

Dual task performance in early Alzheimer's disease, amnesic mild cognitive impairment and depression

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Background. The dual task paradigm (Baddeley *et al.* 1986; Della Sala *et al.* 1995) has been proposed as a sensitive measure of Alzheimer's dementia, early in the disease process.

Method. We investigated this claim by administering the modified dual task paradigm (utilising a pencil-and-paper version of a tracking task) to 33 patients with amnesic mild cognitive impairment (aMCI) and 10 with very early Alzheimer's disease, as well as 21 healthy elderly subjects and 17 controls with depressive symptoms. All groups were closely matched for age and pre-morbid intellectual ability.

Results. There were no group differences in dual task performance, despite poor performance in episodic memory tests of the aMCI and early Alzheimer's disease groups. In contrast, the Alzheimer patients were specifically impaired in the trail-making test B, another commonly used test of divided attention.

Conclusions. The dual task paradigm lacks sensitivity for use in the early differential diagnosis of Alzheimer's disease.

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Introduction

Alzheimer's disease (AD) is the most common form of dementia, estimated to rise dramatically in the future (Wimo *et al.* 2003). Research has focused on early accurate diagnosis and intervention. The construct 'amnesic mild cognitive impairment' (aMCI; Peterson *et al.* 2001) has become increasingly popular to predict those who are most at risk for developing dementia. It is considered a transitional stage between normal ageing and the earliest clinical diagnosis of AD (Petersen, 2005; Petersen & O'Brien, 2006). Research on clinic-based samples has suggested that the conversion rate from aMCI to dementia is 10–15% per year (e.g. Petersen *et al.* 1999; Storandt *et al.* 2006) compared with between 1% and 2% in a normal age-matched non-clinical sample.

While primary impairment in very early AD includes episodic memory function, many authors have reported that attention and executive functioning are

also vulnerable at this stage (Parasuraman & Haxby, 1993; Perry & Hodges, 1999). In particular, people with early AD exhibit marked difficulty dividing their attention between two concurrent tasks. By comparing performance of a synchronous dual task with that of identical task components done separately and consecutively, a deficit in dual performance can be attributed to failure of the central executive that coordinates the simultaneous operation of these components (Baddeley *et al.* 1986). One advantage of the dual task paradigm is that it avoids modality-specific interference between tasks: the tracking task is presented visually and a manual response is required; information for the digit span task is presented aurally with a verbal response (Nebes *et al.* 2001). A further strength is that task demands can be fixed at individual ability levels, controlling for individual variation in performance in the component parts of the dual task. Therefore, each patient is his or her own control, adjusting for the generally poorer performance of AD patients in the baseline tasks (Logie *et al.* 2004).

Research has suggested that failure of the 'coordination' function is characteristic of mild AD in a laboratory setting. Participants with mild AD appear to

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be impaired, irrespective of task demands, and this impairment has been found to worsen with illness progression (Baddeley *et al.* 1986, 1991; MacPherson *et al.* 2004). Proponents of the dual task paradigm suggest such findings are in contrast to normal ageing, which they believe has a relatively minor effect on dual task performance (e.g. Baddeley *et al.* 1986; Hartley & Little, 1999; Logie *et al.* 2004; but see Crossley & Hiscock, 1992). The equipment used for this test is often an expensive computerized tracking device impractical for clinical settings (e.g. Baddeley *et al.* 1991; Logie *et al.* 2004). Della Sala *et al.* (1995) developed a modified pencil-and-paper version of the tracking component for the dual task. This has been reported to produce results comparable with the original instrument (Della Sala *et al.* 1995; Sebastian *et al.* 2006). To our knowledge the dual task paradigm has not been investigated with a sample defined according to recent aMCI criteria (Petersen *et al.* 1999).

The aim of this study was therefore to assess dual task performance in aMCI to ascertain whether this measure can be useful in the early diagnosis of AD. As AD is associated with a specific impairment in the aspect of working memory that coordinates performance of two separate tasks, we predicted that the performance of people with aMCI and very early AD should be significantly lower than that of aged matched controls. Furthermore, the inclusion of a group of elderly patients with symptoms of depression would test the specificity of dual task impairments in AD. On the basis of the previous research, we predicted that the depressed group would show impairment in the dual task compared with controls.

