

# Cerebellar damage produces selective deficits in verbal working memory

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**The cerebellum is often active in imaging studies of verbal working memory, consistent with a putative role in articulatory rehearsal. While patients with cerebellar damage occasionally exhibit a mild impairment on standard neuropsychological tests of working memory, these tests are not diagnostic for exploring these processes in detail. The current study was designed to determine whether damage to the cerebellum is associated with impairments on a range of verbal working memory tasks, and if so, under what circumstances. Moreover, we assessed the hypothesis that these impairments are related to impaired rehearsal mechanisms. Patients with damage to the cerebellum ( $n = 15$ ) exhibited a selective deficit in verbal working memory: spatial forward and backward spans were normal, but forward and backward verbal spans were lower than controls. While the differences were significant, digit spans were relatively preserved, especially in comparison to the dramatic reductions typically observed in classic ‘short-term memory’ patients with perisylvian brain damage. The patients tended to be more impaired on a verbal version compared to a spatial version of a working memory task with a long delay and this impairment was correlated with overall symptom and dysarthria severity. These results are consistent with a contribution of the cerebellum to rehearsal and suggest that inclusion of a delay before recall is especially detrimental in individuals with cerebellar damage. However, when we examined markers of rehearsal (i.e. word-length and articulatory suppression effects) in an immediate serial recall task, we found that qualitative aspects of the patients’ rehearsal strategies were unaffected. We propose that the cerebellum may contribute to verbal working memory during the initial phonological encoding and/or by strengthening memory traces rather than by fundamentally subserving covert articulatory rehearsal.**

**Keywords:** verbal working memory; cerebellum; dysarthria; spatial memory

**Abbreviations:** SMA = supplementary motor area

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## Introduction

Working memory—the ability to maintain task-relevant information actively in mind—is achieved through the engagement of a widely distributed network of cortical and subcortical areas. Regions within the frontal and parietal cortices as well as the cerebellum consistently show increased activation in neuroimaging studies of working memory and much research has been dedicated to exploring the functional contribution of these regions (Paulesu *et al.*, 1993;

Schumacher *et al.*, 1996; Jonides *et al.*, 1997; Honey *et al.*, 2000; Glabus *et al.*, 2003; Crottaz-Herbette *et al.*, 2004). Dorsolateral prefrontal (DLPFC; D’Esposito *et al.*, 1999), ventrolateral prefrontal (VLPFC, BA 45; Thompson-Schill *et al.*, 2002), and superior parietal cortices (BA 7; Marshuetz *et al.*, 2000) are thought to support domain-general processes important for working memory (such as updating items and maintaining sequential order), whereas the right and left

inferior parietal lobes are thought to sustain domain-specific representations of the information to be maintained (Paulesu *et al.*, 1993; Smith *et al.*, 1996; Jonides *et al.*, 1998; but see Ravizza *et al.*, 2004). Ventrolateral prefrontal cortex (Broca's area), premotor regions and the cerebellum have been hypothesized to be essential for the rehearsal of items that are being actively remembered (Paulesu *et al.*, 1993; Awh *et al.*, 1996).

While considerable effort has been devoted to examining functional differences between areas associated with control processes (D'Esposito *et al.*, 1999; Henson *et al.*, 2000; Marshuetz *et al.*, 2000), less attention has been given to defining the contributions of regions assumed to be involved in rehearsal. The experiments reported in this paper focus on the contribution of the cerebellum to verbal working memory. We begin by reviewing neuroimaging and neuropsychological evidence that suggests a role for the cerebellum in verbal working memory. We then turn to the question of how the cerebellum's contribution to verbal working memory and, in particular, rehearsal may differ from that of Broca's area. We emphasize this contrast because much of the neuropsychological and neuroimaging research on articulatory rehearsal in verbal working memory has focused on Broca's area.

### Neuroimaging studies

In one highly influential model of working memory (Baddeley and Hitch, 1974; Baddeley, 2000), an articulatory rehearsal process is assumed to 'refresh' information maintained within a phonological store. Broca's area and the cerebellum are two regions implicated in such a rehearsal process, partly because of their known contributions to speech production (Duffy, 1995). Consistent with such an interpretation, the engagement of these areas in verbal working memory tasks is similar, regardless of whether stimulus presentation is visual (Fiez *et al.*, 1996; Paulesu *et al.*, 1996; Smith *et al.*, 1996; Honey *et al.*, 2000) or auditory (Schumacher *et al.*, 1996; Majerus, 2003). Additionally, in a study of rote rehearsal, the magnitude of activity in each of these areas was associated with recall performance (Davachi *et al.*, 2001). Moreover, these regions display greater activity in verbal working memory tasks when the control condition requires covert speech (Petrides *et al.*, 1993; de Zubicaray *et al.*, 1998; also see Awh *et al.*, 1996). In general, the pattern of activation is quite similar in Broca's area and the cerebellum.

Two notable discrepancies in the activity of these regions are suggested by a few studies, however. First, activity in the cerebellum, but not Broca's area, is modulated by the length of the items to be remembered (Grasby *et al.*, 1994; Chein and Fiez, 2001) and secondly, Broca's area activity is sustained across the delay period in a delayed serial recall task, whereas cerebellar activation was primarily restricted to the encoding phase (Chein and Fiez, 2001). This latter result is at odds with the idea that the cerebellar contribution to working memory is associated with covert rehearsal. The goal of this study was to use neuropsychological methods to provide an alternative

characterization of the role of the cerebellum in working memory.

### Neuropsychological studies

Both patients with lesions of the left ventral prefrontal cortex and the cerebellum exhibit poor articulatory control over their utterances (for a review see Ravizza, 2001). Although lesions to either region can result in articulatory impairments, neuropsychological studies, in general, have confirmed the involvement of the left prefrontal cortex in verbal working memory tasks, but have not provided strong evidence of cerebellar involvement.

In a case study of a prefrontal patient, Vallar *et al.* (1997) reported a marked impairment of verbal working memory and no impairment of spatial working memory. This patient had a lesion medial to Broca's area in the left anterior insula, premotor cortex and the internal capsule. Consistent with the putative involvement of left prefrontal regions in articulatory rehearsal, TO performed more poorly when there was a delay before recall. Moreover, he exhibited two diagnostics of an impaired rehearsal process: he had a reduced word-length effect and his performance was unaffected when he was prevented from rehearsing during the delay period (i.e. articulatory suppression). In contrast, a presumed index of phonological storage, the phonological similarity effect (see Conrad and Hull, 1964; Baddeley, 1966) was not reduced for this patient. These findings were replicated in a larger study of Broca's aphasics (Goerlich *et al.*, 1995).

