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THE DECLINE OF WORKING MEMORY IN ALZHEIMER'S DISEASE

A LONGITUDINAL STUDY

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SUMMARY

A previous study (Baddeley *et al.*, 1986) explored the hypothesis that patients suffering from dementia of the Alzheimer type (AD) are particularly impaired in the functioning of the central executive component of working memory. It showed that, when patients are required to perform 2 concurrent tasks simultaneously, the AD patients are particularly impaired, even when level of performance on the individual tasks is equated with that of age-matched controls. Although the results were clear, interpretation was still complicated by 2 issues: first, the question of comparability of performance on the separate tests between AD and control patients; secondly, the question of whether our results could be interpreted simply in terms of a limited general processing capacity being more taxed by more difficult dual tasks than by the individual tasks performed alone.

The present study followed up the AD and control patients after 6 and 12 mths. We were able to allow for the problem of comparability of performance by using patients as their own control. Under these conditions, there is a very clear tendency for dual task performance to deteriorate while single task performance is maintained. A second experiment varied difficulty within a single task in which patients and controls were required to categorize words as belonging to 1, 2 or 4 semantic categories. There was a clear effect of number of categories on performance and a systematic decline in performance over time. There was, however, no interaction between task difficulty as measured by number of alternatives and rate of deterioration, suggesting that the progressive deterioration in performance shown by AD patients is a function of whether single or dual task performance is required, and is not dependent on simple level of task difficulty.

Implications for the analysis of the central executive component of working memory are discussed.

INTRODUCTION

Impaired memory performance is one of the earliest and most characteristic symptoms of Alzheimer's disease (AD) (McKhann *et al.*, 1984; American Psychiatric Association, 1987; Spinnler and Della Sala, 1988; Wilcock *et al.*, 1989), a symptom that reveals itself both in complaints of lapses of memory in everyday life, and in decrements in the performance of laboratory tasks (Miller, 1977; Wilson *et al.*, 1983; Spinnler *et al.*, 1988). Because the memory deficit is so characteristic, understanding its nature is likely to form an important component of analysing the functional deficit occurring in AD, which in turn may lead to the development of tests which offer early diagnosis.

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The most extensively explored memory deficit is that associated with the amnesic syndrome, the gross impairment in long-term learning that occurs in Korsakoff patients, and in patients with bilateral damage to the temporal lobes, hippocampus and diencephalon (Zangwill, 1946; Milner 1966; Warrington and Weiskrantz, 1970; Butters and Stuss, 1989). Such patients show grossly impaired learning of new material, whether visual or verbal, together with unimpaired short-term memory as measured by digit span or by the recency effect in free recall (Baddeley and Warrington, 1970). The functioning of semantic memory may also be unimpaired, as indicated by retained knowledge of word meanings, and unimpaired speed of access to knowledge of the world, while the capacity to recollect events from well before the onset of the illness may also be relatively normal (Wilson and Baddeley, 1988).

The memory deficit in AD resembles that of the amnesic syndrome in showing a broad impairment in the capacity for new learning, coupled with a relative sparing of the recency effect in free recall (Wilson *et al.*, 1983; Spinnler *et al.*, 1988). The memory deficit in AD is more pervasive than that found in the amnesic syndrome, however, with clear evidence of impairment in access to semantic memory (Chertkow and Bub, 1990) which is typically also accompanied by retrograde amnesia (Kopelman, 1989) and autobiographical amnesia (Dall'Ora *et al.*, 1989). Slowed access to semantic memory and some evidence of retrograde amnesia are, however, not uncommon in any condition where substantial brain damage occurs. Deficits in immediate memory span are shown by AD patients, whether tested verbally using the standard span procedure, or spatially using the Corsi Block Tapping technique (Spinnler *et al.*, 1988).

This deficit of AD patients in short-term or working memory has been explored in some detail (Miller, 1973; Morris, 1984, 1986; Kopelman, 1985). Reviewing this and other data, Morris and Baddeley (1988) suggest that a deficit in the controlling central executive component of working memory may be characteristic of AD patients, and may lie at the root of many of their cognitive processing deficits. A similar position is taken by Becker (1988), who suggests that AD patients suffer from 2 principal deficits, one involving an impairment in new learning similar to that shown in the classic amnesic syndrome, while the other represents a defect in the functioning of the central executive component of working memory. AD patients showing such a dissociation have been reported by Becker himself (1988) and by Baddeley *et al.* (1991).

The concept of working memory was developed by Baddeley and Hitch (1974) to account for the increasingly complex pattern of data from experiments on short-term memory. They proposed that the existing concept of a unitary short-term memory store should be replaced by the assumption of a working memory system comprising a number of sub-components. Three principal sub-systems were identified, namely the central executive, the visuo-spatial scratchpad and the articulatory loop.

The central executive is assumed to be an attentional control system that has access to long-term memory and is served by 2 subsidiary slave systems, the visuo-spatial scratchpad which is assumed to set up and maintain visual images, and the articulatory or phonological loop, which is assumed to be responsible for setting up and maintaining speech-based material. For a number of reasons, the term phonological loop is now preferred (e.g. Baddeley, 1990) and we shall use this term throughout. The model has been successful in accounting for a range of laboratory studies on normal subjects, and

has also proved useful in accounting for neuropsychological deficits in brain-damaged patients (for reviews *see* Baddeley, 1986; Vallar and Shallice, 1990).

Baddeley (1986) has suggested that the attentional control model of Norman and Shallice (1980, 1986) might provide a good hypothesis for the way in which the central executive operates. One of the functions of the central executive is to coordinate information from a number of different sources, and as such it is assumed to play a role in many cognitive tasks, including those requiring short-term storage. The assumption of a central executive deficit would therefore account for the impairment in AD patients on tests of short-term memory (Corkin, 1982; Kopelman, 1985; Morris, 1986; Spinnler *et al.*, 1988). This contrasts with the finding that patients with a pure amnesia are able to perform short-term memory tasks very effectively (Baddeley and Warrington, 1970; Warrington, 1982).