Method

Participants

We examined 33 patients with aMCI, 10 early AD patients, 17 control out-patients with depressive symptoms and 21 healthy elderly controls, following a protocol approved by the local ethics of research committee. All participants also took part in a larger longitudinal study of neuropsychological markers in pre-clinical AD. The aMCI patients were recruited over a 2-year period (September 2003–September 2005) from tertiary referrals to the local neuropsychological assessment service for older adults and met criteria for aMCI (Petersen *et al.* 1999). MCI patients had to give subjective reports of memory difficulty corroborated by an informant and exhibit objective memory impairments on neuropsychological tests of episodic memory. In terms of impairments on tests of episodic memory, 13 participants showed an

impairment of more than 2 standard deviations (S.D.) below our control mean on two or more tests, a further four showed impairments of 1.5 to 2 S.D. on two or more tests, 12 participants were impaired at 1–1.5 S.D. below control means on two or more tests, and the final four participants performed more than 1 S.D. below controls on one episodic memory test. All aMCI patients underwent comprehensive neuropsychological and psychiatric evaluation and medical screening prior to study entry, as well as neuroimaging before or during the study period, if thought to be clinically indicated by the responsible specialist, i.e. in 24 of the 33 participants in this group. Exclusion criteria for the aMCI group were a diagnosis of dementia or other medical/neurological conditions which may account for memory loss, untreated depressive illness, significant or predominant cerebrovascular disease on neuroimaging, significant motor and/or visual problems or an age below 58 years. Mini Mental State Examination (MMSE) scores ranged from 24 to 30, with a mean of 28.3. The final aMCI group consisted of 15 males and 18 females with a mean age of 73.3 years (range 58–85 years).

For the healthy elderly control group (MMSE 28–30), we recruited spouses or carers of patients who had attended the service. Potential participants were excluded if there was a history of medical, psychiatric or neurological conditions (i.e. stroke or cerebrovascular disease, head injury, alcoholism, schizophrenia, etc) that could conceivably affect cognitive functioning. The healthy elderly control group was matched as closely as possible to the aMCI and early AD groups in terms of age and estimated pre-morbid intelligence quotient (IQ). The final elderly control group consisted of eight males and 13 females with had a mean age of 69.5 years (range 59–81 years).

Ten participants diagnosed with AD, in accordance with National Institute of Neurologic, Communicative Disorders and Stroke–AD and Related Disorders Association (NINCDS-ADRDA; McKhann *et al.* 1984) and DSM-IV (APA, 1994) diagnostic criteria, took part in the current study. AD patients were recruited from tertiary referrals to our neuropsychology service or via referrals to the local old age psychiatry service. All early AD patients scored above 23/30 on the MMSE and above 65/100 on the more comprehensive Addenbrooke's cognitive examination (ACE; Mathuranath *et al.* 2000), indicating relatively mild disease. Patients had undergone relevant medical screening and neuroimaging, together with comprehensive psychiatric and neuropsychological evaluation as part of their initial diagnostic workup. The final early AD group consisted of three males and seven females with a mean age of 73.6 years (range 65–81 years).

Seventeen participants with depressive symptoms (MMSE 25–30) were recruited via local psychiatric out-patient clinics and day hospitals. In an attempt to match this patient group with the aMCI group in terms of illness severity, patients with milder forms of depression were included. Fifteen of the 17 participants were receiving treatment for their symptoms at the time of testing; all but two of these pharmaceutical in nature. As it has been suggested that type of depression does not influence the magnitude of cognitive deficits (Christensen *et al.* 1997), participants with a variety of disorders were included. Eight patients had a history of major depression, two of bipolar disorder, two were suffering from anxiety disorders with depressive features, three were considered dysthymic and two were considered to be suffering with a subclinical level of depressive symptoms. Mean geriatric depression scale (30-item version) score for this group was 13.2 (range 0–27). We once again excluded patients with any medical, neurological or psychiatric condition with a known potential to affect cognitive function. The group consisted of three males and 14 females with a mean age of 73.3 years (range 65–84 years). Subjects gave informed written consent to the whole protocol which was approved by the Lothian Research Ethics Committee; the research was completed in accordance with the Helsinki Declaration.

Neuropsychological tests

All participants completed a variation on the modified dual task paradigm (Della Sala *et al.* 1995). This pencil-and-paper test of divided attention consists of two components (a digit span task and a visuospatial tracking task) that are each performed on their own before being performed concurrently. First, participants' digit span was determined. This involved repeating strings of digits read by an experimenter at a rate of approximately two per s. Initially, two-digit strings were presented and these increased one digit at a time if the participant correctly recited five of six examples of each length. When the participant failed to recite two or more strings of the same span, digit span for that person was considered to be the previous length. No time limit was imposed at this stage. Having determined the participants' individual digit span, participants had 90 s to recite as many digit strings, fixed at the individual participants' digit span, as possible (digit span – single). Responses were recorded as correct for each digit recited in the correct order.