In contrast to patients with left prefrontal lesions, patients with cerebellar lesions have not, in the main, demonstrated impairments of verbal working memory. Many studies have reported normal verbal spans for cerebellar patients (Bracke-Tolkmitt *et al.*, 1989; Fiez *et al.*, 1992; Daum *et al.*, 1993; Bürk *et al.*, 2003; Globas *et al.*, 2003; Harrington *et al.*, 2004), whereas other studies have reported mild impairments on standard digit span tasks (Ravizza and Ivry, 2001; Witt *et al.*, 2002; Maddox *et al.*, 2005). In other working memory tasks, cerebellar patients have exhibited normal increases in reaction time with set size (Appollonio *et al.*, 1993) and scored equivalent 'miss' rates in a verbal 2-back task although the false alarm rate was higher (Gottwald *et al.*, 2003; see results when hospital inpatients were removed from the analysis).

Silveri *et al.* (1998) have provided a detailed case study of a patient who had a right-hemisphere cerebellar tumour resected. The patient had a reduced verbal span but not a reduced spatial span 2 weeks after surgery. Similar to frontal patients, he showed a normal phonological similarity effect with auditory presentation. In contrast to frontal patients, the cerebellar patient displayed an effect of articulatory suppression (reduced performance when required to perform a distractor articulation task during the delay interval). An assessment of word length was ambiguous given that both the patient and the control participants failed to show a word-length effect. The patient's performance on both verbal and spatial tasks was within the normal range 5 months after surgery.

## Specifying cerebellar contributions to verbal working memory

Although Broca's area and the cerebellum are consistently active in neuroimaging studies of verbal working memory, patients with lesions of the cerebellum are considerably less affected on tests of verbal span than patients with lesions that encompass Broca's area (and, usually, surrounding cortex). This raises the question as to whether cerebellar activity in imaging studies of verbal working memory really signifies an essential contribution to working memory, such as a role in rehearsal. The cerebellar contribution may be more general, perhaps reflecting a role in sustained attention (Gottwald *et al.*, 2003), attentional shifting (Courchesne and Allen, 1997) or response preparation (Ivry and Fiez, 2000). Indeed, a study comparing neural regions that were active in both attention and verbal working memory tasks (LaBar *et al.*, 1999) reported overlapping regions in the right cerebellum whereas Broca's area was active only in the verbal working memory task.

The following report provides a systematic evaluation of the effects of cerebellar lesions on tests of working memory. We have focused on patients with unilateral lesions since it has been argued that bilateral damage due to atrophy may encroach upon brainstem function (Harrington *et al.*, 2004). The first of three experiments was designed to document the working memory deficits of a large group of patients with cerebellar lesions, and further showed that the impairment was selective for verbal information. In the second experiment, we examined whether this dissociation was dependent on output requirements by using a span-matching procedure. In the final experiment, we tested the rehearsal hypothesis by examining how the patients' performance was affected by manipulations of word-length and articulatory suppression.

If rehearsal processes are disrupted in cerebellar patients, these manipulations should have less of an effect on cerebellar patients than control subjects.

## Experiment 1

To assess the effect of cerebellar lesions on working memory, patients with unilateral cerebellar lesions were tested on digit and spatial span tasks. A selective impairment on the former would be consistent with the hypothesized role of the cerebellum in verbal working memory that has emerged from the neuroimaging literature. However, if verbal and spatial spans are reduced, a problem at the level of executive control would be implicated.

## Methods

### Participants

Fifteen patients with damage restricted to the cerebellum and fifteen healthy control subjects participated in this experiment for monetary compensation (see Table 1 and Fig. 1). All patients scored in the normal range (>23 points: Folstein *et al.*, 1975) on the Mini-Mental State Exam (average = 28.5; range 26–30) and all control participants reported no neurological abnormalities. Cerebellar damage reflected either a focal lesion caused by an ischaemic event to the left ( $n = 6$ ) or right ( $n = 4$ ) cerebellar hemisphere or resection of a tumour/cyst in the left ( $n = 3$ ) or right ( $n = 2$ ) cerebellar hemisphere.

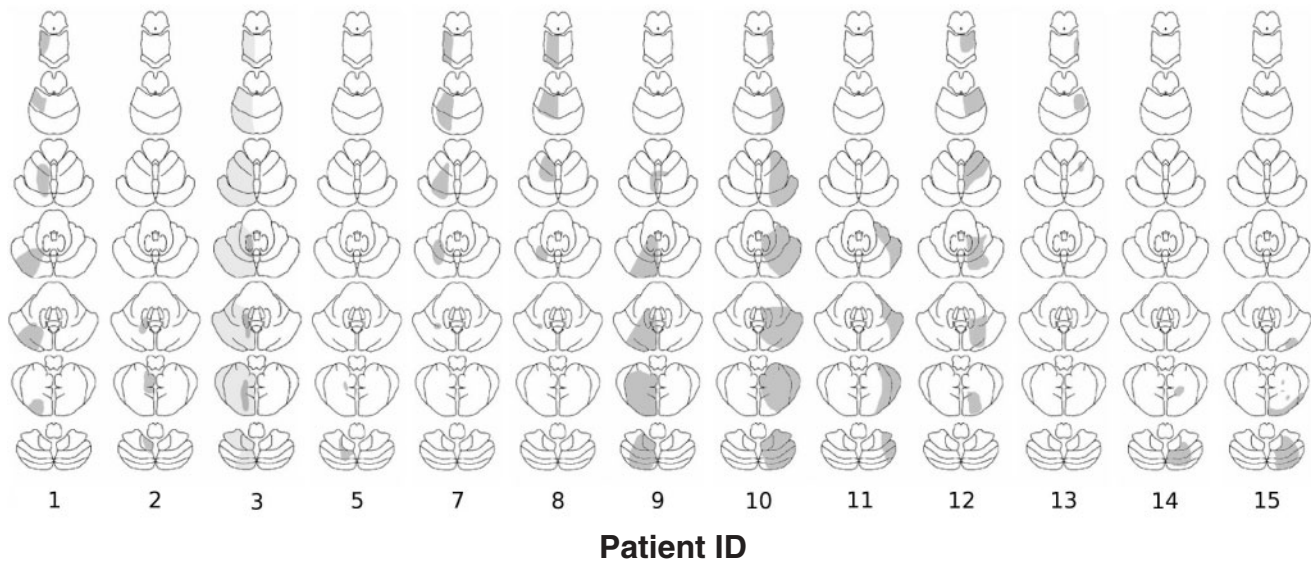
### Stimuli

The digit span and spatial span/block tapping subtests of either the WAIS III (Wechsler Adult Intelligence