While the evidence is broadly consistent with the assumption that AD patients have impaired functioning of the central executive component of working memory, many of the results are open to a more general interpretation which simply assumes that the overall information-processing capacity or efficiency (e.g. Craik, 1984) of patients with AD is reduced. One prediction from this would be that anything that increases the demand placed on the patient will have an exaggerated effect on the performance of the AD group. Baddeley *et al.* (1986) attempted to test the central executive deficit hypothesis. They argued that a central executive deficit should lead to an exaggerated impairment in performance when AD patients are required to coordinate performance simultaneously on 2 different tasks. They attemped to avoid the criticism that the AD patients were being excessively overloaded by adjusting the level of difficulty of the primary task, pursuit tracking, so that the overall performance of patients and controls was equivalent. They then combined this with each of 3 secondary tasks. The first of these involved repeatedly uttering the digits 1 to 5, a task that was assumed to place minimal processing demands on the system, other than the requirement to coordinate it with tracking. The second task involved pressing a foot pedal whenever a sound occurred. The third involved combining tracking with concurrent memory span for digits, where the level of difficulty of the digit task was adjusted to a point at which both AD patients and controls were functioning at an equivalent level, and hence were presumably equally heavily loaded. The tasks of adaptive tracking, articulatory suppression, auditory reaction time and digit span were selected so as to minimize the amount of direct competition for specific resources (*see* Baddeley, 1986 and Baddeley *et al.*, 1986 for details).

The performance of a group of mild-to-moderate AD patients was compared with a group of elderly normal controls matched for age, and with a group of young subjects. The AD patients were substantially more impaired by the combined tasks than were the normal elderly, who showed no greater tendency for performance to be disrupted under equated dual task conditions than did the young. Baddeley *et al.* (1986) interpreted their results as supporting the central executive deficit hypothesis. The fact that the young and the normal elderly showed comparable patterns of performance under dual task conditions suggests that adapting the level of difficulty of the constituent tasks is an appropriate control for the effects of general processing load, since the normal elderly did show an impairment in performance of the constituent tasks when they and the young were tested under equivalent conditions.

While the results are consistent with the central executive hypothesis, at least 2 of

the 3 conditions are open to an alternative explanation. First of all, the effects of articulatory suppression were very slight, and did not produce a significant interaction with patient condition (single vs dual task). The effect of concurrent reaction time was much clearer, with the predicted interaction occurring in both the effect of reaction time on tracking and on the effect of tracking on reaction time. However, it could be argued that the reaction time task was considerably more difficult for the AD patients, and that the overall information processing load of tracking combined with reaction time was thus substantially greater for these subjects. The case rests therefore only on the third condition in which cognitive and digit span were combined, with the overall level of performance adjusted so as to make the 2 constituent tasks equally difficult for all 3 groups. Here there was a highly significant interaction between groups and single vs dual task performance, whether this was measured in terms of tracking or in terms of memory span, thus supporting the hypothesis of a central executive deficit.

The present study aims to explore the central executive hypothesis in more detail. Two experiments will be presented. Experiment 1 involved retesting the AD patients studied by Baddeley *et al.* (1986) and their age-matched controls over time. If the central executive deficit is as crucial as suggested, then the requirement to combine 2 tasks should produce an ever-greater deficit as the illness progresses. Such a longitudinal design has the advantage of using each subject as his or her own control, hence avoiding some of the problems of comparability between patients and controls observed in earlier studies. One of the experiments that follow therefore studies the capacity of AD patients to combine tracking with articulatory suppression, simple auditory reaction time and digit span when tested on 3 occasions separated by intervals of 6 mths.

An alternative interpretation, however, might be that anything that makes a task more difficult will differentially penalize AD patients. This can be contrasted with the central executive deficit hypothesis which predicts that certain types of difficulty will be particularly sensitive to the effects of AD. More specifically, it suggests that coordination of 2 concurrent tasks will be one important role of the central executive, and hence that dual task performance should be more sensitive than a simple within-task increase in difficulty.

Experiment 2 therefore addresses the difficulty hypothesis, by studying the effect of difficulty level on a categorization task, both in a cross-sectional and in a longitudinal design. It is well established that categorization becomes increasingly difficult as the number of categories from which a target is selected increases, where difficulty is measured by error rate and reaction time (e.g. Murdock, 1965; Baddeley *et al.*, 1984). If the greater impairment for AD patients with dual tasks (Baddeley *et al.*, 1986) is explained in terms of the general difficulty of the task, we would expect AD performance on the most difficult version of the categorization task to deteriorate over time, much more so than for the easier version of the task.

EXPERIMENT 1: LONGITUDINAL TRACKING

Subjects

AD patients. The initial test (Baddeley *et al.*, 1986) involved a total of 28 AD patients who formed part of a larger sample, described by Della Sala *et al.* (1986), of patients referred to the neurological service in Milan over a 3-yr period. A total of 224 demented people were referred, of whom 129 were provisionally

diagnosed as AD patients on the basis of clinical history, neurological examination, CT scan and laboratory data which were used to exclude other possible dementing illnesses. The formal diagnostic criteria have been set out elsewhere (Baddeley *et al.*, 1986; Della Sala *et al.*, 1986), and are broadly in line with those of NINCDS-ARDA (McKhann *et al.*, 1984) and DSM III-R (American Psychiatric Association, 1987). Of these 129, patients were excluded from the present study if they had a presumptive length of illness longer than 4 yrs (i.e. likely to have a 'severe' disease); if there was a history of alcohol abuse; if there was a history of drug abuse with drugs which possibly affect central nervous system functions; if their score was less than 50% in a temporal orientation task (Benton *et al.*, 1964) or less than 70% on the test devised by Della Sala *et al.* (1984) on information about family members; and if their score on a scale of everyday coping ability, a revised version of the NUDS (Canter *et al.*, 1961), was less than 70%. They were also excluded if they were not currently living in a family setting and needed special care or if they did not live within Milan or its hinterland. Other criteria for inclusion were availability and willingness to be tested (and retested), and the capacity to read and write as measured informally.

When all these selection criteria were applied, the initial sample of 129 referrals reduced to a sample of 28 patients (22%), comprising 12 men and 16 women. These 28 patients all showed a clinical pattern of dementia associated with AD, together with evidence of deterioration over the previous 6-mth period. This sample constituted the 28 patients who took part in the initial study by Baddeley *et al.* (1986). They were subsequently followed up and tested on 2 further occasions, each separated by a gap of about 6 mths. By the third test, the number of patients still participating in the study had dropped to 15, the other 13 subjects having either died or deteriorated to a point at which they were no longer capable of following experimental instructions. Table 1 shows the characteristics of the experimental samples in the 3 testing sessions. Data refer to the first testing session.

The subgroups of 'survivors' did not appear to differ demographically from the other patients, nor did they differ in level of performance on any of the wide range of psychometric and neuropsychological measures taken on these subjects (*see below*).