Following this, participants completed the tracking task (Della Sala, 1999). An A3-sized sheet with 319 empty circles linked by a meandering line was

presented to the participant. The participant was instructed to trace a line through circles, following the line that was already there, without lifting the pen from the paper. Participants had 90 s for this trial, and the number of circles reached during this time was recorded (tracking – single). The final trial was the concurrent dual task. Here participants had 90 s to simultaneously perform both tasks: recite digit strings fixed at their digit span (digit span – dual) as well as carrying out a tracking task identical to the one used above (tracking – dual). In order to take into account the various strategies one may adopt in performing the two tasks simultaneously, an overall decrement score was calculated using the following formula:

$$\mu = (1 - [(P_m + P_t)/2]) \times 100,$$

where μ is the combined dual task score, P_m is the proportional loss in span performance between single (X_{single}) and dual task (X_{dual}) conditions, $[(X_{\text{single}} - X_{\text{dual}})/X_{\text{single}}]$ while P_t is the equivalent proportional loss in tracking score. Thus a score of 100 would represent no dual task decrement and lower scores reflect greater dual task decrements.

A number of further tests were administered as part of the longitudinal investigation of neuropsychological markers. These included measures of general cognitive ability, such as the ACE, the more widely known MMSE and the National Adult Reading Test, revised version (NART-R; Nelson & Willison, 1991). The NART-R was used to provide an estimate of the pre-morbid level of intellectual functioning. Episodic memory was assessed using the Hopkin's verbal list test, revised (HVLTR; Brandt, 1991) and the paired associates learning test (PAL) from the Cambridge automated neuropsychological test battery (Swainson *et al.* 2001). Participants also completed the trail-making test (TMT) part A and B (Reitan, 1985), considered a measure of attention and executive functioning.

The HVLTR requires participants to recall as many words as possible immediately following presentation of a 12-item word list. The word list is presented on three consecutive learning trials. The participant is also required to recall, and finally recognize, as many words from the list as he or she is able, following a delay of 30 min. The PAL is a computerized measure of visuospatial learning requiring participants to learn the locations of an increasing number (i.e. 1, 2, 3, 6 and then 8) of patterns (Swainson *et al.* 2001). The score of interest was the number of pattern-position errors at the six pattern level. The TMT A requires tracing a line linking numbers in ascending order, while for the TMT B participants have to connect numbers and letters alternatively in ascending order: the

Table 1. Demographic data

Variable	Controls (<i>n</i> = 21)	Depression (<i>n</i> = 17)	aMCI (<i>n</i> = 33)	Early AD (<i>n</i> = 10)	Group differences
Males (<i>n</i>)	8	3	16	3	
Females (<i>n</i>)	13	14	17	7	
Age	69.5 (7.3)	73.3 (6.6)	73.1 (6.3)	73.6 (5.8)	–
NART	118.2 (2.9)	116.8 (6.2)	116.3 (8.5)	115.6 (5.5)	–
MMSE ^a	29.1 (0.7)	28.6 (1.5)	28.4 (1.6)	25.0 (2.3)	Controls = depression = aMCI > AD
ACE	94.6 (3.3)	91.7 (5.0)	89.0 (5.6)	76.7 (6.6)	Controls > aMCI > AD Depression > AD

aMCI, Amnesic mild cognitive impairment; AD, Alzheimer's disease; NART, National Adult Reading Test; MMSE, Mini Mental State Examination; ACE, Addenbrooke's cognitive examination.

Values are given as mean (standard deviation).

^a Games–Howell multiple comparison carried out due to lack of homogeneity of variances.

participant has to divide his/her attention back and forth between multiple lines of thought.

Each of these measures has been shown to be sensitive to very early AD (Chen *et al.* 2000; Nathan *et al.* 2001; Swainson *et al.* 2001; Hogervorst *et al.* 2002; Blackwell *et al.* 2004; Stokholm *et al.* 2006). Neuropsychological assessments lasted approximately 90 min in total. The order of test administration was identical for all assessments.

Statistics

Data were analysed using SPSS 12.0 for Windows (SPSS Inc., Chicago, IL, USA). Demographic variables were analysed using univariate analysis of variance (ANOVA), and Tukey honestly significantly different pairwise comparisons were carried out on all significant analyses where possible. Where the assumption of homogeneity of variance was not met, this was adjusted for using Games–Howell *post-hoc* pairwise comparisons, given that the sample sizes were unequal in the current analysis. A univariate ANOVA was carried out on the overall decrement score (see above). Decrement scores broken down into tracking decrement and digit span decrement were also calculated and examined using ANOVAs. Two participants in the early AD group were incapable of completing the TMT B; in these cases a default ceiling score of 500 s to completion was applied.