**Table 1** Demographic characteristics of cerebellar patients in all experiments and for control participants in Experiments 1–3

| Patient         | Age  | Education | Type  | Aetiology                   | Expts | % Lesion (total) | % Lesion (lobules I-V) | % Lesion (lobules VI-VII) | % Lesion (lobules VIII-X) |
|-----------------|------|-----------|-------|-----------------------------|-------|------------------|------------------------|---------------------------|---------------------------|
| 1               | 45   | 16        | Left  | Stroke                      | I     | 24.69            | 37.44                  | 38.92                     | 12.85                     |
| 2               | 58   | 12        | Left  | Tumour resection            | I     | 26.85            | 0                      | 14.96                     | 62.29                     |
| 3               | 57   | 11        | Left  | Subarachnoid cyst resection | I     | 63.58            | 60.92                  | 65.93                     | 60.56                     |
| 4               | 68   | 18        | Left  | Stroke                      | I–2   | *                | *                      | *                         | *                         |
| 5               | 79   | 21        | Left  | Stroke                      | I–3   | 6.71             | 0                      | 0                         | 9.91                      |
| 6               | 52   | 16        | Left  | Tumour resection            | I–3   | *                | *                      | *                         | *                         |
| 7               | 70   | 12        | Left  | Stroke                      | I–3   | 25.54            | 70.05                  | 21.63                     | 0                         |
| 8               | 83   | 12        | Left  | Stroke                      | I–3   | 24.43            | 64.46                  | 32.30                     | 0                         |
| 9               | 76   | 11        | Left  | Stroke                      | I–3   | 18.90            | 0                      | 6.08                      | 60.61                     |
| 10              | 45   | 18        | Right | Stroke                      | I     | 61.79            | 43.43                  | 69.64                     | 56.11                     |
| 11              | 48   | 18        | Right | Tumour resection            | I     | 40.11            | 1.30                   | 42.60                     | 35.90                     |
| 12              | 77   | 16        | Right | tumour resection            | I     | 40.77            | 65.01                  | 48.52                     | 10.79                     |
| 13              | 64   | 12        | Right | Stroke                      | I–3   | 7.15             | 7.98                   | 6.69                      | 0                         |
| 14              | 46   | 14        | Right | Stroke                      | I–3   | 13.69            | 0.00                   | 0.00                      | 28.27                     |
| 15 <sup>†</sup> | 37   | 12        | Right | Stroke                      | I–3   | *                | *                      | *                         | 43.14                     |
| Pat. avg.       | 60.3 | 14.6      |       |                             |       |                  |                        |                           |                           |
| Con. avg.       | 61.2 | 15.2      |       |                             |       |                  |                        |                           |                           |

SCA = spinocerebellar atrophy. \*Scans unavailable for analysis. <sup>†</sup>Only partial image available.



**Fig. 1** Location of cerebellar damage for 13 of the 15 patients.

Scale—Third Edition; Wechsler, 1997) or WMS-R (Wechsler Memory Scale—Revised; Wechsler, 1987) were used to assess verbal working memory, depending on the standard neuropsychological battery used at each testing site (CA or PA).

### Procedure

In compliance with standard testing procedures, digits were spoken by the experimenter at the rate of 1/s and participants were asked to recall the digits immediately in either a forward or backward order. For the block tapping test, the experimenter tapped the squares at different locations printed on the stimulus card at the rate of 1/s. Patients were instructed to tap the blocks in the same or reverse order immediately after the experimenter indicated he/she was finished. For the WAIS-III verbal and WMS-R spatial tasks, the starting length was two items; for the WMS-R verbal task, the starting length was three items. For all tasks, testing ended when the participant erred on both trials of a given length. As with the standard procedure, correct responses required that the participant recall all of the items in the correct order.

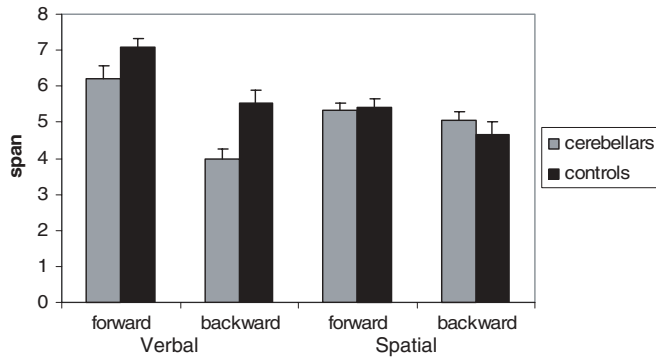
### Lesion analysis

For 13 of 15 patients, medical MRI ( $n = 10$ ) or CT ( $n = 3$ ) scans were available for analysis. Films were digitally scanned and resampled to  $1 \times 1$  mm in-plane resolution, with a slice thickness of 2.5 mm ( $n = 1$ ), 5 mm ( $n = 8$ ), 6 mm ( $n = 1$ ), 8 mm ( $n = 1$ ) or 10 mm ( $n = 2$ ). Cerebella were segmented by an experienced anatomist (J.S.) using the open source software 'NVM' (freely available from Neuromorphometrics, Inc. at <http://neuromorphometrics.org:8080/nvm/>). Healthy cerebellar tissue (grey matter, white matter and deep nuclei, excluding lesion) was outlined using isointensity contours

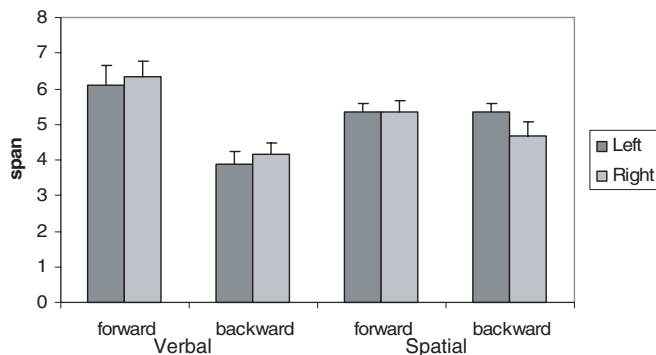
with manual correction. Outlines were then subdivided by hand along the midline, primary fissure and prepyramidal/preinverter fissure with the aid of an MRI atlas of the human cerebellum (Schmahman *et al.*, 2000). This resulted in measurements of the anterior lobe (lobules I–V), superior lobe (lobules VI–VII) and inferior lobe (lobules VIII–X). The measure of 'percent lesion' was calculated from the volume disparity between the healthy and lesioned hemispheres divided by the volume of the healthy hemisphere. Healthy cerebella have previously been measured and found symmetric within 5% using similar methodologies (Makris *et al.*, 2003).

### Results

To measure span length, we used the largest number of items a participant was able to reproduce accurately as our dependent variable. This was a conservative measure of potential span deficits since participants could fail one of the two lists at a given length and it would not be reflected in their score. A 2 (verbal/spatial)  $\times$  2 (forwards/backward)  $\times$  2 (cerebellar/control) mixed-factor ANOVA (analysis of variance) yielded main effects of task and direction (Fig. 2). However, these were qualified by two significant interaction effects. First, there was an interaction of task by direction [ $F(1,28) = 29.42, P < 0.001$ ], indicating that recalling items in the reverse order was more detrimental for the verbal task compared to the spatial task. More important, the task by group interaction was significant [ $F(1,28) = 9.01, P < 0.005$ ]. *Post hoc t*-tests indicated that cerebellar patients' verbal spans were reduced compared to controls [ $t(28) = 3.12, P < 0.005$ ], whereas spatial span was of a normal length [ $t(28) = 0.78, P = 0.44$ ]. The three-way interaction was significant [ $F(1,28) = 5.06, P < 0.05$ ] and inspection of Fig. 2 suggests that cerebellar patients tended to be more impaired on the digits backward than forward subtest



**Fig. 2** Largest list length that control participants and cerebellar patients were able to recall (forwards and backwards) in the digit and spatial span tests of Experiment 1.