TABLE 1. FEATURES OF THE EXPERIMENTAL SUBJECTS ENTERING THE STUDY

	Testing sessions					
	I		*II*		*III*	
	AD/pts (n = 28)	*Controls (n = 28)*	*AD/pts (n = 20)*	*Controls (n = 18)*	*AD/pts (n = 15)*	*Controls* –
(i) Tested subjects						
Age (yrs)	64.9 (7.0)	64.0	64.2 (7.6)	64.4 (5.1)	64.2 (7.0)	–
Ed. level (yrs)	9.5 (4.3)	9.2 (3.7)	9.3 (4.6)	9.4 (3.0)	9.8 (4.9)	–
Age at onset (yrs)	63.0 (7.2)	–	62.6 (7.7)	–	62.7 (8.0)	–
Male/female ratio	0.75	0.86	0.54	0.80	0.66	–
Length of illness (mths)	21.2 (14.1)	–	18.9 (14.8)	–	17.6 (16.4)	–
Raven's PM (range 0–48)	18.0 (11.1)	–	17.8 (10.7)	–	19.5 (10.6)	–
Token Test (range 0.36)	24.8 (7.8)	–	25.0 (7.5)	–	26.7 (5.9)	–
Street CT (range 0.14)	4.8 (3.2)	–	4.6 (3.2)	–	4.8 (3.3)	–
			AD/pts (n = 8)	*Controls (n = 10)*	*AD/pts (n = 5)*	*Controls* –
(ii) Drop outs						
Age (yrs)			66.8 (5.7)	64.2 (4.7)	54.2 (8.1)	–
Ed. level (yrs)			10.1 (3.9)	9.3 (3.9)	7.8 (3.3)	–
Age at onset			63.6 (6.3)	–	62.6 (7.5)	–
Male/female ratio			3.0	0.50	0.25	–
Length of illness (mths)			17.0 (10.6)	–	22.8 (8.9)	–
Raven's PM (range 0.48)			20.5 (8.9)	–	12.5 (10.8)	–
Token Test (range 0.36)			25.2 (9.2)	–	18.5 (9.9)	–
Street CT (range 0.14)			4.2 (3.3)	–	4.0 (3.4)	–

Standard deviations are given in parentheses.

Of the 28 initial AD patients 8 dropped out of the study before the second test (*see* Table 1). Two died as a result of stroke or cardiac disease, 1 had developed laryngeal cancer, 1 refused to be retested and 4 were unable to perform the tests because of a general cognitive deficit. One of these was sufficiently severe as to be institutionalized, 1 showed a severe spatial exploration deficit similar to Balint-Holmes' optic ataxia and 2 showed perseverative behaviour, mirroring that found in a frontal syndrome. Between the second test at 6 mths (mean interval 6.8 mths, SD = 1.7), and the third at 1 yr (mean interval 13.9 mths, SD = 1.9), a further 5 patients dropped out. Three of these showed a severe optic ataxia that rendered the tracking task impossible, 1 showed inertia, presumably due to a frontal syndrome that was sufficiently marked to make testing impossible, while a fifth refused to be tested.

Of the 15 remaining patients who made up the longitudinal group, 3 showed a CT scan that was normal given their age, 5 showed gross atrophy, while 7 had minimal-to-moderate atrophy. On a standard neurological examination, 6 patients showed 'release signs' (chiefly glabellar blink and snout reflex) or paratonia. Some degree of motor impersistence was also found in 5 patients, but apart from 1 patient presenting with some degree of extrapyramidal rigidity, there were no other physical signs of neurological impairment.

Performance of the control group was measured on a short psychometric battery which is also shown in Table 1. These tests were included in order to have the opportunity to check, on a wider range of cognitive performance, whether the AD patients dropping out along the longitudinal assessments did so because of a strictly cognitive difference. The tests were Raven's Progressive Matrices (1938) sets A, B, C and D (score range 0–48) as a measure of non-verbal intelligence, the Token Test (De Renzi and Faglioni, 1978) as a test of language comprehension (score range 0–36) and the Street Completion Test (Street, 1931) as a measure of visuo-perceptual ability (score range 0–14). Normative data for these tests have been collected on a sample of 321 normal subjects in a separate study (Spinnler and Tognoni, 1987). These normative data set the median value for these tests at the following scores: Raven's 28.5, Token 33 and Street 7. The inferential score above which fall the scores for 95% of the population are Raven's 15, Token Test 26.5 and Street Test 2.25. Table 1 shows the mean performance for each of these tests for the patients that took part in all three testing sessions. The data shown were all collected on the first testing session. These scores are indicative of intellectual impairment, with 7 of the 15 longitudinal patients scoring at below the fifth percentile level on Raven's Matrices, 9 scoring below this cut-off for the Token Test, and 4 below the fifth percentile of the Street Test. Performance in this psychometric battery suggests that the surviving AD patients were affected by a relatively mild and slow progressive form of the disease. While they showed few differences from the non-surviving patients on the initial test, it would be unwise to assume that they are a typical sample of all AD patients.

Elderly group subjects. While the principal point of interest involved longitudinal changes in performance of the AD patients, it was considered wise to include a group of normal elderly to serve as a baseline control, to ensure that any decrements that were found were truly characteristic of AD patients, and not attributable to inherent variability in test performance. The control subjects comprised normal subjects matched on age and educational background with the AD patients, but free of evidence of present or past nervous, organic or physical disease that might be expected to impair cognitive performance. They comprised 8 men and 10 women and were tested on 2 occasions separated by a delay of 6 mths (mean interval 6.6 mths, SD = 1.4). They were not retested a third time. Demographic characteristics are given in Table 1.

Experimental procedure

Tracking. The primary task presented to all subjects required them to keep a light-sensitive stylus (light pen) placed on a white square (2×2 cm) moving randomly about the screen of a colour computer monitor. The square remained white as long as the light pen was in contact, but changed to orange immediately contact was lost, returning to white when contact was regained. In the initial *adaptive* phase, the square initially moved quite slowly, with the speed gradually increasing until a point was reached at which the subject failed to maintain contact for more than 60% of the time. At each speed, performance was summed over a period of 20 s before being increased.

The process was controlled by an Apple II computer and took about 4 min to identify the point at which performance approximated 60% time-on-target.

The subject was then given 3 20 s periods of tracking at the 60% time-on-target level. If the subject improved to beyond 60%, then the difficulty level was adjusted and 3 further 20 s trials were given. This procedure continued until the subject's performance had stabilized at some point between 40% and 60%

time-on-target. This completed the *adaptive* tracking phase, and the final level of difficulty was then used for the remainder of that session. The same level of difficulty was used on the retests 6 and 12 mths later, without any readaptation. This was done to allow us to assesss any decrement in performance over time as measured by time-on-target. Subsequent runs in each test session involved combining tracking with other tasks. Since the task was potentially tiring, test runs were limited to 2 min, with the monitor screen at an angle of 30 degrees from the horizontal, since this was found to be less physically tiring than attempting to track on a vertical screen. The standard tracking task was then combined with 3 concomitant tasks in an order that was counterbalanced across tasks, and that was equivalent for AD patients and control subjects.

