A central executive deficit in patients with Parkinson's disease

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Abstract
Eight patients with Parkinson's disease and eight matched controls were tested for concurrent task performance to examine whether Parkinson's disease produces deficits in the coordinating and integrating function of the central executive component of Baddeley's working memory model. Consistent with this prediction, the patients showed a significant decline in performance on a random pursuit tracking task while recalling digit span forward sequences, whereas the controls showed no such change. Performance on the component pursuit and digit span tasks, which did not differ between groups, was equated across subjects by varying the size of a target square and by using individual subjects' digit spans. The patient group also produced poorer word fluency scores and reported higher levels of depression, but there was no significant impairment on the Wisconsin card sort test. There was no association between dual task performance and any psychometric measure, target size, or disease related variables. Baddeley's working memory model is advantageous in providing a rich conceptual basis to explore and characterise cognitive abilities in patients with Parkinson's disease.

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There are reports that patients with Parkinson's disease show cognitive impairments in a range of tasks that measure "frontal lobe function," attention, memory, or visuospatial capacities. On the whole, however, publications contain a substantial number of apparently conflicting findings.[1][2] Although multiple cognitive deficits may be present in patients with Parkinson's disease Brown and Marsden[2][3] point out that there is now a growing consensus that they are impaired only when the tasks are demanding and effortful, rather than when the task requires automatic responses, and when they must rely on internally generated cues to guide attention and behaviour. Also, Brown and Marsden[2-4] made the specific suggestion that Baddeley's[5][6] working memory model may provide a conceptual framework to accommodate many disparate findings and to provide a theoretical basis for the similar descriptions emerging in published work.

This suggestion is echoed by that of two other groups who proposed either general or specific working memory deficits in patients with Parkinson's disease. Cooper *et al*[7] found that early untreated patients showed deficits on "executive function" tasks, that were "dependent on an intact working memory system" and on certain memory tasks, which suggested that "the single origin of this [memory] impairment may be a reduction in the efficiency of working memory". On a more tenuous note, Della Sala *et al* interpreted a reduced primacy effect coupled with an intact recency effect in medicated patients as evidence for a specific working memory deficit in the context of Baddeley's model.[8]

Working memory is considered important in a range of cognitive tasks and comprises the hypothetical set of information processing systems used to store, integrate and update information, especially when performing multiple cognitive operations. A system called the central executive represents the principal component of Baddeley's[5][6] tripartite model, which was developed to accommodate data that were inconsistent with earlier models of short term memory and to encompass these data within a broader framework of working memory. The central executive, currently viewed as a limited capacity attentional system synonymous with Norman and Shallice's[9] supervisory attentional system, is responsible for controlling non-routine current mental activities by coordinating and supervising subordinate short term memory processes and retrieval of information from long term memory. The two subordinate slave subsystems that complete Baddeley's model are the phonological loop system, responsible for setting up and maintaining speech based information, and the visuospatial sketchpad, which allows the temporary storage and processing of visuospatial information.

In a recent study, Brown and Marsden formally tested the idea that patients with Parkinson's disease may have an impairment at the level of central executive processes.[3] They employed a Stroop paradigm[10] on which their previous research had revealed impairments in these patients when no explicit cue was available to guide reaction times to the word/colour presented, but no impairment with an explicit cue available to guide behavior. Their interpretation of these earlier findings, based on the premise that internally cued tasks are more resource demanding, was that parkinsonian patients have fewer processing resources to allocate to

the central executive/supervisory attentional system for tasks guided by internal cues and sufficient resources for most tasks guided by external cues.[4] In their more recent work, they reasoned that the addition of a resource demanding secondary task performed concurrently with the cued Stroop task would reveal an impairment on this Stroop task in these patients, because the extra demands of the secondary task would then overload the capacity of their central executive.[3] Consistent with this prediction, the patient group showed an increase in cued Stroop reaction times when random number generation was used as the secondary task. Of the two secondary tasks presumed to be less resource demanding than random number generation, concurrent foot tapping produced a smaller impairment on the cued Stroop task in the patient group and concurrent articulatory suppression was without effect. Brown and Marsden interpreted these findings as consistent with a resource depletion model or a difficulty in the strategic allocation of resources[3]; either possibility is consistent with a general central executive impairment.