**Fig. 3** Largest list length that cerebellar patients with damage to the left or right hemisphere were able to recall (forwards and backwards) in the digit and spatial span tests of Experiment 1.

than controls, while spatial spans were equivalent to controls regardless of direction of recall.

Lesion size was not reliably associated with performance on any of the subtests. To assess whether there were laterality differences in verbal and spatial spans, the same  $2 \times 2 \times 2$  mixed-factor ANOVA was performed comparing left- versus right-hemisphere cerebellar patients (Fig. 3). Neither the main effect of group [ $F(1,13) = 0.02$ ,  $P = 0.91$ ] nor any interactions with group were significant. Damage in the inferior cerebellar lobe, regardless of laterality, was reliably associated with performance on the digits backward test [ $r(1,11) = -0.63$ ,  $P < 0.05$ ]; that is, greater damage to this region was associated with lower backward digit spans. This was true for both left- ( $r = -0.87$ ) and right-hemisphere patients ( $r = -0.07$ , but if one outlier removed  $r = -0.64$ ). Forward digit span also displayed a negative correlation with damage to the inferior lobe, although the relationship was not significant [ $r(1,11) = -0.4$ ,  $P = 0.176$ ]. In contrast, both spatial tasks were positively correlated with damage to this region. Damage to the anterior and superior lobes was not reliably associated with any of the subtests.

To determine the extent to which verbal and spatial spans were associated, we calculated the correlation between the verbal and spatial forward subtests. These correlations,

while positive, failed to reach significance for either the control participants [ $r(1,13) = 0.27$ ,  $P = 0.32$ ] or the cerebellar patients [ $r(1,13) = 0.14$ ,  $P = 0.61$ ]. These values are slightly higher or lower for controls and patients, respectively, than that reported in the test norms ( $r = 0.19$ , Wechsler, 1987) for individuals in the same age range as our participants (55–64).

## Discussion

Experiment 1 provides strong evidence of an impairment in working memory in patients with lesions of the cerebellum. Moreover, this impairment appears to be selective for verbal working memory tasks; spatial spans were equivalent to those of healthy control participants whereas verbal spans were lower in both the forward and backward direction. This effect cannot be attributed to a difference in task difficulty. First, the verbal task was easier than the spatial task for the controls. Secondly, the patients' impairment on the verbal span tasks was observed on the easiest (verbal forward) and hardest test (verbal backward), at least as measured by their performance.

Although this result is consistent with neuroimaging evidence showing cerebellar activation during verbal working memory tasks, it is unclear why previous patient studies have yielded inconsistent results. Most of the studies have involved a small number of participants (Bracke-Tolkmitt *et al.*, 1989; Fiez *et al.*, 1992). However, even in studies involving larger groups of patients (e.g. 14 in Bürk *et al.*, 2003), performance was comparable to controls on verbal span tasks. Other differences between studies may account for variability in cerebellar performance such as using more meaningful word lists (Daum *et al.*, 1993; Globas *et al.*, 2003) rather than digits or letters, or including patients with damage not restricted to the cerebellum (Bürk *et al.*, 2003). It may also be the case that measures of overall span are not the most sensitive technique for assessing subtle, more qualitative changes in verbal working memory (Justus *et al.*, 2005). Moreover, previous studies may not have included patients with damage to the inferior cerebellum which was important for verbal working memory performance in our study and in previous neuroimaging studies (Desmond *et al.*, 1997; Chen and Desmond, 2005).

It is important to note that even in the current study, the patients' verbal spans were quite good and fell within the normal range based on standardized norms (Wechsler, 1987, 1997). The deficit here was apparent in comparison to age- and education-matched control participants. The mild impairment evidenced in our group contrasts with the severely constricted span observed in patients with temporoparietal damage (range of 2–4 items) (Warrington and Shallice, 1969; Vallar and Baddeley, 1984; Martin and Saffran, 1997; Vallar *et al.*, 1997). Perhaps the number of regions assumed to contribute to rehearsal [e.g. Broca's area, left premotor cortex, supplementary motor area (SMA) and cerebellum] versus the solitary region associated in the literature with storage functions (e.g. inferior parietal cortex) influences the degree to which lesions in one area can

be compensated by preserved function in another area. For instance, the SMA or Broca's area may be able to compensate for the lack of cerebellar contributions to rehearsal. If this were true, left ventral prefrontal patients should also exhibit milder verbal working memory deficits. However, this is not the case (Vallar *et al.*, 1997; Martin *et al.*, 1999).

We were surprised that performance was unaffected by the laterality of the damaged hemisphere. Anatomical considerations as well as some imaging results would suggest that lesions of the right cerebellar hemisphere would be more disruptive to verbal working memory than lesions of the left cerebellar hemisphere. Certain verbal tasks such as covert speech and verb generation consistently show correlated left prefrontal/right cerebellar activation (Petersen *et al.*, 1988; Raichle *et al.*, 1994; Ackermann *et al.*, 1998). On the other hand, imaging studies of verbal working memory have not shown consistent laterality effects within the cerebellum; the activation is usually bilateral (Fiez *et al.*, 1996; Becker *et al.*, 1996; Smith *et al.*, 1996; Jonides *et al.*, 1998; Henson *et al.*, 2000; Glabus *et al.*, 2003). This result is consistent with our finding that damage to the inferior cerebellar lobe was related to digit span performance regardless of laterality. Although functional differences likely exist between the cerebellar hemispheres, our results suggest that a simple verbal–nonverbal dichotomy will prove insufficient.

We observed a mild impairment of digit span in patients with unilateral lesions. This result is puzzling given that patients with bilateral cerebellar atrophy have been reported to perform comparable to controls on such tasks (Daum *et al.*, 1993; Bürk *et al.*, 2003; Globas *et al.*, 2003). However, in our own research, atrophy patients performed similar to the unilateral patients (neuropsychological records from seven atrophic patients previously tested demonstrated that all but one of the atrophy patients had a forward verbal span of 5 items or greater). Studies involving patients with atrophy typically include individuals with a range of aetiologies and pathologies. It would be informative to re-examine the effect of cerebellar degeneration on digit span, focusing on the extent of pathology in the inferior lobe.