Concomitant tasks

Articulatory suppression. Subjects were required to count from 1 to 5 repeatedly at a regular rate of approximately 2 per s. This rate was demonstrated by the experimenter who monitored the subject's performance, encouraging speeding up if articulation rate dropped.

Reaction time to tones. Subjects were presented with a sequence of clearly audible tones from a loudspeaker, and required to press a footswitch as rapidly as possible in response to each tone. Inter-tone interval varied randomly between 4 s and 6 s, producing between 23 and 25 tones within each 2 min test run. On each session, the tones began a few seconds before the start of the 2 min run so as to ensure that the subject was performing the reaction-time task. Reaction time and any missed signals were recorded by an Apple II computer.

Memory span. Memory span was first established using the standard auditory digit-span technique in which subjects were read out a list of digits at a rate of 1 per s and asked to repeat them back in the same order. Testing began with a single digit, and was incremented by 1 digit until a point was reached at which the span was exceeded. Subjects were presented with 3 sequences at each length, and testing ceased when the subject was unable to recall 2 of the 3 sequences. For the purpose of the present study, the subject's span was assumed to be 1 digit less than this. Hence if a subject had been successful on all occasions at length 6 but had failed 2 of the 3 at length 7, he would subsequently be presented with sequences of 6 digits. Subjects were then tested for a 2 min period during which they performed the span test alone, and for a further 2 min during which they combined digit span with tracking. In each case, performance was measured in terms of the percentage of sequences that were recalled completely correctly. The number of lists presented during the 2 min test depended of course on the length of the individual subject's span, and ranged from 11 to 15 sequences. As with tracking, the digit memory task can be adjusted to a level of difficulty appropriate for the ability of each individual subject. The span was reassessed on each of the 3 test sessions. By recording the number of errors produced by subjects, we have a means to equate initial group performance.

To summarize, subjects were required to perform the tracking task alone, followed by the 3 dual task conditions in counterbalanced order. The subjects who remained testable on the subsequent test sessions were tested in the same counterbalanced order, as were the age-matched controls.

Results

Demographic and psychometric data from the AD patients were subjected to a series of one-way ANOVAs to determine whether any of the subject variables measured on the first test session could distinguish between patients who could be retested and those who could not, either on the second or third testing session. The 4 patients who could not be retested for reasons unrelated to severity were not included in these analyses. The variables tested were age, years of formal education, length of illness, performance on the Raven's Matrices, performance on the Token Test and performance on the Street Test. The analyses revealed that none of these measures significantly discriminated between these subgroups of patients.

We carried out a similar series of analyses on our single and dual task performance measures. Once the level of significance had been adjusted for multiple comparisons, only one of the measures significantly discriminated between the groups, namely response time to tones when these were combined with tracking [$F(1, 20) = 11.17$; $P < 0.005$]. The mean RT for the group tested on all 3 occasions was 828 ms, and for the 'drop-outs' was 1298 ms.

Next, we considered the performance measures from the 3 different testing sessions. The principal prediction of the central executive deficit hypothesis is that as dementia progresses, its effect on dual task performance should be disproportionately greater than its effect on performance of the constituent task. Therefore the main analyses considered the extent to which the difference between single and dual task

performance interacted with test session. It is possible that performance on any task will deteriorate over time with AD patients. In order to investigate this possibility, we also analysed data over 3 test sessions for the response to tones task when performed alone. Finally, we carried out subsidiary analyses on the 20 AD patients who performed on test sessions 1 and 2. In all cases, the mean data for this group were very similar to those for the subgroup who completed all 3 test sessions. Therefore only the data for this latter subgroup are reported.

Tracking and articulatory suppression. Table 2 shows the mean performance of the AD patients and the normal controls over successive tests. In order to illustrate changes in dual task performance over time, Fig. 1 shows the dual task tracking performance expressed as a percentage of single task performance.

TABLE 2. MEAN TRACKING PERFORMANCE, AS MEASURED BY PERCENTAGE TIME ON TARGET, AS A FUNCTION OF PATIENT GROUP, EXPERIMENTAL CONDITION AND TEST SESSION

	Test session		
	I	*II*	*III*
AD patients			
Tracking alone	59.8	61.8	65.5
+ suppression	54.0	48.3	47.8
+ tones	51.7	46.9	37.7
+ digit span	42.2	34.9	30.1
Normal elderly			
Tracking alone	54.3	54.9	–
+ suppression	52.4	53.3	–
+ tones	53.3	51.5	–
+ digit span	48.4	46.6	–

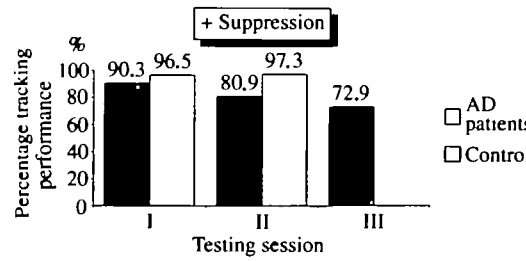

FIG. 1. Tracking performance with concurrent articulatory suppression as a percentage of single task performance for Alzheimer patients and normal elderly subjects.

In the case of the AD patients who completed all 3 test sessions, analysis of variance indicated a significant main effect of suppression [$F(1, 14) = 39.5, P < 0.001$] together with the predicted interaction between the presence of suppression and test session, with suppression having an ever-increasing effect on performance as the disease progresses [$F(2, 28) = 4.4, P < 0.025$]. When we considered the data for the larger groups of patients who completed only test sessions 1 and 2, there was again a significant disruption by suppression [$F(1, 19) = 18.45, P < 0.001$], but there was no effect of test session and no interaction. With the normal elderly there was no significant effect of suppression on tracking [$F(1, 17) < 1$], and no effect of test session [$F(1, 17) < 1$].

Tracking and response to tones. Fig. 2 shows the effect of reacting to auditory tones on tracking performance as a function of test session, expressed as a percentage of single task performance. The mean performance of AD patients and elderly controls over successive tests are shown in Table 2. Over 3 test sessions, once again the AD patients show both a clear effect of tones on tracking [$F(1, 14) = 81.64$, $P < 0.001$], and an interaction of this effect with test session [$F(2, 28) = 18.57$, $P < 0.001$].

