do you have a problem remembering peoples' names?	Yes	No
3. Is your memory as good as ever?	Yes	No
4. Do you have difficulty finding words?	Yes	No
5. Do you forget where you have placed things?	Yes	No
6. Has your memory got worse?	Yes	No

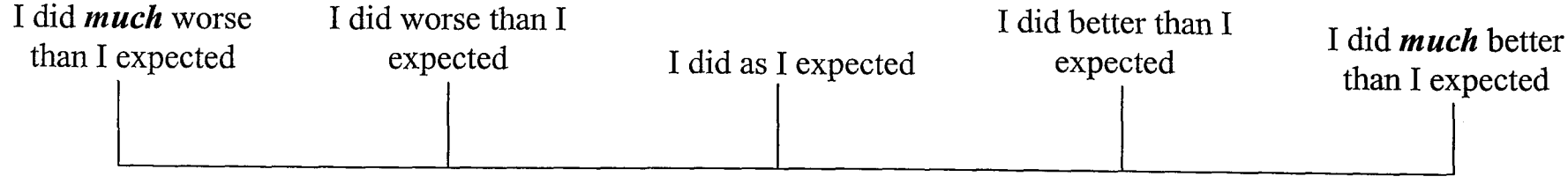
Appendix 10: AMIS – Informant adaptation

AMIS - Informant

I would like you to circle "YES" or "NO" in answer to the following questions about your relative/friend.

1. Does your relative/friend have a problem with their memory?	Yes	No
2. Does your relative/friend have a problem remembering peoples' names?	Yes	No
3. Is his/her memory as good as ever?	Yes	No
4. Does s/he have difficulty finding words?	Yes	No
5. Does s/he forget where they have placed things?	Yes	No
6. Has his/her memory got worse?	Yes	No

Appendix 11: Judgement of performance scale



Appendix 12: Experimental script

EXPERIMENTAL SCRIPT

INTRODUCTION

My name is Eleanor Ansell *(pause)*. I'm a Trainee Clinical Psychologist at Southampton University *(pause)*. Thank you for agreeing to help me with my research *(pause)*. I'm conducting some research with Dr Wilkinson looking at peoples' experiences of memory problems *(pause)*. I will need just over an hour of you time today *(pause)*. However, if you want to stop at any time, just let me know *(pause)*.You don't have to continue if you don't want to.

Show information sheet

Dr Wilkinson sent you an information sheet with his letter about the research *(pause)*. I have brought another copy with me here *(pause)*. It sets out what the study is about *(pause)*, why I am asking for your help *(pause)* and what I'd like you to help me with *(pause)*. Lets work through it together before you decide whether or not you want to take part.

Go through the information sheet - After each section check that they understand.

CONSENT

Have you any questions about taking part in the research?

Answer any questions they might have

If you are happy to carry on, please sign this consent forms. I need you to sign three copies *(pause)*. One for you to keep *(pause)*, one for me *(pause)* and one for your file *(pause)*.This is to say that you are happy to take part in the research *(pause)* and that I have explained everything to you.

Give the participant the forms to sign

CARER ASSENT

If you are happy for your relative/friend to take part in the research, please could you sign this form. I need you to sign three copies *(pause)*. One for you to keep *(pause)*, one for me *(pause)* and one for your file *(pause)*. This is to say that you are happy about your relative/friend taking part and that I have explained everything to you.

Give the carer/friend the forms to sign

OUTLINE OF THE TESTING

I am going to ask you to complete some brief tasks involving memory and reading *(pause)*. I am going to be looking at your performance on the tasks and comparing you with yourself *(pause)*. So it does not matter how well you do by comparison with anyone else, I am interested in your abilities under different conditions.

We don't expect people to remember everything on the memory tasks, so just do the best you can *(pause)*.

With some tasks I will be asking you to do the same thing repeatedly *(pause)*. I won't be trying to catch you out with this *(pause)*; it's merely a means of seeing if there is any change with practice *(pause)*. Okay?

Before we start there are two things I need to tell you:

Firstly, I won't be able to discuss the results of the questions with you today because I'll need time to score them afterwards *(pause)*. And secondly, any information you give me is confidential *(pause)*. This means that I am the only one who knows that it's you who is helping me. No one's results will be identifiable.

Before we start, do you wear glasses? If so we will probably need them.