## Experiment 2

One concern of Experiment 1 was that the patients were required to make fairly rapid, sequential movements when responding. For the verbal task, they had to articulate a series of words; for the spatial task, a sequence of arm movements was required. Failing to do this quickly might limit performance given the rapid degradation of short-term memory. Moreover, a 'cognitive' deficit might be secondary to an increase in attentional resources required to execute these movements (Ravizza and Ivry, 2001). The fact that the patients were unimpaired on the spatial task argues against this form of a 'motor' hypothesis, although there remains a confound between stimulus domain (verbal/spatial) and output domain (oral/manual). Indeed, patients with assumed rehearsal-related deficits have shown verbal span

improvements if allowed to point to items rather than speak (Silveri *et al.*, 1998).

Given this concern, we used a span-matching procedure (Allport, 1984) in Experiment 2 to see if the observed deficits may be related to the motor requirements of the tasks. Instead of responding orally on the digit span task, the patients made manual responses to indicate whether two lists of words were presented in the same order or in a different order. We also included a spatial version of the span-matching procedure.

Moreover, to explore the contribution of the cerebellum to rehearsal, we increased the demand on the rehearsal system while decreasing the amount of information to be maintained. To this end, we presented only four items on each trial, well within the span range observed in Experiment 1, but introduced a delay interval between encoding and recall. We assumed that the participants would have to rehearse during this interval to prevent memory decay. An impairment on this task would be consistent with a hypothesized role in rehearsal (Vallar *et al.*, 1997).

## Methods

### Participants

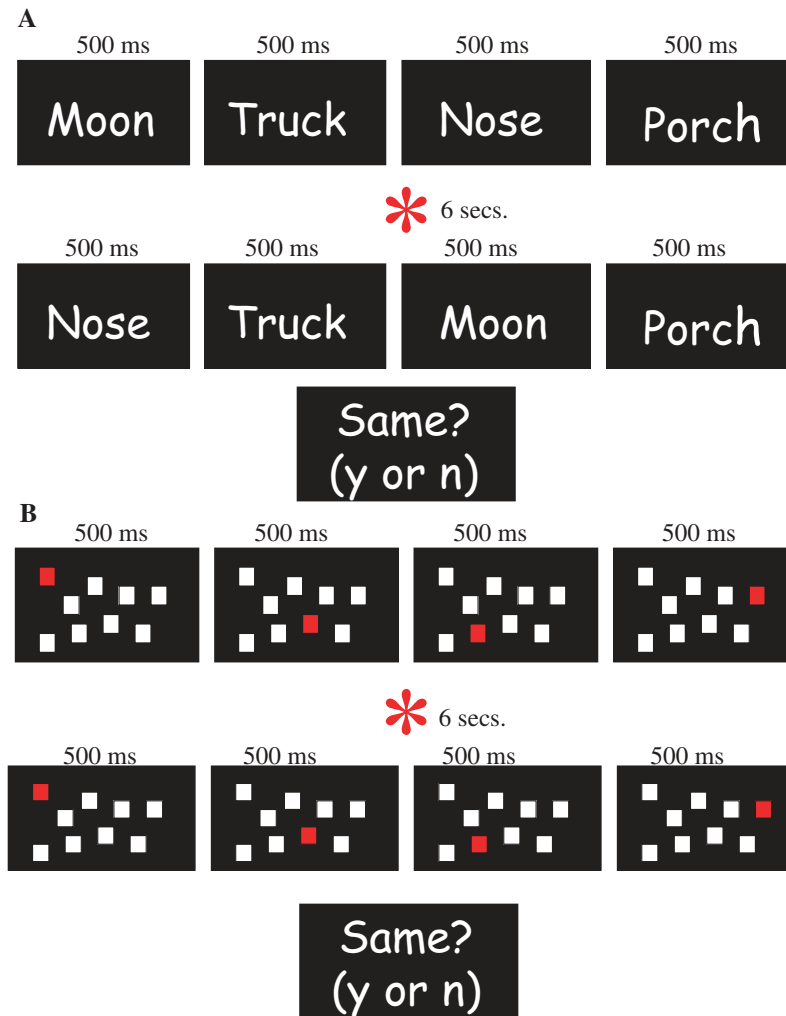
Nine patients (64 years of age; 14.2 years of education) with unilateral damage to the cerebellum and nine healthy control subjects (60 years; 14.5 years) were available for further testing and participated in this experiment for financial compensation (Table 1).

### Stimuli

For the verbal task, four words were randomly selected on each trial from a pool of nine high-frequency words (i.e. bread, coat, inch, moon, nose, porch, ring, snake and truck). For the spatial task, nine white boxes were distributed across the computer screen at all times. Four of these boxes were randomly selected on each trial to comprise the memory set.

### Procedure

For both tasks, participants were presented with the four-item memory set twice and had to judge if the same order was used in the first and second presentation (Fig. 4). For the verbal task, each word was presented for 500 ms at the centre of the screen. For the spatial task, the colour of one of the nine boxes changed from white to red for the 500 ms stimulus duration. There was a 500 ms gap prior to the presentation of the next item, creating a stimulus-onset-asynchrony within each list of 1 s. After the entire list had been presented the first time, there was a 6 s delay interval in which only the fixation cross was displayed. The list was then presented a second time, either in the identical order or a different order. If the order was changed, two randomly chosen items exchanged positions within the list. The order was the same on 50% of the trials and changed on 50% of the trials. Following the presentation of the second list, the question 'Same? (y or n)' was displayed and stayed on the screen until participants responded. Participants responded by pressing a computer key labelled



**Fig. 4** Examples of the (A) verbal and (B) spatial span-matching tasks of Experiment 2. Stimuli were presented sequentially for 500 ms with an inter-stimulus interval of 500 ms. A delay was imposed for 6 s and was indicated by a red asterisk. Following the delay, the same stimuli were presented again in a different or identical order. Participants indicated whether the sequences were the same or different with a button press.

‘yes’ if the list orders were identical and ‘no’ if different. Each participant completed 16 trials in the verbal and spatial tasks each. The order of verbal and spatial blocks was counterbalanced across subjects.

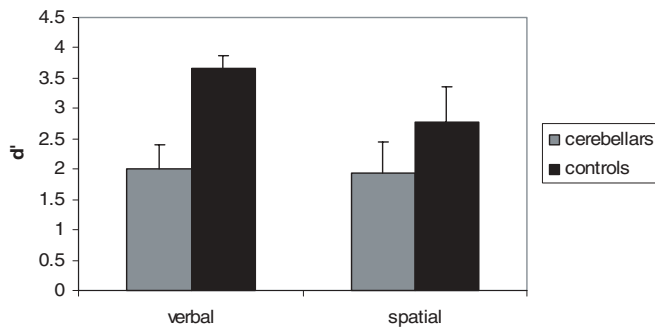
During the test session, the patients were also rated on 10 subtests of the Frenchay Dysarthria Battery (Enderby, 1983) by one of the authors (C.M.), a speech pathologist. These assessed (i) lips alternate, (ii) lips in speech, (iii) jaws in speech, (iv) soft palate maintenance, (v) soft palate in speech, (vi) laryngeal time, (vii) laryngeal pitch, (viii) laryngeal volume, (ix) tongue in speech and (x) intelligibility words/repetition. For each test, a score of 1 indicated no impairment and 5 indicated maximum impairment, yielding a global dysarthria rating ranging from 10 to 50. Upper (manual ataxia and dysmetria) and lower (ataxia and gait disturbance) limb symptoms were also rated at this time on a scale of 1–4 with a greater score indicating more impaired motor functions.