Results

Participant characteristics

Demographic matching characteristics are presented in Table 1. There were no group differences in age [$F(3,77) = 1.73$] or estimated pre-morbid full-scale IQ [$F(3,75) = 0.55$]. The mean MMSE score for the early

AD group was, as expected, significantly lower than that of the other groups [$F(3,77) = 17.70$, $p < 0.0001$] (AD *v.* healthy controls, $p = 0.001$; AD *v.* controls with depressive symptoms, $p < 0.005$; AD *v.* controls, $p < 0.005$). No other group differences in mean MMSE score were noted. As expected, the early AD patients had significantly lower mean ACE scores than did all other groups [$F(3,77) = 29.30$, $p < 0.0001$] (*post-hoc* tests as above in all cases, $p < 0.0001$). The ACE also discriminated between normal elderly control participants and aMCI patients, with the latter group obtaining a significantly lower mean ACE score (*post hoc* $p = 0.01$).

Dual task performance

Group means and s.d.s for the digit span task and the tracking measures of the modified dual task paradigm are presented in Table 2. Mean percentage scores for performance on the concurrent tasks, the digit span tasks and the visuospatial tracking tasks for each of the four groups are presented in Table 3. On carrying out a one-way non-repeated ANOVA on the overall decrement score, no group difference was found [$F(3,77) = 0.63$]. Similarly, no significant group differences were found for any of the other component tasks or decrement scores.

Other cognitive functions

Group mean scores and s.d.s for the HVLTR, the number of errors at the six pattern level of the PAL and the TMT B are presented in Table 4. On analysing the HVLTR delayed recall data, there was a significant group effect [$F(3,77) = 12.39$, $p < 0.0001$]. On closer analysis, the AD group recalled significantly fewer words than the healthy control ($p < 0.0001$) and depression groups ($p < 0.0001$). Similarly, the aMCI

Table 2. Digit span and individual component measures of the dual task (span and tracking, performed separately and together)

Task	Controls (n=21)	Depression (n=17)	aMCI (n=33)	Early AD (n=10)
Digit span	5.5 (0.7)	5.8 (1.0)	5.6 (0.9)	5.1 (0.7)
Digit span (single) ^a	1.0 (0.03)	0.9 (0.05)	1.0 (0.05)	1.0 (0.03)
Digit span (dual) ^a	0.9 (0.05)	0.9 (0.08)	0.9 (0.08)	1.0 (0.02)
Tracking (single) ^b	141 (56.5)	140 (51.7)	126 (38.9)	120 (46.3)
Tracking (dual) ^b	122 (46.0)	126 (58.3)	114 (36.3)	107 (35.6)

aMCI, Amnesic mild cognitive impairment; AD, Alzheimer's disease.

Values are given as mean (standard deviation).

^a Proportion of digits recalled in the correct position (1 = all correct).

^b Number of circles joined in 90 s.

Table 3. Percentage loss of performance in component tasks and overall decrement score during the dual task^a

Task	Controls (n=21)	Depression (n=17)	aMCI (n=33)	Early AD (n=10)
Digit span	96 (3.8)	97 (8.6)	98 (7.6)	100 (3.3)
Tracking	90 (22.8)	88 (16.8)	92 (15.4)	93 (17.6)
Overall decrement	93 (11.1)	92 (8.2)	95 (8.6)	97 (9.1)

aMCI, Amnesic mild cognitive impairment; AD, Alzheimer's disease.

Values are given as mean (standard deviation).

^a Percentage loss of performance scores were calculated as $(1 - [(X_{\text{single}} - X_{\text{dual}}) / X_{\text{single}}]) \times 100$ and the overall decrement score as $\mu = (1 - [(P_m + P_t) / 2]) \times 100$, as described in the Method section.

Table 4. Other cognitive domain measures

Task	Controls (n=21)	Depression (n=17)	aMCI (n=33)	Early AD (n=10)	Group differences
HVLT-R delay	8.1 (2.8)	8.1 (3.3)	4.9 (3.3)	2.1 (3.7)	Controls = depression > aMCI = AD
PAL errors ^a	7.8 (6.9)	10.9 (7.8)	16.5 (12.9)	40.7 (10.6)	Controls, depression, aMCI < AD Controls < aMCI
TMT A	40.3 (11.2)	54.1 (23.1)	49.6 (36.1)	57.6 (25.3)	–
TMT B	87.6 (31.5)	134.2 (53.6)	106.3 (49.4)	216.7 (157.7)	Controls < depression Controls, depression, aMCI < AD ^b

aMCI, Amnesic mild cognitive impairment; AD, Alzheimer's disease; HVLT-R, Hopkins verbal learning test, revised; PAL errors, six pattern stage errors from the paired associates learning test; TMT A, trail-making test part A; TMT B, trail-making test part B.