QUESTIONNAIRE 1 - GERIATRIC DEPRESSION SCALE

The first questionnaire asks you about how you have been feeling in your mood lately. Please circle "yes" or " no" in answer to the questions. Ask me if anything is unclear.

Give them the questionnaire

QUESTIONNAIRE 2: AMIS

I am now going to ask you a few questions about your memory *(pause)*. Just answer "yes" or "no" to the questions.

1. Do you have a problem with your memory?	Yes	No
2. Do you have a problem remembering peoples' names?	Yes	No
3. Is your memory as good as ever?	Yes	No
4. Do you have difficulties finding words?	Yes	No
5. Do you forget where you have placed things?	Yes	No
6. Has your memory got worse?	Yes	No

Write down any comments

TEST 1: LIST (A) PREDICTION 1 - LIST 2

For the next task *(pause)* , I am going to show you 10 words *(pause)*. I want you to try and remember as many as you can *(pause)*, but before I show you the words I want you to guess how many of the words you will remember.

So there are 10 words *(pause)*, how many do you think you will be able to remember?

Predicted number...../10

How did you decide on that number?

➢ **If the person finds it difficult to say how many say:**

Just give me a guess as to how many you think you will recall? It doesn't matter if you end up recalling more or less than you predicted.

EXPOSURE TO LIST 2 WORDS

O.K., now I am going to show you the words, one at a time *(pause)*. Look at each word carefully and read it aloud *(pause)*. I will ask you to recall them in a few minutes time.

Show the words one at a time for a duration of 2 seconds each.

If they misread it, ask them to look at it again and then read it for them if they have problems, and ask them to repeat it.

PREDICTION 2

I am going to ask you to recall the words in a moment, but before you do, I want you to have another guess at how many of the 10 words you will recall.

So there are 10 words *(pause)*, how many do you think you will be able to remember?

Predicted number...../10

➢ **If they start counting the number of words they can recall, say:**
O.K I am going to interrupt you for a moment. What I want you to do is try to

guess how many without actually counting the words you can remember.

- **If the person finds it difficult to say how many say:**

Just give me a guess as to how many you think you will recall? It doesn't matter if you end up recalling more or less than you predicted.

FREE RECALL OF LIST 2

Now tell me as many words as you can from the list.*(Pause)* It doesn't matter if you recall more or less than your prediction. Just do the best that you can.

Stop after 1 minute of no recall.

Write Down The Words Recalled In The Order They Are Said (Including Incorrect Words)	

QUESTIONNAIRE 3: JUDGEMENT QUESTIONNAIRE

Show them the judgement scale

How do you think you did? *(pause)* Point to the scale. *(pause)* Do you think you did much better than you expected, better than you expected, the same as you expected, worse than you expected or much worse than you expected?

I did *much* **better** than I expected	-------
I did **better** than I expected	-------
I did the **same** as I expected	-------
I did **worse** than I expected	-------
I did *much* **worse** than I expected	-------

TEST 2: LIST (B) PREDICTION - LIST 1

I am going to show you another 10 words *(pause)*. Like before *(pause)*, I want you to try and remember as many as you can, *(pause)* as I am going to test you on them in a few minutes time *(pause)* but before I show you the words I want you to guess how many you will remember.

So there are 10 words, how many do you think you will be able to remember?

Predicted number...../10

How did you decide on that number?

> ➢ **If the person finds it difficult to say how many say:**
>
> *Just give me a guess as to how many you think you will recall? It doesn't matter if you end up recalling more or less than you predicted.*

EXPOSURE TO LIST 1 WORDS

O.K., now I am going to show you the words, one at a time *(pause)*. Look at each word carefully and read it aloud *(pause)*. I will ask you to recall them in a few minutes time.

Show the words one at a time for a duration of 2 seconds each.

If they misread it ask them to look at it again and then read it for them if they have problems and ask them to repeat it.

PREDICTION 2

I am going to ask you to recall the words in a moment, but before you do I want you to have another guess at how many of the 10 words you will recall.

So there are 10 words *(pause)*, how many do you think you will be able to remember?