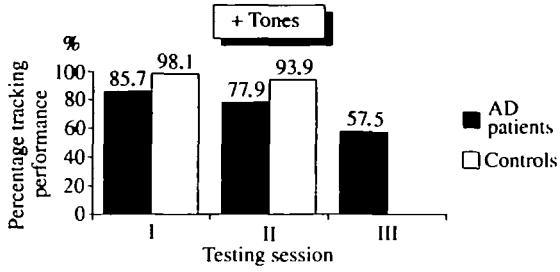

FIG. 2. Tracking performance with concurrent reaction to tones as a percentage of single task performance for Alzheimer patients and normal elderly.

Over 2 test sessions, the ANOVA revealed a significant effect on tracking of responding to tones [$F(1, 19) = 34.76, P < 0.001$], an effect that interacted with test session [$F(1, 19) = 5.18; P < 0.05$]. There was not a significant effect of test session overall. In the case of the normal elderly, there was no significant effect of tones on tracking, and no interaction with test session, and no effect of test session overall [$F(1, 17) = 1.915$, 1.106 and 0.061, respectively].

Table 3 shows the mean reaction times to auditory tones with and without tracking. Unfortunately, the timing data for 1 AD patient was not recorded, reducing the number to 14 for this analysis. There was a main effect of concurrent tracking on reaction time [$F(1, 13) = 28.77, P < 0.001$], an effect of test session [$F(2, 26) = 7.63, P < 0.01$] and an interaction between the tracking effect and session [$F(2, 26) = 5.51, P < 0.01$]. The ANOVA on single task performance revealed that there was no tendency for response time to change over time ($F < 1$). This confirms that the change over time was due solely to performance on the dual task condition.

TABLE 3. MEAN RESPONSE TIME (MILLISECONDS) AND MEAN NUMBER OF TONES MISSED AS A FUNCTION OF PATIENT GROUP, EXPERIMENTAL CONDITION AND TEST SESSION

	Test session		
	I	*II*	*III*
AD patients			
Response time alone	767	704	746
+ tracking	828	1011	1184
Missed tones alone	1.7	3.9	4.5
+ tracking	7.7	13.0	16.1
Normal elderly			
Response time alone	398	418	–
+ tracking	461	503	–
Missed tones alone	0.0	0.2	–
+ tracking	0.2	0.3	–

Turning back to a comparison of single and dual task performance, there was no overall difference in response time between sessions 1 and 2 ($F < 1$). However, there was a highly significant effect on reaction time of concurrent tracking with the 18 subjects for whom these data were recorded [$F(1, 17) = 16.96$; $P < 0.001$], an effect that interacted with test session [$F(1, 17) = 4.44$; $P < 0.05$].

In the case of the normal elderly, there was a significant effect of tracking on reaction time [$F(1, 17) = 34.56, P < 0.001$], but no effect of test session [$F(1, 17) = 2.99$] and no interaction between the effect of tracking and test session [$F(1, 17) = 1.82$].

Table 3 also shows the mean number of errors of omission in which subjects failed to respond to a tone. In the case of the AD patients, concurrent tracking significantly increased the probability of missing a signal [$F(1, 14) = 57.09, P < 0.01$]. Missed signals increased as a function of test session

[F(2, 28) = 11.96, $P < 0.001$], an effect that interacted with tracking [F(2, 28) = 3.88, $P < 0.05$]. An analysis of single task performance indicated that the number of missed tones did not alter over time [F(2, 28) = 2.64].

Over 2 test sessions, the number of missed tones increased [F(1, 19) = 16.39; $P < 0.001$], and also increased when the response task was combined with tracking [F(1, 19) = 41.20; $P < 0.001$]. However, these variables did not interact [F(1, 19) = 2.06].

The normal elderly subjects showed a significant effect of concurrent tracking on the tendency to miss signals [F(1, 17) = 4.50, $P < 0.05$], but this effect did not increase over sessions [F(1, 17) = 1.51], nor was there an interaction between the effect of tracking and session (F < 1).

Tracking and concurrent digit span. Table 2 shows the mean tracking with and without concurrent digit span for AD patients and the elderly controls. Fig. 3 shows dual task performance on tracking as a percentage of single task tracking performance. In the case of the AD patients, concurrent span significantly impaired

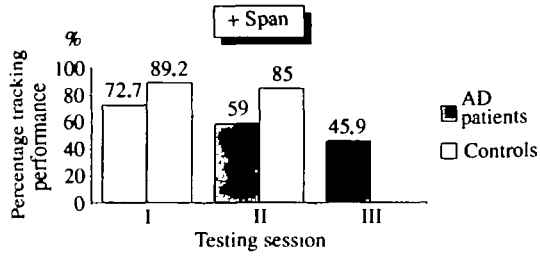

FIG. 3. Tracking performance with concurrent digit span as a percentage of single task performance for Alzheimer patients and normal elderly subjects.

tracking [F(1, 14) = 110.5, $P < 0.001$]. There was not a significant effect of test session on performance (F < 1), but there was a significant interaction between the effect of concurrent load and test session [F(2, 28) = 8.05, $P < 0.01$].

For the 20 AD patients who completed just 2 test sessions, there was a significant drop in tracking performance when combined with span [F(1, 19) = 81.49; $P < 0.001$], an effect that interacted with test session [F(1, 19) = 8.48; $P < 0.01$], but there was no effect of test session overall (F < 1).

In the case of the normal elderly, concurrent span had a significant effect on tracking [F(1, 17) = 14.19, $P < 0.01$], but there was no effect of test session (F < 1), and no interaction between the concurrent digit effect and test session [F(1, 17) = 1.71].

Table 4 shows the effect of tracking on digit span errors. In the case of the AD patients, there was a main effect of tracking on errors [F(1, 14) = 285.96, $P < 0.001$], a main effect of test session [F(2, 28) = 12.44, $P < 0.001$] and a significant interaction between the tracking effect and test session [F(2, 28) = 8.22, $P < 0.01$]. Over 2 test sessions, there was an effect on span performance when this was combined with tracking [F(1, 19) = 158.53; $P < 0.001$]. In addition, span performance was poorer on the second test session [F(1, 19) = 6.41; $P < 0.05$], but this did not interact with the effect of concurrent tracking [F(1, 19) = 2.71].