Values are given as mean (standard deviation).

^a Games-Howell multiple comparison was used because of unequal variances.

^b After removing effects of TMT A (see text).

group performed more poorly than the healthy control ($p < 0.005$) and depression groups ($p < 0.01$). No significant difference was found between the AD and aMCI groups. The performance of the elderly control

and depression groups on the HVLT-R delayed recall did not differ. However, the AD group made significantly more errors at the six pattern stage of the PAL compared with all other groups [$F(3,755) = 22.82$,

$p < 0.0001$] (*post hoc* tests comparing AD with other groups were in all cases $p < 0.0001$). The aMCI group's error scores fell between those of the healthy control and AD groups, and significantly differed from both of these (aMCI *v.* healthy controls, $p < 0.05$; aMCI *v.* AD, $p < 0.0001$). A significant group effect was also found for the TMT B [$F(3,77) = 8.62$, $p < 0.0001$]. In the *post hoc* analyses, only the control and depression groups differed in terms of TMT B scores ($p < 0.05$); participants with depressive symptoms took significantly longer to complete the task. However, once time to completion on TMT part A (a measure of psychomotor speed) was statistically controlled for, a different pattern of group differences emerged [$F(3,76) = 7.76$, $p < 0.0001$]. Specifically, it was found that the participants with AD took longer to complete the second task compared with all other groups (aMCI *v.* AD, $p < 0.0001$; healthy controls *v.* AD, $p < 0.0001$; controls with depressive symptoms *v.* AD, $p < 0.05$). The group difference between the control and depressive symptom groups was no longer significant. No other group differences were uncovered.

Discussion

This study investigated the claim that the dual task paradigm can be used in the early diagnosis of dementia of the Alzheimer's type. We assessed the concurrent performance of a visuospatial tracing task and a digit span forward task in four diagnostic groups with aMCI (MMSE 24–30), early AD (MMSE 23–29), symptoms of depression (MMSE 25–30) and healthy elderly controls (MMSE 28–30). Our results show that aMCI is not associated with impaired dual task performance; those with aMCI had comparable performance to healthy older adults and older adults with depressive symptoms. Our early AD group was similarly unimpaired on the modified dual task paradigm relative to depressive and non-depressive elderly control groups and the presence of depressive symptoms appeared to have no effect on dual task performance. By contrast, and indeed by definition, episodic memory impairments were present in the aMCI and early AD groups. The early AD group also exhibited an impaired ability to divide their attention at pace, as indicated by part B of the TMT.

These results shed some light on previous findings. One line of research has suggested that dual task performance is vulnerable to the influence of AD, even early in the disease course (Baddeley *et al.* 2001; Logie *et al.* 2004). However, such studies generally involve participants varying in severity from minimal to mild AD. When participants with AD are divided by severity using the MMSE, only the more severely ill patients (e.g. MMSE < 24) are impaired on the dual

task paradigm (Greene *et al.* 1995; Perry *et al.* 2000; Crossley *et al.* 2004). This result is in agreement with the absence of impairment on the dual task measure observed in the current study in early AD. The combined findings suggest that dual task impairments are generally not observed early on in the AD process, with MMSE scores above 23/30.

Only one other study has investigated the dual task performance of a group of older adults with cognitive impairment without a diagnosis of dementia (Holtzer *et al.* 2004). Cognitively impaired adults, defined by a dementia rating scale (DRS) cut-off score of < 124 (Mattis, 1988), performed two tasks in different modalities at the same time. Two combinations of tests were used: a visual cancellation task (where participants were required to cross out a specified stimulus type from a field of stimuli) combined with a digit span task, and the same visual cancellation task combined with a verbal fluency task. The researchers report that their cognitively impaired group exhibited a significantly larger dual task decrement than age-matched controls. However, the cognitively impaired group in the Holtzer *et al.* (2004) study was identified solely on the basis of a DRS cut-off score falling at or below levels that are indicative of an underlying dementia. It is for this reason difficult to be certain of, or to compare, disease severity of this 'minimally cognitively impaired' group with other studies, which commonly use well-established clinical and research criteria to define patient groups. Furthermore, the cognitively impaired group in the Holtzer *et al.* (2004) study were significantly less well educated than the control groups, while in the current study participant groups were well matched both in terms of age and estimated levels of pre-morbid intelligence.