Predicted number...../10

> ➢ **If they start counting the number of words they can recall, say:**
> *O.K I am going to interrupt you for a moment. What I want you to do is try to guess how many without actually counting the words you can remember.*

➢ **If the person finds it difficult to say how many say:** *Just give me a guess as to how many you think you will recall? It doesn't matter if you end up recalling more or less than you predicted.*

FREE RECALL OF LIST 1

Now tell me as many words as you can from the list.*(Pause)* It doesn't matter if you recall more or less than your prediction. Just do the best that you can.

Stop after 1 minute of no recall.

Write Down The Words Recalled In The Order They Are Said (Including Incorrect Words)	

QUESTIONNAIRE 4: JUDGEMENT QUESTIONNAIRE 2

Show them the judgement scale

How do you think you did? *(pause)* Point to the scale. *(pause)* Do you think you did much better than you expected, better than you expected, the same as you expected, worse than you expected or much worse than you expected?

I did *much* **better** than I expected	-------
I did **better** than I expected	-------
I did the **same** as I expected	-------
I did **worse** than I expected	-------
I did *much* **worse** than I expected	-------

TEST 3: LIST (C) PREDICTION - LIST 3

I am going to show you another 10 words *(pause)*. Like before *(pause)*, I want you to try and remember as many as you can, *(pause)* as I am going to test you on them in a few minutes time *(pause)* but before I show you the words I want you to guess how many you will remember.

So there are 10 words, how many do you think you will be able to remember?

Predicted number...../10

How did you decide on that number?

> ➢ **If the person finds it difficult to say how many say:**
>
> *Just give me a guess as to how many you think you will recall? It doesn't matter if you end up recalling more or less than you predicted.*

EXPOSURE TO LIST 3 WORDS

O.K., now I am going to show you the words, one at a time *(pause)*. Look at each word carefully and read it aloud *(pause)*. I will ask you to recall them in a few minutes time.

Show the words one at a time for a duration of 2 seconds each.

If they misread it ask them to look at it again and then read it for them, if they have problems and ask them to repeat it.

PREDICTION 2

I am going to ask you to recall the words in a moment, but before you do I want you to have another guess at how many of the 10 words you will recall.

So there are 10 words *(pause)*, how many do you think you will be able to remember?

Predicted number...../10

> ➢ **If they start counting the number of words they can recall, say:**
> *O.K I am going to interrupt you for a moment. What I want you to do is try to guess how many without actually counting the words you can remember.*

> ➢ **If the person finds it difficult to say how many say:**
>
> *Just give me a guess as to how many you think you will recall? It doesn't matter if you end up recalling more or less than you predicted.*

FREE RECALL OF LIST 3

Now tell me as many words as you can from the list.*(Pause)* It doesn't matter if you recall more or less than your prediction. Just do the best that you can.

Stop after 1 minute of no recall.

Write Down The Words Recalled In The Order They Are Said (Including Incorrect Words)	

QUESTIONNAIRE 5: JUDGEMENT QUESTIONNAIRE 2

Show them the judgement scale

How do you think you did? *(pause)* Point to the scale. *(pause)* Do you think you did much better than you expected, better than you expected, the same as you expected, worse than you expected or much worse than you expected?

I did *much* **better** than I expected	-------
I did **better** than I expected	-------
I did the **same** as I expected	-------
I did **worse** than I expected	-------
I did *much* **worse** than I expected	-------

QUESTIONNAIRE 6: AMIS

I am now going to ask you a few questions about your memory *(pause)* . These are the same questions I asked you earlier *(pause)*. It does not matter if you don't remember what you said last time *(pause)*. Just, answer the questions as if it is the first time that you have heard them *(pause)*. Answer "yes" or "no" to the questions *(pause)*.

Read out the questions to the person.

1. Do you have a problem with your memory?	Yes	No
2. Do you have a problem remembering peoples' names?	Yes	No
3. Is your memory as good as ever?	Yes	No
4. Do you have difficulties finding words?	Yes	No
5. Do you forget where you have placed things?	Yes	No
6. Has your memory got worse?	Yes	No

Write down any comments

START OF 20 MINUTE DELAY PERIOD

Start time:..... **Finish Time:.....**

TASK 3: NART

The next task is a reading task *(pause)*. It is not a memory task *(pause)* so don't worry about trying to remember the words *(pause)*. Please read each word aloud *(pause)*. Some of the words are quite unusual *(pause)*, but have a guess if you are not sure.