## Results

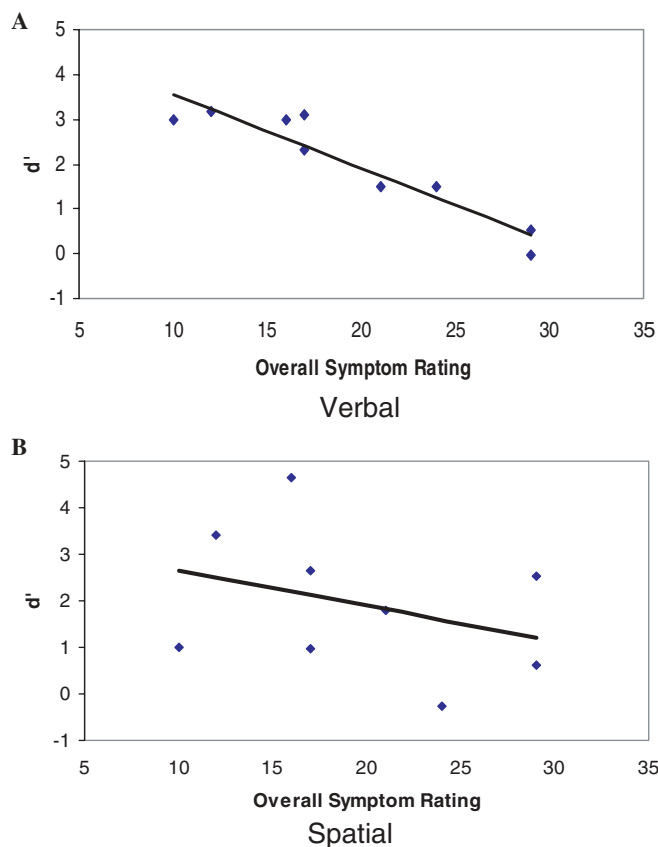
A *d*-score for each task was calculated so that both hit and false alarm rates were reflected in the data. Values of 1 and 0 were assigned Z-scores of 2.33 and  $-2.33$ , respectively. A 2 (task)  $\times$  2 (group) mixed-factor ANOVA produced a significant group effect [ $F(1,16) = 5.66, P < 0.05$ ], but the effect of task and, most importantly, the group by task interaction was not significant ( $P > 0.1$ , Fig. 5). However, when performance on each task was analyzed separately, the patients were impaired on the verbal task [ $t(16) = 3.66, P < 0.005$ ], but not on the spatial task [ $t(16) = 1.11, P > 0.1$ ].

To see if there were differential effects on hit and false alarm rate, the same ANOVA was run using these dependent variables. The results demonstrated a significant group effect for hit rate [ $F(1,16) = 6.38, P < 0.05$ ] and no significant effect of false alarm rate [ $F(1,16) = 3.07, P = 0.099$ ].

To assess how the severity of cerebellar symptoms affected performance on the verbal and spatial span-matching tasks,



**Fig. 5** Performance data ( $d'$ ) of patients and controls on the verbal and span-matching tasks presented in Experiment 2.



**Fig. 6** Correlation of overall cerebellar symptom severity with performance on the (A) verbal and (B) spatial span-matching task in Experiment 2.

we correlated ratings of global dysarthria, upper and lower limb disturbance and the overall severity of symptoms with patients'  $d$ -scores (Fig. 6). Significant negative correlations were observed between all cerebellar symptoms and performance in the verbal condition [dysarthria:  $r(1,7) = -0.827$ ,  $P < 0.01$ ; upper limb:  $r(1,7) = -0.872$ ,  $P < 0.005$ ; lower limb:  $r(1,7) = -0.732$ ,  $P < 0.05$ ; overall:  $r(1,7) = -0.94$ ,  $P < 0.001$ ], but not with performance in the spatial task ( $P_s > 0.1$ )

In contrast to Experiment 1, damage to the inferior cerebellar lobe was not significantly related to performance on the

verbal test [ $r(1,5) = 0.424$ ,  $P = 0.344$ ] and, in fact, was in the opposite direction. Instead verbal span was negatively correlated with lesion size in the anterior [ $r(1,4) = -0.831$ ,  $P < 0.05$ ] and superior lobes [ $r(1,4) = -0.926$ ,  $P < 0.005$ ] and tended to be associated with overall lesion size [ $r(1,4) = -0.738$ ,  $P = 0.094$ ]. Performance in the spatial task was also negatively correlated with lesion size in the anterior ( $r = -0.171$ ) and superior lobes ( $r = -0.397$ ), but correlations were not significant. Moreover, greater lesion sizes in these regions were significantly associated with greater severity of symptoms overall [anterior:  $r(1,4) = 0.837$ ,  $P < 0.05$ ; superior:  $r(1,4) = 0.879$ ,  $P < 0.05$ ] (upper ataxia and dysarthria were close to significance) whereas the opposite trend was shown in relation to lesions of the inferior cerebellum [ $r(1,4) = -0.592$ ,  $P = 0.161$ ].

We also compared performance on the two tasks. For both groups, there was a positive correlation between the two tasks (controls:  $r = 0.51$ ; patients:  $r = 0.48$ ), although neither value reached significance ( $P_s > 0.1$ ).

## Discussion

The results of Experiment 2 demonstrate that the impairment in patients with cerebellar lesions on a verbal working memory task is also observed when overt motor demands are minimized. This finding suggests that the deficits exhibited by the patients in Experiment 1 were not simply due to speech output problems at retrieval. In contrast to Experiment 1, the deficit in Experiment 2 was not restricted to the verbal task as evidenced by the lack of a group by task interaction. However, separate analyses for the verbal task and the spatial task indicated a significant impairment for only the former condition. Thus, there is some indication that the patients' impairments were more marked on the verbal working memory task.

The fact that performance was related to motor symptoms including dysarthria severity suggests that motor speech difficulty can affect the maintenance of verbal information even when responses are manual rather than oral. Moreover, with no delay (Experiment 1), the patients easily recalled four items but performance fell markedly when the time between encoding and retrieval was lengthened in Experiment 2. Taken together, these findings are consistent with the hypothesis that the cerebellum contributes to the rehearsal process. However, they do not rule out a role of the cerebellum in other aspects of the verbal working memory task, such as the initial encoding or recoding of verbal items. For example, information may decay more quickly in patients with cerebellar lesions, because the cerebellum aids in computing a motoric representation of the stimuli (i.e. of the articulatory pattern) that can in turn yield stronger memory traces that are less prone to decay.