Holtzer *et al.* (2004) did not investigate the potential influence of depression on dual task performance. This is crucial where consideration is being given to the early and differential diagnostic value of a neuropsychological measure. Hasher & Zacks (1979) confirmed our result that people with depression show impaired attention during effortful processing tasks, for instance on measures of divided attention such as the TMT B (Nathan *et al.* 2001; Mahurin *et al.* 2006). Only one study has investigated the effect of depressive symptoms on Baddeley *et al.*'s (1986) original dual task paradigm (Nebes *et al.* 2001). This indicated that people with depression had a significantly greater decrement in computerized tracking performance and a composite decrement measure than non-depressed controls. No study to date has investigated the effects of clinically depressed mood on the modified version of the dual task paradigm to replicate or contradict our negative result (Della Sala *et al.* 1995).

A strength of the current investigation relates to the availability of additional neuropsychological data demonstrating the existence of significant episodic memory impairments in aMCI and early AD and additional impairment of speeded divided attention (as assessed by TMT B) in early AD. The TMT B assesses the ability to divide attention back and forth between multiple lines of thought (connecting numbers and letters, respectively), but differs from the dual task paradigm in that its different components are not drawn from separate modalities. Performance is thus more vulnerable to reduced processing capacity. Several previous studies have demonstrated that TMT B is impaired in the very early and even pre-clinical stages of AD (Lafleche & Albert, 1995; Arnaiz *et al.* 2000; Perry *et al.* 2000; Nathan *et al.* 2001; Crowell *et al.* 2002; Crossley *et al.* 2004; Alladi *et al.* 2006; Baudic *et al.* 2006; Stokholm *et al.* 2006), although its specificity for AD, as distinct from, for example, depression, has not been established.

The Holtzer *et al.* (2004) study compared the dual task performance of minimally cognitively impaired participants only with their performance on tests comprising the single task conditions (i.e. visual cancellation, digit span and letter fluency). However, these tests are not, generally speaking, associated with impairments in very early and pre-clinical AD and it is therefore not surprising that they are insensitive to cognitive deficits in the minimally impaired group, as was the case in this study.

One important methodological feature may have influenced the current results: While those studies reporting general dual task impairment in early AD used both computerized and pencil-and-paper versions of the tracking task, only the modified version utilising the pencil-and-paper tracking task (Della Sala *et al.* 1995) has been used in studies that separated participants by symptom severity. Thus, while patients who are minimally affected do not show impairments on the modified version of the task, it remains possible that they would show impairments if the test were more taxing – for instance if the dual task paradigm included the original computerized version of the tracking task. This version of the task requires increased effort and attention, as participants are required to adjust to an external influence (i.e. the speed of the light dot on the screen) rather than working at a self-defined rate. It may therefore be sufficiently taxing to identify those who are not picked up by the more straightforward pencil-and-paper tracking task. However, the paper-and-pencil version (as opposed to the computerized task) is more likely to be adopted for widespread use in clinical and research practice, which underscores the relevance of our negative result.

A further methodological issue is the variability of dual task administration, which can lead to difficulties comparing findings across studies. We administered each of the three trials in blocks of 90 s, while some previous studies set the trial time at 120 s (e.g. Perry *et al.* 2000). Most dual task studies have utilized pencil-and-paper tracking tasks that required participants to cross out boxes on an A4-size sheet of paper to form a chain (e.g. Baddeley *et al.* 1997). The current task required participants to trace a line through linked empty circles on an A3-size sheet. While the initial dual task paradigm involved recording the number of completely correct digit strings (Baddeley *et al.* 1986), many subsequent studies, including the current investigation, have calculated the number of digits recalled in the correct order for this measure. The significance of such alterations to dual task administration requires further investigation.

A partial alternative explanation for our negative result is that a majority of individuals forming our aMCI group may fail to convert to AD in the future. If this proves to be the case, then the absence of dual task impairment would not be surprising. The issue will be resolved through the longitudinal follow-up of participants with aMCI, currently underway. However, the sound performance of our early AD group on the dual task measure makes it more likely that the negative result for our aMCI patients is due to lack of test sensitivity rather than absence of underlying AD pathology. The impaired performance of the early AD group on an alternative popular measure of speeded divided attention implies that the dual task measure lacks sensitivity to very early changes of an attentional/executive nature in AD.

In conclusion, people with early AD and aMCI did not display impaired performance on the modified version of the dual task paradigm at a time when episodic memory, and in the case of early AD, speeded divided attention, were significantly impaired. The likely explanation is that the dual task paradigm is insufficiently sensitive for use as an adjunctive cognitive tool in the early diagnosis of AD. Future longitudinal research is needed to investigate the use of dual task tests of varying demand in aMCI and very early AD participants in an effort to determine the potential influence of task demands and complexity on performance.

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Declaration of Interest

None.