Stop after 14 errors in 15 consecutive words.

QUESTIONNAIRE 7: SOCIO-DEMOGRAPHIC QUESTIONNAIRE

Before we have a break I'd like to get a few details from you.

Can you tell me your:

Gender
Date of birth
Age
Years of education

Are you suffering form any current illnesses
E.g. cancer, Huntingdon's, Parkinson's, Depression, Infection

---

BREAK

Time to return:.....

DO NOT DISCUSS THE TESTS OR THEIR MEMORY DURING THE BREAK

END OF 20 MINUTES DELAY PERIOD

QUESTIONNAIRE 8: AMIS

I am now going to ask you a few questions about your memory *(pause)* . These are the same questions I asked you earlier *(pause)*. It does not matter if you don't remember what you said last time *(pause)*. Just, answer the questions as if it is the first time you have heard them *(pause)*. Answer "yes" or "no" to the questions *(pause)*.

Read out the questions to the person.

1. Do you have a problem with your memory?	Yes	No
2. Do you have a problem remembering peoples' names?	Yes	No
3. Is your memory as good as ever?	Yes	No
4. Do you have difficulties finding words?	Yes	No
5. Do you forget where you have placed things?	Yes	No
6. Has your memory got worse?	Yes	No

Write down any comments

TASK 4: PREDICTION OF RECALL

If I told you that I was going to show you another 10 words *(pause)* and then ask you to recall as many of the words as you could *(pause)*, how many words do you think you would recall?

Predicted number...../10

How did you decide on that number?

> ➢ **If the person finds it difficult to say how many say:**
>
> *Just give me a guess as to how many you think you will recall? It doesn't matter if you end up recalling more or less than you predicted.*

O.K, thank you for your prediction *(pause)*. I am sure you will be pleased to know that I am not actually going to ask you to do the recall task *(pause)*. What I was interested in was how many words you thought you would recall.

TASK 5: CLOX

CLOX 1:

Give the patient the CLOX sheet face down with a plain piece of white paper behind it, so that the circle at the bottom right corner is partially visible through the paper.

Draw me a clock that says 1:45. *(pause)* Put the hands and numbers on the face so that a child could read them.

Repeat the instructions if needed, but give no further assistance.

> ➢ **If s/he has started but is having difficulty, say:**
>
> *Just draw me a clock that says 1:45.*

CLOX 2:
Sit side by side. Return to the front of the CLOX form.

I am going to draw a clock here (**point to circle)** Watch carefully

Draw clock: Place the 12, 6, 3, 9 first. Set the hands at 1:45. Make the hands into arrows. Invite the person to copy the clock

Please copy my clock here **(point to space).**

TASK 6: MMSE

This is the last task and it takes about 5 minutes. Alright?

I am now going to ask you some questions about your memory and thinking skills. I will ask you a range of questions, *(pause)* some of them you may find quite simple; please don't be insulted. I do have to ask everyone the same questions *(pause).* Some of the questions you may find more difficult. Please have a go at everything. If you don't know the answer, just have a guess *(pause).* Is that o.k?

See score sheet.

ENDING AND DEBRIEF

Thank you for taking part in my research. Do you have any questions or comments about the research? **Explain what happens to the results.**

Thank you again for taking part. You have been very helpful.

Appendix 13: Psychology Sub-committee, Southampton & S.W. Hants, Joint Ethics communications.

- Conditional approval letter
- Response to conditional approval letter
- Full approval letter

University of Southampton

Department of Psychology

University of Southampton
Highfield
Southampton
SO17 1BJ
United Kingdom

Telephone +44 (0)23 8059 5000
Fax +44 (0)23 8059 4597
Email

9 April 2002

Eleanor Ansell
Department of Clinical Psychology
University of Southampton
Highfield, Southampton

Dear Eleanor,

Submission No: PSY/11/02

The ethics sub-committee for Psychology considered your application for the above study at its recent meeting and I am pleased to inform you that **Conditional Approval** was granted subject to the following changes being made:-

1. Question 15 needs to specify 'who' the participants will be and 'how' they will be recruited. You must approach the participants indirectly and therefore it was suggested that Dr Wilkinson's letter could include a tear-off for people who wanted to be contacted.

2. You must include in your risk assessment any possible risks to yourself as well as to the participants. You are also asked to specify how long people should wait without contact from you before telephoning the police.