Digit span was assessed on each of the 3 test sessions. Mean memory span, indicating the point at which performance ceased to be perfect, did not decline significantly, being 4.2, 4.3 and 4.1 for sessions 1, 2 and 3, respectively [F(2, 28) = 1.77]. However, there was a small but significant increase over time in the number of errors made subsequently when subjects were performing the span task alone, as shown in Table 4 [F(2, 28) = 3.76; $P < 0.05$].

The normal elderly showed a significant effect of concurrent span on tracking [F(1, 17) = 38.52, $P < 0.001$], but no effect of test session (F < 1) and no interaction between the tracking effect and test session (F < 1).

Discussion

Our results indicate that articulatory suppression, concurrent reaction time and concurrent digit span all disrupt tracking in AD patients, and that the extent of this disruption increases systematically over 3 successive tests, separated by 6-mth intervals. The influence of such secondary tasks on tracking in the

TABLE 4. MEAN ERRORS IN DIGIT SPAN PERFORMANCE AS A FUNCTION OF PATIENT GROUP, EXPERIMENTAL CONDITION AND TEST SESSION

	Test session		
	I	*II*	*III*
AD patients			
Errors in span alone	1.5	5.3	6.3
+ tracking	39.1	48.4	62.8
Normal elderly			
Errors in span alone	3.8	4.1	–
+ tracking	22.3	22.6	–

normal elderly group was consistently less, and showed no evidence of increasing over successive tests. As such, the results are consistent with the hypothesis that AD patients characteristically suffer from an impairment in their ability to coordinate performance on 2 tasks, and this is consistent with these patients having a deficit of the central executive component of working memory.

The tracking task was chosen to involve the operation of the visuo-spatial scratchpad. The 3 concurrent tasks were chosen so as to have little or no direct loading on this component of working memory, and hence as tasks which will interfere with tracking only in as far as they and tracking make demands on the central executive.

In the case of articulatory suppression it may be recalled that it was not sufficient to produce any significant impairment in the earlier study (Baddeley *et al.*, 1986), nor does it have any impact on tracking in the normal elderly tested in the present study. This is consistent with the fact that repeatedly counting from 1 to 5 is a relatively undemanding overlearned task that loads principally the phonological loop system. Also, it has little influence on the operation of the visuo-spatial scratchpad which is heavily involved in tracking performance (Baddeley and Lieberman, 1980). As dementia progresses, however, even the undemanding task of articulatory suppression is sufficient to interfere progressively more with concurrent tracking. It seems implausible to assume that articulatory suppression involves an increase in spatial processing over time, and hence the obvious interpretation is that even the relatively minor load of coordinating tracking with an overlearned counting task is sufficient to cause substantial impairment in performance. These results are in line with those of Morris (1984, 1986) who observed that the operation of the phonological loop was not qualitatively impaired in AD patients. However, articulatory suppression was sufficient to cause

TABLE 5. ACCURACY AND SPEED OF SEMANTIC CATEGORIZATION AS A FUNCTION OF PATIENT GROUP AND NUMBER OF CATEGORIES

	Number of categories		
	1	*2*	*4*
AD patients			
Correct (max. = 32)	27.2	23.8	23.1
Misses	2.6	4.6	5.5
False alarms	2.2	3.6	3.4
Correct RT	1588	2030	2335
Normal elderly			
Correct (max. = 32)	31.6	31.3	30.7
Misses	0.1	0.0	0.6
False alarms	0.4	0.7	0.6
Correct RT	620	741	918

For the misses, there was a main effect of group: [$F(1, 58) = 48.94$, $P < 0.001$], and an effect of category number: [$F(2, 116) = 12.51$, $P < 0.001$], and these variables interacted: [$F(2, 116) = 6.98$, $P < 0.001$]. *Post-hoc* comparisons supported the group difference ($P < 0.01$), and the interaction. The means for the control group did not differ. The mean for 1 category in the Alzheimer group differed from that for 2 categories ($P < 0.01$) and for 4 categories ($P < 0.01$). The difference between 2 and 4 categories was not significant.

For the false alarms, there was a significant difference between the groups: [$F(1, 58) = 13.24$, $P < 0.001$]; and a significant increase with category number: [$F(2, 116) = 5.21$, $P < 0.01$]. These variables did not interact: [$F(2, 116) = 2.17$]. *Post-hoc* comparisons supported the difference between the groups in that all the means for the AD patients were poorer than those for the normal elderly ($P < 0.01$).

Next, we conducted the same analysis for the correct response times. The mean data are shown in Table 5. There was a main effect of group: [$F(1, 58) = 126.78$, $P < 0.001$]; a main effect of number of categories: [$F(2, 116) = 64.29$, $P < 0.001$]; and a significant interaction: [$F(2, 116) = 12.56$, $P < 0.001$]. Here, for the control group, there appeared to be an increase in response time with increasing number of categories (1 vs 4 categories, $Q = 6.45$, $P < 0.01$). However, the *post-hoc* comparison for 1 vs 2 categories was not significant. The Alzheimer group produced much slower responses overall, with all the means significantly slower ($P < 0.01$) than those for the controls. The Alzheimer patients were much more severely affected by increasing the number of categories as evidenced by the significant groups by category interaction. Also, all of the means for each category number were significantly different from one another: 1 vs 2 categories ($Q = 9.58$; $P < 0.01$), 1 vs 4 categories ($Q = 16.19$; $P < 0.01$), and 2 vs 4 categories ($Q = 6.61$; $P < 0.01$).

Longitudinal study

Subjects

AD patients. Of the 30 patients included in the cross-sectional study, 15 (6 males and 9 females), were retested formally at a mean interval of 7.4 mths (SD = 1.8). The 'drop-outs' in 2 cases were due to death. In 3 cases, patients became too severely ill to be retested, that is they did not understand the test instructions. In 2 cases the patients became easily distracted, and their scores were considered unreliable. Two patients became aphasic and alexic, and therefore could not read the words correctly, and 3 of them showed frontal signs that rendered the results unreliable because of a tendency to perseverate. Finally, 3 patients were unwilling to be retested.

For the remaining 15 patients, their mean age at the time of the first test sessions was 64.8 yrs (SD = 7.5, range 51–80), their mean educational level was 10.0 yrs (SD = 4.7, range 5–17) and their mean length of illness was 18.7 mths (SD = 14.5, range 3–48).

Control group. To match the 15 patients, 15 normal elderly controls were chosen randomly from the original group of 30, and 14 of these agreed to be retested. The control group mean age was 65.5 yrs, range 57–76, and their mean educational level was 10.5 yrs, range 5–17. The mean interval between testing sessions 1 and 2 was 6.3 mths (SD = 0.9).