References

- Alladi S, Arnold R, Mitchell J, Nestor PJ, Hodges JR (2006). Mild cognitive impairment: applicability of research criteria in a memory clinic and characterization of cognitive profile. *Psychological Medicine* **36**, 507–513.
- APA (1994). *Diagnostic and Statistical Manual of Mental Disorders*. American Psychiatric Association: Washington, DC.
- Arnaiz E, Blomberg M, Fernaeus SE, Wahlund LO, Winbald B, Almkvist O (2000). Psychometric discrimination of Alzheimer's disease and mild cognitive impairment. *Alzheimer's Reports* **2**, 97–103.
- Baddeley AD, Baddeley HA, Bucks RS, Wilcock GK (2001). Attentional control in Alzheimer's disease. *Brain* **124**, 1492–1508.
- Baddeley AD, Bressi S, Della Sala S, Logie R, Spinnler H (1991). The decline of working memory in Alzheimer's disease: a longitudinal study. *Brain* **114**, 2521–2542.
- Baddeley AD, Della Sala S, Papagno C, Spinnler H (1997). Dual-task performance in dysexecutive and nondysexecutive patients with a frontal lesion. *Neuropsychology* **11**, 187–194.
- Baddeley AD, Logie R, Bressi S, Della Sala S, Spinnler H (1986). Dementia and working memory. *Quarterly Journal of Experimental Psychology Section A Human Experimental Psychology* **38**, 603–618.
- Baudic S, Barba GD, Thibaudet MC, Smagghe A, Remy P, Traykov L (2006). Executive function deficits in early Alzheimer's disease and their relations with episodic memory. *Archives of Clinical Neuropsychology* **21**, 15–21.
- Blackwell AD, Sahakian BJ, Vesey R, Semple JM, Robbins TW, Hodges JR (2004). Detecting dementia: novel neuropsychological markers of preclinical Alzheimer's disease. *Dementia and Geriatric Cognitive Disorders* **17**, 42–48.
- Brandt J (1991). The Hopkins verbal learning test: development of a new memory test with 6 equivalent forms. *Clinical Neuropsychologist* **5**, 125–142.
- Chen P, Ratcliff G, Belle SH, Cauley JA, DeKosky ST, Ganguli M (2000). Cognitive tests that best discriminate between presymptomatic AD and those who remain nondemented. *Neurology* **55**, 1847–1853.
- Christensen H, Griffiths K, Mackinnon A, Jacomb P (1997). A quantitative review of cognitive deficits in depression and Alzheimer-type dementia. *Journal of the International Neuropsychological Society* **3**, 631–651.
- Crossley M, Hiscock M (1992). Age-related differences in concurrent-task performance of normal adults: evidence for a decline in processing resources. *Psychology and Aging* **7**, 499–506.
- Crossley M, Hiscock M, Foreman JB (2004). Dual-task performance in early stage dementia: differential effects for automatized and effortful processing. *Journal of Clinical and Experimental Neuropsychology* **26**, 332–346.
- Crowell TA, Luis CA, Vanderploeg RD, Schinka JA, Mullan M (2002). Memory patterns and executive functioning in mild impairment and Alzheimer's disease. *Aging Neuropsychology and Cognition* **9**, 288–297.
- Della Sala S (1999). Paper and Pencil Dual Task – University of Edinburgh (<http://www.psy.ed.ac.uk/people/sdsala/tests/sdsdualtask>). Accessed 1 February 2008.
- Della Sala S, Baddeley A, Papagno C, Spinnler H (1995). Dual task paradigm: a means to examine the central executive. In *Annals of the New York Academy of Sciences*, vol. 769. *Structure and Functions of the Human Prefrontal Cortex* (ed. J. Graham, K. J. Holyoak and F. Boller), pp. 161–171. New York Academy of Sciences: New York.
- Greene JDW, Hodges JR, Baddeley AD (1995). Autobiographical memory and executive function in early dementia of Alzheimer type. *Neuropsychologia* **33**, 1647–1670.
- Hartley AA, Little DM (1999). Age related differences and similarities in dual-task interference. *Journal of Experimental Psychology: General* **128**, 416–449.
- Hasher L, Zacks RT (1979). Automatic and effortful processes in memory. *Journal of Experimental Psychology: General* **108**, 356–388.
- Hogervorst E, Combrinck M, Lapuerta P, Rue J, Swales K, Budge M (2002). The Hopkins verbal learning testing and screening for dementia. *Dementia and Geriatric Cognitive Disorders* **13**, 13–20.
- Holtzer R, Burright RG, Donovan PJ (2004). The sensitivity of dual-task performance to cognitive status in aging. *Journal of the International Neuropsychological Society* **10**, 230–238.
- Lafleche G, Albert MS (1995). Executive function deficits in mild Alzheimer's disease. *Neuropsychology* **9**, 313–320.
- Logie RH, Cocchini G, Della Sala S, Baddeley AD (2004). Is there a specific executive capacity for dual task coordination? Evidence from Alzheimer's disease. *Neuropsychology* **18**, 504–513.
- MacPherson SE, Della Sala S, Logie RH (2004). Dual-task interference of encoding and retrieval processes in healthy and impaired working memory. *Cortex* **40**, 183–184.
- Mahurin RK, Velligan DI, Hazleton B, Davis JM, Eckert S, Miller AL (2006). Trail making test errors and executive function in schizophrenia and depression. *Clinical Neuropsychologist* **20**, 271–288.
- Mathuranath PS, Nestor PJ, Berrios GE, Rakowicz W, Hodges JR (2000). A brief cognitive test battery to differentiate Alzheimer's disease and frontotemporal dementia. *Neurology* **55**, 1613–1620.
- Mattis S (1988). *Dementia Rating Scale (DRS)*. Psychological Assessment Resources, Inc.: Odessa, FL.
- McKhann G, Drachman D, Folstein M, Katzmann R, Price D, Stadlan EM (1984). Clinical diagnosis of Alzheimer's disease: report of the NINCDS-ADRADA work group under the auspices of Department of Health and Human Services Task Force on Alzheimer's disease. *Neurology* **34**, 939–944.