3. Relatives/carers can only give assent.

You are asked to address the issues or make these changes and send a copy of the paperwork or letter of clarification to this office for approval.

Following conditional or withheld approval, recruitment of patients must not commence until full approval has been confirmed.

May I draw your attention to the enclosed conditions of approval which **must be complied with.**

Should any unforeseen problem of either an ethical or procedural nature arise during the course of this research and you feel the Joint Ethics Committee may be of assistance, please do not hesitate to contact us.

Yours sincerely,

PP **Professor Peter Coleman**
Chairman
Psychology Sub-Committee, Southampton & S.W. Hants, Joint Ethics Committee

37 Arlott Court
Northlands Road
Southampton
HANTS
SO15 2RZ

023 80635142

5th May 2002

Dear Professor Coleman

Re: Conditional approval for research PSY/11/02

Thank you for the feedback regarding my ethics application. I have discussed the recommendations with my research supervisors, Dr Romola Bucks and Dr Irene Coulson and we are happy to make the following changes.

1. Question 15 on the ethics form has been altered to give further details of the recruitment of participants (enclosed).

2. The risk assessment form has been altered to include possible risks to myself and a more detailed protocol on management of this risk (enclosed). There are no risks to participants.

3. References to "consent" have been changed to "assent" on the relative/carer assent form (enclosed).

However, having taken advice from Dr Wilkinson and, after careful consideration, we feel in our situation it would not be appropriate to require people to send back a tear off note to acknowledge interest in the study. Dr Wilkinson's team have found that tear off slips are too impersonal and do not give the person the opportunity to ask questions about the research prior to making a decision as to whether they would be interested in taking part. In contrast, the team have found that a follow up telephone call is more anxiety reducing. As it gives the participant the opportunity to question the researcher and to "get a feel for them" before deciding whether they wish to meet them. Furthermore, as I will be working as a member of Dr Wilkinson's department, telephoning the person does not breech the Data Protection Act. I hope that the committee will feel that this decision is acceptable.

Thank you for all your help.

Yours sincerely,

Eleanor Ansell

Eleanor Ansell

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28 May 2002

Eleanor Ansell
Department of Clinical Psychology
University of Southampton
Highfield, Southampton
SO17 1BJ

Dear Eleanor,

Re: Submission No. PSY/11/02

Following the conditional approval and in response to your correspondence dated 5 May 2002, I am pleased to confirm **full approval** having received the required amendments.

This committee is fully compliant with the International Committee on Harmonisation/Good Clinical Practice (ICH) Guidelines for the Conduct of Trials involving the participation of human subjects as they relate to the responsibilities, composition, function, operations and records of an Independent Ethics Committee/Independent Review Board. To this end it undertakes to adhere as far as is consistent with its Constitution, to the relevant clauses of the ICH Harmonised Tripartite Guideline for Good Clinical Practice, adopted by the Commission of the European Union on 17 January 1997.

Yours sincerely,

pp Professor Peter Coleman
Chairman
Psychology Sub-Committee, Southampton & S.W. Hants. Joint Ethics Committee

Appendix 14: Journal submission criteria

Aging and Mental Health

Instructions for Authors:

Note to Authors:** please make sure your contact address information is clearly visible on the **outside** of all packages you are sending to Editors.

Aging and Mental Health welcomes original contributions from all parts of the world on the understanding that their contents have not previously been published nor submitted elsewhere for publication. All submissions will be sent anonymously to independent referees. It is a condition of acceptance that papers become the copyright of the publisher. **Books for review** should be sent to Dr Chris Gilleard, Psychology Department, Springfield Hospital, Tooting, London SW17 7DJ, UK.

Manuscripts
Manuscripts may be in the form of: (i) regular articles (not exceeding 10,000 words); or, (ii) short reports for rapid publication (not exceeding 2,000 words). Four complete copies should be submitted to either Editor: **Dr Martin Orrell**, Department of Psychiatry and Behavioural Sciences, University College London, Wolfson Building, 48 Riding House, London W1N 8AA, UK. Tel: +44 (0)207 679 9452. Fax: +44 (0)207 323 1459, or **Dan G. Blazer**, J. P. Gibbons Professor of Psychiatry and Behavioral Sciences, Duke University Medical Center, School of Medicine, Box 3005, Durham, NC 27710, USA.