The ability of a subject to coordinate resource demanding concurrent tasks successfully is a crucial aspect of the function of Baddeley's central executive system. According to Baddeley, this system is "capable of selecting strategies and integrating information from a variety of different sources".[5] The present study made an explicit test of whether these central executive functions are impaired in Parkinson's disease by employing one of the dual task paradigms described by Baddeley et al in their work on Alzheimer's disease.[11] These workers found that the clearest demonstration of a central executive deficit in patients with mild Alzheimer's disease occurred when they performed a pursuit tracking primary task while simultaneously recalling their optimal digits forward span. A follow up study found that this dual task deficit worsened with the progression of the dementia whereas single task performance was maintained.[12]

The tracking/span dual task used by Baddeley et al[11] offers particularly useful advantages for the assessment of central executive function. Firstly, in the context of Baddeley's model, the two tasks employed are especially relevant as each component task is presumed to make separate demands on specific resources of the two slave subsystems. The tracking task is conceived as dependent primarily on the visuospatial sketchpad whereas digit span information is presumed to be processed primarily by the phonological loop system. Thus as tracking and verbal span theoretically produce minimal structural interference, their use in a dual task is especially appropriate for considering the coordinating operations of the central executive. By contrast, both random number generation and Stroop tasks employed by Brown and Marsden are heavily speech based in nature.[3]

The tasks used by Brown and Marsden[3] are primarily related to Baddeley's model in terms of their presumed demands on general processing resources. Random number generation places heavy demands on the central executive,[5 6] and the same probably holds for the Stroop task because it involves a high degree of response conflict.[13] As such, performance during the combination of these tasks provides a good indication of general processing resources or their allocation. A potential complication with Brown and Marsden's study arises, however, from the fact that performance on their component tasks could not be equated across subjects and, whereas no group differences presumably existed on the cued Stroop task (single Stroop performance was not reported), the patient group was already inferior to controls on the number generation task. When one task is performed at a lower performance level by any individual or group of subjects then difficulties with a subsequent dual task may be a reflection of an already increased load on general information processing systems, rather than a reflection of problems related to the issue of the supervisory control characteristics of the central executive.

The second valuable feature of Baddeley's approach was to consider this problem of component task load by adjusting the level of difficulty on each task so that performance could be equated and thereby controlled across subjects irrespective of individual ability. Our study followed Baddeley's use of a digit span procedure as a secondary task on which it is assumed that each subject is performing at his or her optimal level. The way Baddeley et al altered primary task difficulty was to vary the speed of the target in their random tracking task to obtain single task performance levels of 40%-60% time on target for their subjects.[11] By this means, it was assumed that subjects were performing at a similar level below ceiling, to satisfy the objective that the difficulty of the task, and hence the presumed information processing load, was generally comparable across subjects. In our adaptation of their procedure, we also maintained tracking performance at 40%-60% time on target, but equated performance across subjects by varying the size rather than the speed of the target. Our modification meant that the spatial and temporal characteristics of the random target waveform were identical for all subjects. We consider that this procedural modification affords greater assurance that both the rate of information processing demand and the difficulty level of the tracking task were as comparable as possible across all subjects.

Methods
SUBJECTS
The study tested eight patients with idiopathic Parkinson's disease and eight age, socioeconomic, and education matched controls, five of whom were patients' spouses. All patients had been diagnosed by a consultant neurologist and had mild Parkinson's disease of relatively recent onset (mean duration 4·4

(SE 1·03) years). At the time of testing, each patient was rated for severity of symptoms with the Hoehn and Yahr scale.[14] Two patients were classified at Hoehn and Yahr stage I, three at stage II, and three at stage III; the impaired side was predominantly right sided in three subjects, left sided in two subjects, and bilateral in three subjects. All patients were on stable medication levels, taking levodopa and a decarboxylase inhibitor (six patients), plus an anticholinergic (one patient) or a dopamine agonist (one patient), supplemented in some subjects by either selegiline (two patients) or amantadine (two patients). One patient was taking selegiline only and one other was taking selegiline and a dopamine agonist. Subjects were selected from volunteers who were screened on the basis of an interview to ensure that all were maintaining independent function, and that none was showing any clinical sign of dementia or had a history of major neurological, musculoskeletal or psychiatric disorder, or alcohol abuse. Informed consent was obtained from all participants. All subjects reported adequate vision and hearing.