Results

As for Experiment 1, data from the AD patients and the normal elderly were subjected to a series of one-way ANOVAs to determine whether any of the subject variables measured on the first test session could distinguish between patients who could be retested and those who could not. As before, the variables tested were age, years of formal education, length of illness, performance on the Raven's Matrices, performance on the Token Test, and performance on the Street Test. The analyses revealed that none of these measures significantly discriminated between groups.

A similar analysis carried out on categorization performance indicated that neither number of correct responses nor mean response time significantly discriminated when retested from 'drop-out' subjects.

The mean number of correct responses for test sessions 1 and 2 for 15 Alzheimers are shown in Table 6 and Fig. 4. These data were entered into a two-way ANOVA, with 2 repeated measures, namely test session and number of categories. This analysis showed that the demented subjects deteriorated over time: [$F(1, 14) = 6.42$, $P < 0.05$], and performance was poorer with a larger number of target categories: [$F(2, 28) = 8.08$, $P < 0.01$]. However, there was no indication of an interaction between these variables ($F < 1$).

TABLE 6. ACCURACY AND SPEED OF CATEGORIZATION AS A FUNCTION OF PATIENT GROUP, NUMBER OF CATEGORIES AND TEST SESSION

		Number of categories		
	Test session	*1*	*2*	*4*
AD patients				
Correct (max. = 32)	1	27.9	25.3	24.7
	2	25.5	23.3	21.8
Misses	1	2.3	3.5	4.0
	2	3.3	5.2	6.1
False alarms	1	1.8	3.2	3.3
	2	3.3	3.8	4.1
Correct RT	1	1613	1855	2195
	2	1821	2206	2347
Normal elderly				
Correct (max. = 32)	1	31.6	31.3	31.2
	2	31.6	31.4	31.4
Misses	1	0.1	0.0	0.1
	2	0.0	0.1	0.2
False alarms	1	0.4	0.8	0.6
	2	0.4	0.4	0.4
Correct RT	1	607	784	937
	2	620	783	972

The data for number of errors, both the number of missed target items and number of false alarms are shown in Table 6. An analysis of variance on misses revealed that errors increased with a larger number of categories [$F(2, 28) = 6.23$; $P < 0.01$], but there was no effect of test session [$F(1, 14) = 3.4$] and no interaction ($F < 1$). For the false alarms, there was no tendency for these to change according to number of categories [$F(2, 28) = 2.1$], no effect of test session ($F < 1$) and no interaction ($F < 1$).

The mean correct response times also are shown in Table 6. An analysis of these data showed that the

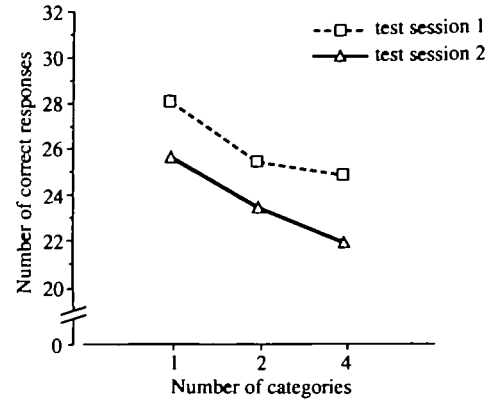

FIG. 4. Mean number of correct categorization responses for 15 Alzheimer patients on 2 test sessions.

demented subjects were slower with a larger number of target categories: [$F(2, 28) = 26.83$, $P < 0.01$]. There was no slowing of responses over time: [$F(1, 14) = 2.9$], and no interaction ($F < 1$).

The data for the 14 normal elderly control subjects also are shown in Table 6. For the number of correct responses, it is evident from the table that the control subjects were at ceiling, and therefore formal analyses were considered neither necessary nor appropriate. An analysis of variance on correct response times revealed a significant effect of number of categories [$F(2, 26) = 104$; $P < 0.001$], but no effect of test session ($F < 1$), and no interaction ($F < 1$).

Discussion

The cross-sectional data suggest that increasing the number of categories increases difficulty as measured by response times for both groups, and by accuracy for the AD patients; control subjects made virtually no errors. This pattern is consistent with earlier work (e.g. Yntema, 1963), and suggests that the task is suitable for studying the effect of within-task difficulty on the rate of cognitive deterioration. The clear impairment in performance on this task of the AD group suggests that it is suitable for deciding whether the longitudinal decrement shown in Experiment 1 reflects the vulnerability of the central executive component of working memory, or is simply a reflection of a tendency for rate of performance decrement to increase with task difficulty.

The longitudinal response time data for the group of normal elderly indicated that their performance on this task was stable over time. Both the normal elderly and the demented subjects were affected by the increase in number of categories. In addition, the demented subjects performed more poorly overall on the second test session, as measured by number of correct responses. There was, however, no significant deterioration in response time over test sessions, possibly due to the high variability in this measure for the AD group, although it is also possible that the demented subjects were maintaining their response time at the expense of accuracy.

The data for the demented subjects showed a clear effect on performance of an increase in task difficulty as the number of target categories increased. However, the magnitude of this effect of task difficulty did not increase as the disease progressed. Moreover, there was no indication that this lack of an interaction with time could be due to a floor effect in AD performance. Looking at the data from individual subjects, only 1 of the 15 patients who were retested performed close to chance level, and this occurred only on the second test session. Taken together, these findings support the view that the results for Experiment 1 cannot easily be explained only in terms of task difficulty.

The lack of an interaction of difficulty with time in the dementia patients is a crucial aspect of these data. A possible alternative interpretation of our results from Experiment 1 was that the AD subjects were simply more sensitive to the effects of task difficulty than to the requirement to coordinate dual task performance. This interpretation of our results for AD patients' dual task performance now appears much less convincing.

Any *post-hoc* interpretation in terms of the relative difficulty of different tasks necessarily involves the question as to how difficulty might be measured. A within-task manipulation of task difficulty, such as

that used in the categorization task, seems, in part at least, to avoid this risk of circularity. It suggests that the concept of difficulty probably is not sufficient to explain the results of Experiment 1, nor the results of Baddeley *et al.* (1986).

GENERAL DISCUSSION

The results of Experiment 1 showed that demented patients have a particular difficulty with dual task performance, and that this disadvantage becomes more pronounced with the progression of the disease. Notably, performance on the single tasks used in this study did not show this same degree of sensitivity to the deterioration of cognitive ability over time. The findings from Experiment 2 mitigated the possibility that the results from Experiment 1 were due to the overall greater difficulty of dual tasks compared with single tasks, and that the AD patients were simply more sensitive to task difficulty than were the normal elderly. This reinforced the importance of the dual task nature of the tasks used in Experiment 1.