All submissions should be in the style of the **Publication Manual** of the American Psychological Association (4th edition, 1994). Papers should be typed on one side of the paper, double spaced throughout (including the references), with margins of at least 2.5 cm (1 inch). All pages must be numbered.

The first page should include the title of the paper, first name, middle initial(s) and last name of the author(s), and for each author a short institutional address, and an abbreviated title (for running headlines within the article). At the bottom of the page give the full name and address (including telephone and fax numbers and e-mail address if possible) of the author to whom all correspondence (including proofs) should be sent. The second page should repeat the title and contain an abstract of not more than 200 words. The third page should repeat the title as a heading to the main body of the text.

The text should normally be divided into sections with the headings Introduction, Methods, Results, and Discussion. Long articles may need subheadings within some sections to clarify their content. Within the text section headings and subheadings should be typed on a separate line without numbering, indentation or bold or italic typeface.

Electronic Submissions. Authors should send the final, revised version of their articles in both hard copy paper and electronic disk forms. It is essential that the hard copy (paper) version **exactly** matches the material on disk. Please print out the hard copy from the disk you are sending. Submit three printed copies of the final version with the disk to the journal's editorial office. Save all files on a standard 3.5 inch high-density disk. We prefer to receive disks in Microsoft Word in a PC format, but can translate from most other common word processing programs as well as Macs. Please specify which program you have used. Do not save your files as "text only" or "read only".

References
References should follow APA style. All publications cited in the text should be listed following the text; all references listed must be mentioned in the text. Within the text references should be denoted by the author's name and year of publication in parentheses, e.g. (Woods, 1995) or (Mansell & McGill, 1995) or, if there are more than two authors, (Gallico **et al.**, 1986). Where several references are quoted consecutively within the text the order should be alphabetical, e.g. (Elford & Sherr, 1989; Folkman, 1992). Similarly, where several references are quoted within a single year, the order should be alphabetical (Mansell & McGill, 1995; Woods, 1995). If more than one paper from the same author(s) and year is listed, the date should be followed by (a), (b) etc., e.g. (Blazer, 1995a).

References should be listed at the end of the paper in alphabetical order, typed in double spacing. Responsibility for the references and their verification against the original documents lies with the author(s).

References should be listed on a separate sheet(s) in the following standard form, capitalisation and punctuation:

a) for periodical articles (titles of journals should not be abbreviated):

WOODS, B. (1995). *Dementia care: progress and prospects.* **Journal of Mental Health, 5,** 115-124.

b) for books:

NORMAN, A. (1987). **Aspects of ageism.** London: Centre for Policy on Ageing.

c) for chapters within multi-authored books:

ROBERTSON, I. T. (1994). Personality and personnel selection. In C. L. COOPER & D. M. ROUSSEAU (Eds.), **Trends in organizational behavior** (pp. 75-89). Chichester: Wiley.

Units of measurement
All measurements must be cited in SI units.

Illustrations
All illustrations (including photographs, graphs and diagrams) should be referred to as Figures and their position indicated in the text (e.g. Fig. 3). Each should be submitted on a separate sheet of paper, numbered on the back with Figure number (Arabic numerals) and the title of the paper. The captions of all figures should be submitted on a separate sheet, should include keys to symbols, and should make interpretation possible without reference to the text.

Figures should ideally be professionally drawn and designed with the format of the journal (A4 portrait, 297 x 210 mm) in mind and should be capable of reduction.

Tables
Tables should be submitted on separate sheets, numbered in Arabic numerals, and their position indicated in the text (e.g. Table 1). Each table should have a short, self-explanatory title. Vertical rules should not be used to separate columns. Units should appear in parentheses in the column heading but not in the body of the table. Any explanatory notes should be given as a footnote at the bottom of the table.

Proofs
Proofs will be sent to the author nominated for correspondence. Proofs are supplied for checking and making essential typographical corrections, not for general revision or alteration. Proofs must be returned (by air mail or fax if overseas) within 72 hours of receipt.

Offprints
Fifty offprints of each paper are supplied free, to the nominated author for correspondence for further distribution, together with a complete copy of the relevant issue of the journal. Additional offprints may be purchased and should be ordered when proofs are returned. Offprints are sent approximately two weeks after publication.

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