In contrast to the first experiment, lesions of the superior and anterior lobe were related to performance in the verbal condition. Although these correlations should be interpreted with caution as our sample size was small, they are consistent with the results of neuroimaging studies supporting superior



lobe involvement in articulatory rehearsal (Desmond *et al.*, 1997; Chen and Desmond, 2005). We will return to this issue in General discussion.

It is somewhat puzzling that the patients were impaired on the spatial span-matching task given their normal performance on what might seem like a more demanding spatial recall task in Experiment 1. One possibility is that the delay period led to greater reliance on verbal codes, even in the spatial task (Coltheart, 1972). An attempt to use verbal strategies would be less effective in improving performance for the patients if their verbal working memory ability is affected.

### Experiment 3

Experiments 1 and 2 have provided some insights into the role of the cerebellum in verbal working memory:

- (i) In an immediate span task, cerebellar patients had impaired verbal but not spatial spans.
- (ii) In a delayed span task, cerebellar patients' verbal and spatial spans were deficient.
- (iii) Verbal spans were not improved when motor demands were reduced, suggesting that impairments do not simply arise as a consequence of difficulties with motor output.
- (iv) Severity of cerebellar symptoms was related to performance on the verbal version of the delayed span-matching task, but not the spatial version.

These results suggest that the cerebellum does contribute to verbal working memory, although its contribution to spatial working memory is less clear. While the group  $\times$  task interaction was not significant in Experiment 2, there was a trend for the patients to be more impaired in the verbal span-matching task compared with its spatial counterpart. We propose that cerebellar patients manifest a problem on spatial working memory tasks when performance can be enhanced by verbal recoding. Two aspects of the patients' verbal working memory performance suggest that the deficits emerge from a disruption in the verbal maintenance system. First, the reductions in verbal span become more notable when a delay was introduced in the serial recall task (Experiment 2). Second, significant correlations were found between cerebellar symptoms and verbal, but not spatial, working memory abilities (Experiment 2).

Note that, although we have provided evidence that the cerebellum is engaged by verbal working memory tasks, we have not directly tested a role for the cerebellum in articulatory rehearsal. In Experiment 3, we tested the rehearsal hypothesis by assessing the impact of experimental manipulations that have been theoretically linked to articulatory rehearsal. Rehearsal is assumed to be sensitive to the length of the items to be remembered: for example, lists of shorter words are better recalled than longer words. Word-length effects have been assessed in previous studies of cerebellar patients with mixed results (Silveri *et al.*, 1998; Justus, 2003; Experiment 1), and thus further attention to this issue is warranted.

Rehearsal is also prone to disruption by articulatory suppression. Typically, the requirement to speak (e.g. 'the, the, the') while trying to remember verbal material is detrimental to recall because articulatory mechanisms are unavailable for rehearsal (Baddeley *et al.*, 1975). If the rehearsal mechanism is disrupted due to cerebellar damage, then suppression should have little effect on recall. Articulatory suppression has been examined in a case study of a patient with a cerebellar lesion. Contrary to this prediction, the patient displayed a normal reduction of recall in the concurrent articulation condition (Silveri *et al.*, 1998).

In Experiment 3, we re-examined these issues in a group study, manipulating both word-length and articulatory suppression. We used an immediate span task with only four items to prevent floor effects. Given that word-length and articulatory suppression influence rehearsal efficiency (Baddeley, 2000), the patients should exhibit a reduced or absent effect of word-length or articulatory suppression on their ability to recall verbal items. These predictions follow from the assumption that rehearsal processes are impacted by cerebellar pathology and thus, recall by these patients is less dependent on covert rehearsal. (Similar logic has been used to account for the absence of word-length and articulatory suppression effects in subjects with damage to inferior prefrontal cortex.)

Moreover, we included a manipulation of lexical status (i.e. word versus non-word) as a control condition. Lexical status may affect rehearsal mechanisms if articulatory commands are more difficult to derive from non-words and, in fact, lexicality effects tend to be diminished under articulatory suppression (Besner and Davelaar, 1982). However, lexical status may have more of an effect on Broca's area as other studies have suggested preferential activity of this region for effortful rather than automatic speech production (Herbster *et al.*, 1997; Fiez *et al.*, 1999). Indeed, cerebellar activity was not affected by this variable in an imaging study (Chein and Fiez, 2001). Instead of displaying qualitative deficits in all aspects of serial recall performance (i.e. articulatory suppression, word-length and lexicality effects), we predicted that both controls and patients would show equivalent effects of lexical status, but not word-length or articulatory suppression.

Our task also allowed an examination of serial position effects. Prior neuropsychological work has reported intact recency effects for patients with assumed rehearsal deficits, such as those with ventral prefrontal damage (Vallar *et al.*, 1997) or cerebellar damage (Silveri *et al.*, 1998) whereas damage to a putative store is associated with a reduced recency effect. Thus, we predicted that cerebellar patients would show the same advantage for recalling items in the last position, as controls do.

## Methods

### Participants

Sixteen participants, eight patients (average age = 63; average education = 13.75) and eight controls (average age = 65;

average education = 16), received financial compensation for their time and effort.

### Stimuli

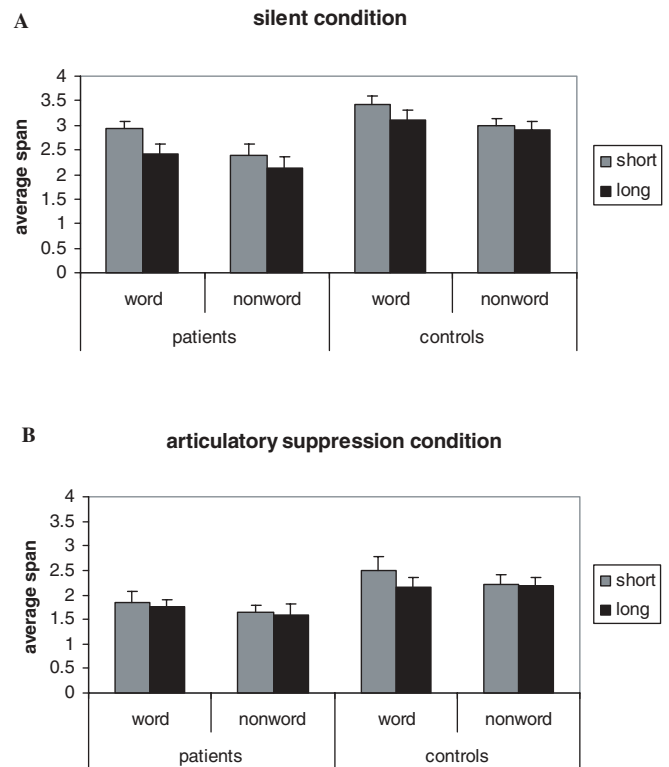
Four ten-item word lists, two composed of two-syllable words and two composed of four-syllable words, were created by selecting items matched for frequency (see Appendix I). Four non-word lists were created by making lists of equivalent frequency real words and then changing the identity of two letters so that the item was still pronounceable. For each list, three pairs started with the same letter so that participants would not be able to abbreviate items by remembering only the initial grapheme. Across participants, the lists were counterbalanced such that one member of the list pair for each of the four conditions was assigned to the silent condition and the other was assigned to the suppression condition. Thus, there were eight conditions in total, formed by the factorial combination of lexical status (word/non-word), length (2/4 syllable) and rehearsal demands (silent/suppression).