The results of these 2 experiments therefore support the view that AD patients suffer from a deficit in their ability to cope with the cognitive processing necessary for carrying out 2 tasks simultaneously. These results coupled with the conclusions of Baddeley *et al.* (1986), Morris (1986), Becker (1988) and Spinnler *et al.* (1988) are consistent with the hypothesis of a deficit in Alzheimer's disease of the central executive component of working memory.

The central executive is essentially concerned with attentional control. To what extent could other models of attention account for our results? One such model assumes that subjects have a limited pool of attentional resources, with greater task demands using more of this capacity leading to impaired performance when 2 tasks compete (e.g. Kahneman, 1973). While this view was influential in the 1970s, in recent years it has become increasingly clear that it is oversimplified (Wickens, 1984). In particular, it has difficulty in accounting for evidence suggesting separable modular sub-systems. A verbal task such as articulatory suppression can be shown to impair other verbal tasks such as digit span, or verbal reasoning, but has little effect on visuo-spatial tasks such as pursuit tracking or tests of spatial manipulation (Baddeley and Lieberman, 1980; Farmer *et al.*, 1986; Logie *et al.*, 1990). Moreover, when applied to our own data, such an interpretation would presumably predict an interaction with difficulty for both the dual tasks studied in Experiment 1, and the unitary tasks used in Experiment 2; the results of Experiment 2 are not in agreement with this prediction.

One development from the earlier limited resources model has been concerned with the concept of automaticity, the tendency for certain types of skill to reduce their attentional demands as learning proceeds. This allows such skills to be performed with little interference from other concurrent tasks (Schneider and Shiffrin, 1977). It has been argued (Jorm, 1986) that such automatized skills are particularly resistant to the effects of Alzheimer's disease. However, the concept of automaticity is consistent with any attentional model that assumes limited processing capacity, including the working memory model. Furthermore, the concept of automaticity does not bear directly on the interpretation of our present results, which depend on the performance of relatively novel tasks for which there is likely to be little automatization.

One can argue that the dementing process in AD calls for progressively more control resources, even for tasks that in the normal elderly brain continue to run

quasi-automatically (Spinnler, 1991). This increasing demand on the central executive functions of time- and accuracy-sharing in AD patients renders them particularly sensitive to tasks and conditions whose 'difficulty' stems from their central executive involvement (Jorm, 1986).

In recent years, the concept of a single attentional resource pool has tended to be replaced by multiple resource theory, which assumes a number of specialized pools of processing resources, that may act either independently or in concert, depending on task demands (Wickens, 1984). It is usual for multiple resource models to assume that when 2 tasks must be performed at the same time, there is a 'cost of concurrence' (Navon and Gopher, 1979). One way of interpreting this cost is to assume that dual task coordination requires some form of central attentional processing module, what Hunt and Lansman (1982) refer to as an 'executive time sharer'.

The working memory model is of course a multiple resource system in which executive time-sharing is assumed to be dependent on the central executive. It has the advantage over more generally specified multiple resource models, such as that of Wickens (1984), of attempting to specify the constituent modules in more detail and of linking them closely to neuropsychological evidence (Baddeley, 1986, 1990).

In the case of the central executive, the neuropsychological link has been facilitated by adopting the Norman and Shallice (1980) model of attentional control, which Shallice (1982) has used to interpret the cognitive deficits found in the frontal lobe syndrome. The model assumes that most ongoing actions are controlled by establishing routines, with contention-scheduling procedures operating automatically to minimize conflict between concurrent well-learned skills such as talking and driving. The system is assumed to be monitored by a Supervisory Attentional System (SAS) which is able to override ongoing activities when necessary, for instance when a driver might stop a conversation to ask the way.

Shallice (1982, 1988) suggests that the frontal syndrome (Goldberg and Bilder, 1987; Stuss and Benson, 1987) may reflect damage to the operation of the SAS, leading to problems in the attentional control of behaviour. This produces both perseveration, when the SAS fails to break into the ongoing activity when appropriate, and distractability, when the SAS fails to maintain the desired activity in the face of other distracting sources of information (Shallice *et al.*, 1989). While a full account of both normal attentional control and of the frontal syndrome will clearly demand a more detailed theory, the model has proved useful in further investigating frontal deficits (Shallice, 1982, 1988; Baddeley and Wilson, 1988; Shallice *et al.*, 1989). In the case of AD, we and others have suggested that the capacity to direct and control attentional resources may be one of the principal cognitive deficits observed (Baddeley *et al.*, 1986; Becker, 1988; Morris and Baddeley, 1988; Cossa *et al.*, 1989; Spinnler, 1991). While the working memory model is not the only way of conceptualizing such a deficit, it has so far provided a convenient and fruitful framework for investigating the nature of the observed cognitive decline.

We have described 2 group studies which we have so far discussed as if they reflect a uniform pattern of performance deficits. This is not of course the case for our sample or, we suspect, for any sample of AD patients. The heterogeneity of cognitive deficits found among carefully diagnosed AD patients has often been documented (Capitani *et al.*, 1986; Della Sala *et al.*, 1986; Martin *et al.*, 1986; Becker, 1988; Spinnler and

Della Sala, 1988), and was observed within the group of patients from which the present sample were drawn (Baddeley *et al.*, 1991). Patients were found to differ quite widely in their precise pattern of cognitive deficits, with occasional patients having relatively pure and isolated areas of impairment, although other deficits would typically appear in such patients as the disease progressed. The fact that group studies are able to show an apparently coherent pattern of impairment presumably reflects the fact that certain deficits are particularly prevalent and marked in AD patients, possibly reflecting the nature and distribution of the underlying pathological processes.

We are aware that this deficit may not be unique to AD. It might well be that other conditions resulting from brain damage lead to similar impairments. Nonetheless, in terms of sensitivity to the progress of the disease, our dual task measures appear to have considerable advantages. Our earlier study (Baddeley *et al.*, 1986) has shown that they are sensitive in discriminating the effects of AD from the normal processes of ageing, suggesting that they may provide good measures for both detecting AD and monitoring its progress. This is in agreement with findings reported by other researchers on normal ageing (Wright, 1981; Light and Anderson, 1985; Gick *et al.*, 1988; Morris *et al.*, 1988, 1990) which suggested that normal ageing does not lead *per se* to a substantial impairment of the time-sharing component of working memory. The current experimental procedure is, however, somewhat inconvenient logistically, and we therefore plan to develop and validate a version that will be more suitable for clinical practice.

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