PROCEDURE

In the tracking task, subjects were requested to track a green bordered square target, presented on a computer screen (224 × 184 mm, 800 × 600 pixels resolution), by using a large floor mounted joystick to maintain the centre of a 10 × 10 mm orange cross (arm width, 3·0 mm) inside the target. The subject was seated and the eye to screen distance was 90 cm. The joystick, based on one developed by Kondraske et al,[15] had an 11 cm long grip above a circular disk hand rest. The hand rest was at a height of 63 cm so that the elbow was at about 90 degrees. Subjects were free to choose the hand (one only) they were most at ease with for the tracking task. The tracking task was generated and analysed by a 486 PC with dual monitors, one for the subject and one for the assessor.[16] The horizontal and vertical components of the two dimensional target trajectory were random signals comprising 28 equal amplitude harmonically related sinusoids at 0·007 Hz intervals up to 0·196 Hz. The resultant average speed for a two minute period was 22·7 mm/s, with a maximum of 65·1 mm/s. The transducer sensitivity was 10 mm on screen per degree on joystick in both lateral and fore and aft directions and with both joystick signals sampled at 28·63 Hz (half the screen vertical interrupt rate). Two measures of tracking performance were obtained. One measure was the % time that the subject was able to maintain the centre of the cross inside the borders of the target square (time on target); this was the principal measure given that the design of the experiment was to vary target size to equate performance on this task. The second measure, the mean absolute error, was the mean deviation of the centre of the cross from the centre of the target. This measure provides an index of the actual error or accuracy of a subject when performing the tracking task, assuming that

the subject makes the presumptive optimal strategy of maintaining the centre of the cross near the centre of the target and does not track by constantly maintaining the cross just inside the target, particularly a large one.

Subjects were familiarised with the tracking task by being shown that movement of the target square commenced once the cross had been kept inside the target for six seconds and that the joystick could be used to track the target by maintaining the centre of the cross inside the target. Subjects were told only that correct tracking would entail keeping the centre of the cross inside the target and that the experimenter would take the subject through a few trials to make the target large enough for him or her to be accurate about 50% of the time, after which they would be asked to perform the tracking task while repeating back some numbers. After this initial familiarisation and assurance that the subject understood the tracking task, a number of 20 second practice trials were run. For these trials, the target square was adjusted from a start size of 20–40 mm, depending on the subject's initial ability to track a 20 mm square, to obtain a performance level of 40–60% time on target. Once this performance level had been achieved, three further 20 second trials were run at the appropriate difficulty level (using the adjusted target square size). If the subject's performance seemed to be stable on these three trials then single task tracking for a two minute period was assessed at this difficulty level. If performance on baseline 20 second trials deviated from a stable performance (in the 40–60% time on target range), then the target size was again adjusted and initial tracking reassessed.

The secondary task of digits forward span was then assessed, equating performance across subjects by obtaining the optimal digit span for each subject in the manner described by Baddeley et al.[11] This performance level was obtained by presenting digit sequences of increasing length, starting with three sequences of three digits. When the subject was unable to recall in the correct order the digits of two of the three sequences of any given length, their digits forward span was taken to be the previous sequence length. Having obtained digit span in this manner, single task span performance was evaluated for a two minute period.

The various test periods were brief enough not to cause fatigue problems in any subject. Having assessed performance on the component single tasks, subjects were requested to perform the tracking and digit span tasks concurrently for a two-minute trial, commencing once again when the subject was ready to maintain the cross inside the target. Subjects were given the following instruction by the experimenter: "We are now going to combine the tracking task with the number recall task. As you do the tracking task on the computer, I will ask you to recall some more numbers for me." A check was made to ensure that the subject understood this instruction, but

no suggestion was made as to the priority of either task. The first digit sequence began as the target began to move.

Diagnostic and demographic data were collected before experimental tasks. After these tasks, subjects were examined for categorisation and behavioural regulation on the Wisconsin card sorting test (WCST),[17] assessed on a verbal fluency test in which subjects must generate words starting with the letters F, A, and S over one minute periods,[18] and administered the Beck depression inventory (BDI).[19] One subject pair was not examined on the verbal fluency test and the WCST. Throughout testing, including the experimental work described, the subjects' comfort was ensured and considerable emphasis was placed on the use of adequate breaks to avoid any fatigue as assessed by behavioural observation and response to questions.