- Nathan J, Wilkinson D, Stammers S, Low JL** (2001). The role of tests of frontal executive functioning in the detection of mild dementia. *International Journal of Geriatric Psychiatry* **16**, 18–26.
- Nebes RD, Butters MA, Houck PR, Zmuda MD, Aizenstein H, Pollock BG, Muslant BH, Reynolds CF** (2001). Dual-task performance in depressed geriatric patients. *Psychiatry Research* **102**, 139–151.
- Nelson HE, Willison JR** (1991). *The Revised National Adult Reading Test – Test Manual*. NFER-Nelson: Windsor, UK.
- Parasuraman R, Haxby J** (1993). Attention and brain function in Alzheimer's disease: a review. *Neuropsychology* **7**, 242–272.
- Perry RJ, Hodges JR** (1999). Attention and executive deficits in Alzheimer's disease: a critical review. *Brain* **122**, 383–404.
- Perry RJ, Watson P, Hodges JR** (2000). The nature and staging of attention dysfunction in early (minimal and mild) Alzheimer's disease: relationship to episodic and semantic memory impairment. *Neuropsychologia* **38**, 252–271.
- Petersen RC** (2005). Mild cognitive impairment as a clinical entity and treatment target. *Archives of Neurology* **62**, 1160–1163.
- Petersen RC, Doody R, Kurz A, Morris JC, Rabins JC, Ritchie K, Rossor M, Thal L, Winbald B** (2001). Current concepts in mild cognitive impairment. *Archives of Neurology* **58**, 1985–1992.
- Petersen RC, O'Brien J** (2006). Mild cognitive impairment should be considered for DSM-V. *Journal of Geriatric Psychiatry and Neurology*, **19**, 147–154.
- Petersen RC, Smith GE, Waring SC, Ivnik RJ, Kokmen E** (1999). Mild cognitive impairment: clinical characterization and outcome. *Archives of Neurology* **56**, 303–308.
- Reitan RM** (1985). *Halstead-Reitan Neuropsychological Test Battery*. Reitan Neuropsychology Laboratory Press: Tucson, AZ.
- Sebastian MV, Menor J, Elousa MR** (2006). Attentional dysfunction of the central executive in AD: evidence from dual task and perseveration errors. *Cortex* **42**, 1015–1020.
- Stokholm J, Vogel A, Gade A, Waldemar G** (2006). Heterogeneity in executive impairment in patients with very mild Alzheimer's disease. *Dementia and Geriatric Cognitive Disorders* **22**, 54–59.
- Storandt M, Grant EA, Miller JP, Morris JC** (2006). Longitudinal course and neuropathologic outcomes in original vs. revised MCI and in pre-MCI. *Neurology* **67**, 467–473.
- Swainson R, Hodges JR, Galton CJ, Semple J, Michael A, Dunn BD, Iddon JL, Robbins TW, Sahakian BJ** (2001). Early detection and differential diagnosis of AD and depression with neuropsychological tasks. *Dementia and Geriatric Cognitive Disorders* **12**, 265–280.
- Wimo A, Winbald B, Aguero-Torres H, von Strauss E** (2003). The magnitude of dementia occurrence in the world. *Alzheimer's Disease and Associated Disorders* **17**, 63–67.