Results

Table 1 shows a summary of the demographic and psychometric characteristics of the two subject groups. The two groups were matched for age and years of education. As is commonly found, patients with Parkinson's disease reported more symptoms of depression (BDI: $t = 2.60$, $df = 14$, $p < 0.05$). The patient group also achieved lower scores on the FAS verbal fluency test ($t = 3.53$, $df = 12$, $p < 0.01$). Measures obtained from the

Table 1 Demographic and psychometric characteristics

	Subjects with Parkinson's disease	Controls
Age (y)	65·6(3·0)	62·4 (2·1)
Education (y)	10·1(0·5)	10·1 (0·6)
Duration of Parkinson's disease (y)	4·4(1·03)	
BDI score	11·1(2·5)*	4·3 (0·7)
Digit span	5·3(0·5)	5·5 (0·5)
Tracking target size (mm)	21·3(5·5)	12·4 (2·8)
Word fluency (FAS)	28·0(2·5)**	41·0 (2·8)
Wisconsin card sort task:		
No of categories	2·0(0–6)	3·5 (2–6)
% Total errors	44·7(5·4)	36·7 (2·7)
% Perseverative errors	23·4(7·7)	23·4 (4·9)

*$p < 0.05$; **$p < 0.01$ v controls.
Values are mean (SE) except No of categories which are median (range).

Table 2 Performance on single and dual experimental tasks

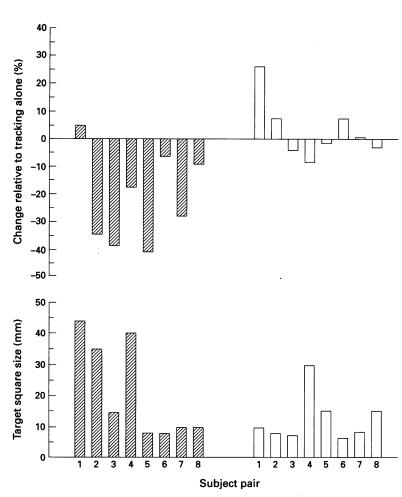|	Subjects with Parkinson's disease	Controls
Tracking:		
% Time on target, single	53·38 (2·94)	57·25 (2·31)
% Time on target, dual	43·25 (4·14)**	58·75 (2·80)
Mean absolute error, single	12·89 (3·73)	6·09 (1·30)
Mean absolute error, dual	15·18 (3·82)**	6·41 (1·59)
Digit span:		
No of sequences recalled, single	12·38 (1·36)	11·50 (1·09)
No of sequences recalled, dual	12·50 (1·28)	11·75 (1·19)
% Correct sequences recalled, single	85·44 (4·74)	81·86 (8·13)
% Correct sequences recalled, dual	76·89 (7·71)	73·35 (8·26)

**$p < 0.002$ v single task tracking.
Values are mean (SE); mean absolute error = mm deviation from target centre.

WCST did not reveal a difference that approached statistical significance, except the number of categories achieved, which would have shown a difference had one patient not obtained the maximum score of 6 (U = 10, $p < 0.10$).

Patients and controls had similar digit spans. In terms of tracking performance, the mean target size used for the two task conditions was greater in the patient group, but this difference did not approach significance ($t = 1.44$, $df = 14$, $p > 0.10$; table 1). The mean value of the single task tracking mean absolute error, the average distance the subject is away from the centre of the target, was also greater in the patient group, but this difference again did not approach significance ($t = 1.72$; $df = 14$; $p > 0.10$), whereas single task % time on target was similar between the two groups (table 2). There was no clear indication, then, that single task performance on either concurrent task differed systematically as a function of group, although it was apparent that some patients were worse at tracking than other patients and most controls (figure). The number of trials to achieve a satisfactory baseline single task tracking performance level also did not differ between groups (mean (SE): patients = 12·38 (0·98), controls = 10·25 (1·08), $t < 1·0$) and it can thus be assumed that both groups had near equal opportunity to experience the tracking task before baseline. Performance during these preliminary trials indicated that most controls required a steady decline of the target size, except one who needed an initial increase first and a second who needed only an increase from an already large target size. The patients were more variable in this respect, three requiring a steady decline in target size, two showing fluctuation then a decline, two needing an increase in an already large value, and one requiring an increase then a decrease in target size.

Table 2 shows performance on the single and dual task tracking and digit span tasks. A 2 × 2 ANOVA (group: patients/control × the repeated measure of condition: single/dual) was computed separately for each measure of tracking performance and for the number of digit sequences presented and the % sequences recalled correctly. In terms of digit span performance, a mean decrease was present in both groups resulting in a condition main effect (F (1,14) = 4·99, $p < 0.05$), but no group effect or group by condition interaction (Fs < 1·0). The number of digit sequences completed by subjects was equal between groups and constant across single and dual task conditions (Fs < 1·0).

By contrast, both measures of pursuit tracking showed clear evidence of a dual task impairment in the patient group. The analysis revealed that patients achieved a significantly lower % time on target over the two conditions (group main effect, F (1,14) = 5·68, $p < 0.05$), but there was also a main effect of condition (F (1, 14) = 6·23, $p < 0.05$) and, importantly, a highly significant group by condition interaction (F(1,14) = 11·31,

Relative % change in tracking (top panel) and corresponding target size used (bottom panel) for patients with Parkinson's disease (▨) and controls (□).

quent analysis of the simple main effects of these tracking error scores revealed that patients and controls did not differ significantly when tracking alone $(F(1,14) = 2.97, p > 0.10)$, and controls did not show any change from tracking alone to tracking while repeating digit sequences $(F < 1.0)$ whereas the patient group showed a highly significant increase in this tracking error score from alone to dual task conditions $(F(1,14) = 17.87, p < 0.002)$.

Neither performance changes on dual task tracking nor digit span showed any suggestion of a relation to any of the other measures obtained from the subjects with Parkinson's disease, including duration of illness, Hoehn and Yahr rating, or parkinsonian medication. As far as other correlations in the patient group were concerned, age was positively correlated with the size of target $(r = +0.72, p < 0.05)$, years of education was negatively correlated with % total errors on the WCST $(r = -0.83, p < 0.05)$, and span size was positively correlated with FAS score $(r = +0.83, p < 0.05)$ and negatively with % total errors on the WCST $(r = -0.74, p < 0.05)$.

Discussion

Our study found that patients with Parkinson's disease were less able than matched controls to coordinate successfully two concurrent tasks in that they showed a clear decline in tracking performance during dual task conditions. Given the relatively small sample size of patients, the effect shown in our study provides strong support for the general suggestion that Parkinson's disease impairs working memory[7] and, in particular, the more specific prediction[2 3 8] that these patients have an inefficient central executive in terms of Baddeley's[5 6] model. Coupled with the close ties between short term memory tasks and working memory as conceptualised by Baddeley, the view proposed here may also provide a basis for the prevalence of short term memory deficits found in patients with Parkinson's disease,[20] including reports that they show a mild impairment on the Brown-Peterson task[7 21] because a central executive deficit is considered a major reason for poor performance on this task.[22] As our patient group exhibited moderate levels of general Parkinsonian deficits with a relatively recent onset, our data suggest that central executive problems exist early in the disease process. This suggestion is reinforced by reports that early untreated patients show a clear pattern of impairments suggestive of working memory deficits[7] and that increasing a cognitive load had disproportionate effects on reaction time in early Parkinson's disease.[23]

The immediate value of our results is to augment the earlier work by Della Sala *et al*[8] and Brown and Marsden.[3] Della Sala *et al* inferred a central executive deficit on the basis of a reduced primacy effect in patients with Parkinson's disease, but they acknowledged that this result is open to several alternative explanations.[8] Brown and

$p < 0.005$). Analysis of the simple main effects of this interaction confirmed that patients and controls did not differ on single task % time on target tracking $(F(1,14) = 1.07)$, but whereas controls did not show a mean change in performance from single to dual task conditions $(F < 1.0)$ the clear finding was that the patient group showed a highly significant decrease in tracking between the single and dual task conditions $(F(1,14) = 17.16, p < 0.002)$. The top panel of the figure shows individual relative % change in the time on target measure of tracking between the single and dual task for all subjects, expressed on the basis of each subject's performance on the single task as representing 100%. The bottom panel of the figure shows the corresponding target size used by each subject. The patients showed a decrease in tracking under dual task conditions that was unrelated to the general difficulty of the task as defined by the size of the target required to maintain performance at about 50% time on target in the single task condition $(r = +0.32, p > 0.10)$. Similar conclusions to those obtained by analysing time on target were evident from the analysis of the mean absolute error measure of tracking performance in that there was a significant condition effect $(F(1,14) = 11.65, p < 0.005)$ and a significant group by condition interaction $(F(1,14) = 6.57, p < 0.05)$. Subse-

Marsden's paradigm was more relevant to the issue of depleted processing resources or resource allocation, but was such that they were unable to equate initial performance on the component tasks used in dual task testing.[3] This problem was compounded by the fact that their patients were already deficient on the random number generation component. Both random number generation and the Stroop task are likely to place heavy demands on central executive resources, plus interference between these tasks may reflect problems in verbal processing itself rather than in some aspect of any superordinate resources. By contrast, our study is more closely related to Baddeley's[5][6] model, as we used his dual task paradigm of tracking combined with digit span recall. These two component tasks are presumed each to rely most heavily on the two major subsystems, the phonological loop system and the visuospatial sketchpad respectively, so that in theory the two tasks exert minimal interference by virtue of their independent actions on the corresponding subsystems. Such a combination of tasks thus places a focus on the supervisory control of working memory. Also, performance levels of all subjects were equated in this paradigm before testing on the dual task. It is therefore unlikely that the dual task impairment we found was the result of taxing resources that had already been depleted excessively by any one task in any one subject. Thus our results, although consonant with a more neutral notion that patients with Parkinson's disease have reduced general processing resources, have the added benefit of being more directly consistent with the hypothesis of a deficit in the integrating and coordinating functions of Baddeley's central executive. Whether this result is a reflection of reduced attentional resources, poorer allocation of resources, or both is a difficult question warranting further study.

It was notable that the dual task impairment was selectively one that affected the visuospatial task and not the verbal task. It is tempting to consider that the source of this difference may reside in the differential psychomotor demands of the two tasks. One way we are considering this issue is to explore the utility of mental rotation as a "non-motor" visuospatial task. At at least one level of difficulty, however—namely, target size used—there was no significant difference between groups, plus the single task time on target and mean absolute error measures were comparable between groups whereas these measures showed a reduction under dual task conditions for the patient group only. The two groups also did not differ in the number of trials used before evaluating baseline performance on the tracking task, although their performance during this time was not identical. One reason, then, to be cautious about the conclusion that the patient group showed a dual task deficit on tracking because of poorer coordination of resources is that we cannot be sure that the tracking task was learned equally well by the two groups. The

controls' dual task tracking could have been less disrupted by the secondary verbal task because their performance may have been improving at a more steady rate than that of the patients. Tracking performance was relatively stable just before single task testing but we cannot rule out whether any subtle systematic changes were occurring. We also acknowledge that our procedure does not exclude the possible influence of classical order effects such as fatigue, although we consider the extent of such effects to be negligible. Suffice to say that, in terms of a time on target measure or speed in making a skilled action, there is some evidence that motor skill learning may differ between parkinsonian patients and controls,[24][25] whereas other evidence indicates that only those parkinsonian patients with clinical signs of dementia show obvious deficits in learning psychomotor skills when an adjustment of the speed of a target is made to equate initial time on target.[26][27]

A second, related explanation for a dual task deficit emerging on the tracking task only may be that the tracking task, but not the verbal task, demanded more "effort" on the part of the patients to maintain performance at the required difficulty index. Thus, if patients were already using more attentional resources for tracking than were controls, the resource demands of the dual task condition may be more evident on tracking, it being the more sensitive of the two tasks. If this assertion is correct, then the claim of a general central executive deficit is weakened in favour of resource difficulties specific to a psychomotor task. The lack of a relative impairment in the patient group on the verbal task used in the present study also raises the suggestion that the findings may reflect different strategies used by the two groups or problems with the time sharing aspects of central executive functions in the patient group, rather than a simple reflection of poorer coordination of central executive processes. Both groups, however, showed a decline on the verbal task, so any obvious notion that a differential trade off occurred between concurrent tasks is more consistent with the behaviour of the controls, not the patient group. It is also relevant that the number of digit sequences recalled did not vary as a function of single and dual task conditions and was similar for both groups on both occasions, so that the demands of the verbal task were ostensibly comparable between groups and across tasks. A more general concern is the assertion that visuospatial tasks are more likely than verbal tasks to place demands on general processing resources even in neurologically unimpaired subjects.[28][29] If this view is correct (but see Logie and Baddeley[30] for an alternative viewpoint), it suggests that performance decrements on visuospatial rather than verbal tasks are more likely in subjects or patients for whom central resources are a problem.

The contentious issue of whether patients with Parkinson's disease have a selective visuospatial impairment has received considerable

attention.[1][2] The study on visuospatial functions by Bradley et al[31] is the most pertinent one to our current concern with the structural relevance of Baddeley's model to cognition in Parkinson's disease. These workers employed a verbal (letter identification) memory task and a visuospatial (route following) imagery task, each combined with and without "unattended" verbal and "unattended" visual interference conditions along the lines of those described by Baddeley[5] and Logie[32] respectively. They claimed that the visuospatial subsystem of working memory is selectively impaired in Parkinson's disease, on the basis that only the patient group showed increased response latencies in their visuospatial imagery task compared with the verbal task. Their remaining data did not, however, provide clear support for this conclusion, although the possibility of other visuospatial deficits remains an open issue. Firstly, their patients and controls were equal in making more errors on the visuospatial task. Secondly, no conclusions can be derived from their use of the interference conditions as these were completely without effect on either task in either group. The third and especially problematic result was that both groups had equivalent visual memory spans. As suggested by Bradley et al an alternative to their preferred viewpoint is that the relatively more difficult imagery task presumably placed heavy demands on the internal generation of the relevant cues, as would be expected in Parkinson's disease if this disorder disrupts information processing efficiency or depletes available resources. This suggestion, of course, is compatible with Brown and Marsden's view that a deficit exists at the level of central executive processes in these patients.

It would be valuable to have more evidence on the relation between central executive/working memory deficits and other variables. The present study does not permit any firm conclusions on this matter and it is feasible that such associations will be more apparent in studies that employ much larger sample sizes to accommodate the variability in performance evident in patient and control groups. Yet it is pertinent that there was no association between cognition and depression in the large sample employed by Cooper et al,[7] so the lack of an association between dual task performance and depression in our study may be a robust finding. It is less clear, however, whether dual task deficits are related to frontal measures such as verbal fluency and card sorting in parkinsonian patients. A dysfunctional central executive is also envisaged by Baddeley[5][6] as a reason to expect impairments on tasks that are sensitive to frontal lobe dysfunction. Although the evidence is far from unequivocal, such deficits are often reported in studies on parkinsonian patients.[2][7] Our study found poorer performance only on verbal fluency in the patient group, and no association between either frontal measure and dual task performance. Again, these findings may reflect sample size,

but this is an important issue that merits further investigation because it may suggest possible dissociations within central executive functions as has been speculated by Baddeley.[6]

Our work was inspired by the demonstration by Baddeley and colleagues[11][12] of central executive deficits in patients with Alzheimer's disease and it would be especially interesting to compare dual task performance in these patients with that of parkinsonian patients showing clinical signs of dementia. Judging by comparative work on explicit and implicit memory tasks, many interesting similarities and differences exist between patients with these two neurodegenerative disorders and dementia, and multiple cognitive impairments may be present in many of these patients.[26][33] For example, deficits in procedural memory tasks in parkinsonian patients[34] may have a basis distinct from any working memory problem. Yet it seems likely that a comparative approach also promises to implicate further elaborations of the nature of central executive functions and their role in neurological disorders that have associated cognitive deficits.

As pointed out by Brown and Marsden,[2] the utility of a working memory approach to cognitive impairments in patients with Parkinson's disease is that it currently provides a unifying hypothesis for many disparate published results, including mixed results on "set shifting" and visuospatial tasks. A focus on the central executive is yet more appealing, as this embodies a specific notion of attentional control in action and memory, and is consistent with several suggestions that attentional control is a problem for this patient group.[11][23][35][36] Our data encourage us to agree with Morris et al[37] that neither the locus of control (internal v external) idea nor a simple attention switching hypothesis provide an adequate explanation of relevant published research and that a better explanation can be found in suggesting a deficient supervisory system. An obvious and potentially fruitful contender in this respect is the central executive hypothesised by Baddeley. Clearly, Baddeley's working memory model provides a rich conceptual basis to explore and characterise cognitive abilities in patients with Parkinson's disease.

1 Brown RG, Marsden CD. 'Subcortical dementia': the neuropsychological evidence. *Neuroscience* 1988;25: 363–87.
2 Brown RG, Marsden CD. Cognitive function in Parkinson's disease: from description to theory. *Trends Neurosci* 1990;13:21–9.
3 Brown RG, Marsden CD. Dual task performance and processing resources in normal subjects and patients with Parkinson's disease. *Brain* 1991;114:215–31.
4 Brown RG, Marsden CD. Internal versus external cues and the control of attention in Parkinson's disease. *Brain* 1988;111:323–45.
5 Baddeley AD. *Working memory.* London: Oxford University Press, 1986.
6 Baddeley AD. *Human memory: theory and practice.* New York: Allyn and Bacon, 1990.

7 Cooper JA, Sagar HJ, Harvey NS, Jordan N, Sullivan EV. Cognitive impairment in early, untreated Parkinson's disease and its relationship to motor disability. *Brain* 1991;114:2095–122.

8 Della Sala S, Pasetti C, Sempio P. Deficit of the "primacy effect" in Parkinsonians interpreted by means of the working memory model. *Arch Suisses Neurochir Psychiatr* 1987;138:5–14.

9 Norman DA, Shallice T. Attention to action: Willed and automatic control of behavior. In: Davidson RJ, Schwarts GE, Shapiro D, eds. *Consciousness and self-regulation. Advances in research and theory*, Vol 4. New York: Plenum Press, 1986:1–18.

10 Stroop JR. Studies of interference in serial verbal reactions. *J Exp Psychol* 1935;18:643–62.

11 Baddeley A, Logie R, Bressi S, Della Sala S, Spinnler, H. Dementia and working memory. *Q J Exp Psychol* 1986; 38A:603–18.

12 Baddeley A, Bressi S, Della Sala S, Logie R, Spinnler H. The decline of working memory in Alzheimer's disease. *Brain* 1991;114:2521–42.

13 Dalrymple-Alford EC, Azkoul J. The locus of interference in the Stroop and related tasks. *Perception and Psychophysics* 1972;11:385–8.

14 Hoehn MM, Yahr MD. Parkinsonism: onset, progression and mortality. *Neurology* 1967;17:427–42.

15 Kondraske GV, Potvin AR, Tourtellotte WW, Syndulko K. A computer-based system for automated quantification of neurologic function. *IEEE Trans Biomed Eng* 1984;31:401–14.

16 Watson RW, Jones RD. The development of PC-based two-dimensional tracking tasks for the study of neurological disorders [abstract]. In: *Proceedings of the annual conference of New Zealand Branch of the Australasian College of Physical Scientists in Medicine;* 1992; Christchurch.

17 Heaton RK. *Wisconsin card sort test manual.* Florida: Psychological Assessment Resources Inc, 1981.

18 Beck AT, Steer RA. *Beck depression inventory manual.* San Antonio: Harcourt Brace Javanovich Inc, 1987.

19 Lezak M. *Neuropsychological assessment.* 2nd ed. New York: Oxford University Press, 1983.

20 Sullivan EV, Sagar HJ. Double dissociation of short-term and long-term memory for nonverbal material in Parkinson's disease and global amnesia. A further analysis. *Brain* 1991;114:893–906.

21 Tweedy JR, Langer KG, McDowell FH. The effects of semantic relations on the memory deficit associated with Parkinson's disease. *J Clin Neuropsychol* 1982;4: 235–47.

22 Morris RG, Baddeley AD. Primary and working memory functioning in Alzheimer type dementia. *J Clin Exp Neuropsychol* 1988;10:279–96.

23 Zimmermann P, Sprengelmeyer R, Fimm B, Wallesch CW. Cognitive slowing in decision tasks in early and advanced Parkinson's disease. *Brain Cogn* 1992;18: 60–9.

24 Frith C, Bloxham DA, Carpenter KN. Impairments in the learning and performance of a new manual skill in patients with Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1986;49:661–8.

25 Soliveri P, Brown RG, Jahanshahi M, Marsden CD. Effect of practice on performance of a skilled motor task in patients with Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1992;55:454–60.

26 Heindel WC, Salmon DP, Shults CW, Walicke PA, Butters N. Neuropsychological evidence for multiple implicit memory systems: a comparison of Alzheimer's, Huntington's and Parkinson's disease patients. *J Neurosci* 1989;9:587–92.

27 Bondi MW, Kaszniak AW. Implicit and explicit memory in Alzheimer's disease and Parkinson's disease. *J Clin Exp Neuropsychol* 1991;13:339–58.

28 Phillips WA, Christie DFM. Interference with visualization. *Q J Exp Psychol* 1977;29:637–50.

29 Christie DFM, Phillips WA. Simple drawing and pattern completion techniques for studying visualization and long-term knowledge. *Memory and Cognition* 1979;7: 360–7.

30 Logie RH, Baddeley AD. Imagery and working memory. In: Richardson J, Marks D, Hampson P, eds. *Imagery: current developments.* London: Routledge, Kegan Paul, 1990:103–28.

31 Bradley VA, Welch JL, Dick DJ. Visuospatial working memory in Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1989;52:1228–35.

32 Logie RH. Visuospatial processing in working memory. *Q J Exp Psychol* 1986;38A:229–47.

33 Baddeley AD, Della Sala S, Spinnler H. The two-component hypothesis of memory deficit in Alzheimer's disease. *J Clin Exp Neurol* 1991;13:372–80.

34 Saint-Cyr JA, Taylor AE, Lang AE. Procedural learning and neostriatal dysfunction in man. *Brain* 1988;111: 941–59.

35 Cossa F, Della Sala S, Spinnler H. Selective visual attention in Alzheimer's and Parkinson's patients: memory- and data-driven control. *Neuropsychologia* 1989;27: 887–92.

36 Sharp MH. Distractability in early Parkinson's disease. *Cortex* 1990;26:239–46.

37 Morris RG, Downes JJ, Sahakian BJ, Evenden JL, Heald A, Robbins TW. Planning and spatial working memory in Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1988;51:757–66.