### Procedure

The eight conditions were tested in separate blocks, with the order of the blocks randomized across participants. On each trial, four items from the designated condition were chosen at random and presented serially on a computer monitor for 1 s with a 500 ms inter-stimulus interval. List length was fixed at four items, and longer spans were not assessed. The participant silently read the four items as they were presented until a red asterisk appeared in the centre of the computer screen. Upon appearance of the asterisk, the participant referred to a corresponding list of ten words or non-words presented in hard-copy format. He/she was instructed to silently select and record the order in which the four stimuli appeared by writing a one, two, three and four next to their choices (note that, although they had to recall serial order correctly, the sequence in which they responded did not necessarily reflect the input sequence). Participants were told that guessing was unacceptable and would adversely affect their overall score. Although they were permitted to advance at their own pace, they were discouraged from deliberating extensively.

In the articulatory suppression condition, participants continually said the word 'the' throughout the length of the entire trial. Participants articulated no less than one and no more than three words per second, and verbalization was discontinued between trials. On occasion the examiner needed to prompt the participants verbally, reminding them that continuous articulation was essential.

Each block was composed of 20 trials. A short break was provided between blocks, although this was extended by 5–10 min if the participant requested an extra break. The duration of the experiment was ~2 h for both the patients and the control subjects.

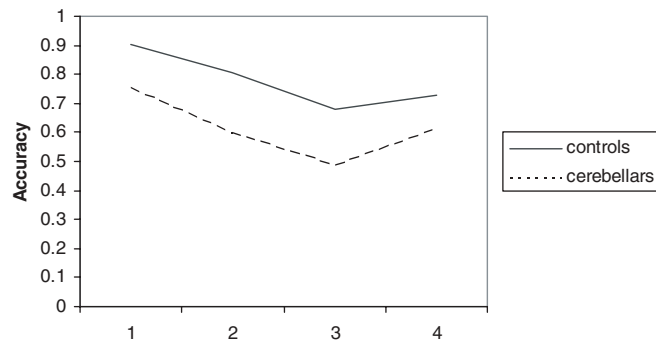


**Fig. 7** Span length of cerebellar patients and control participants when required to immediately recall both long and short words and non-words in (A) silence and (B) under articulatory suppression in Experiment 3.

### Results

The dependent variable in this task was the average number of items recalled in their correct order in each trial across conditions. All of the main, within-subject effects were significant (Fig. 7): Participants recalled more short items than long items [ $F(1,14) = 8.73, P = 0.01$ ], more words than non-words [ $F(1,14) = 12.77, P < 0.005$ ] and fewer items under articulatory suppression [ $F(1,14) = 161.32, P < 0.001$ ]. In addition, the between-subject effect of group was significant [ $F(1,14) = 7.39, P < 0.05$ ], reflecting the fact that the cerebellar patients recalled fewer lists correctly than the control participants. The only interaction to reach significance was that between lexical status and suppression [ $F(1,14) = 9.37, P = 0.01$ ]. Most importantly, the group factor did not interact with any of the other variables; the patients showed length, lexicality and suppression effects comparable to controls ( $P_s > 0.1$ ).

We also calculated accuracy for items in the silent conditions based on their position in the list to determine whether cerebellar patients would show primacy and recency effects. A 4 (position)  $\times$  2 (group) mixed-factor ANOVA produced significant main effects of position [ $F(3,42) = 14.69, P < 0.001$ ] and group [ $F(3,42) = 9.03, P < 0.01$ ] (Fig. 8). *Post hoc t*-tests indicated that the main effect of position was primarily due to the superior recall of the first item compared to items in the second [ $t(1,14) = 7.04, P < 0.001$ ], third [ $t(1,14) = 7.07,$



**Fig. 8** Serial position curves of patients and controls in Experiment 3.

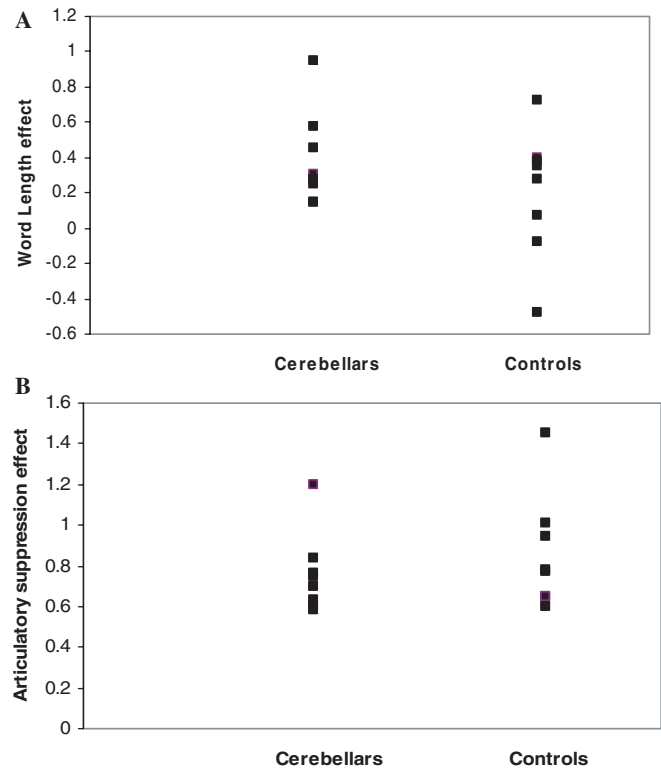
$P < 0.001$ ] and fourth positions [ $t(1,14) = 3.68$ ,  $P < 0.005$ ]. Primacy (accuracy of item in 1st position – 2nd) and recency (4th – 3rd) effects did not differ between groups ( $P$ s  $> 0.1$ ).

Overall symptom severity (dysarthria rating and lower limb ataxia scores approached significance) was negatively correlated to accuracy only in the first position [ $r(1,6) = -0.74$ ,  $P < 0.05$ ], but positively correlated with the size of the recency effect [ $r(1,6) = 0.81$ ,  $P < 0.05$ ]. None of the symptom measures were significantly associated with word-length and lexicality status (restricted to the silent conditions, only), or with articulatory suppression effects. Lesion size in the anterior and superior cerebellum were not significantly related to any measure of performance; however, a tendency for smaller recency effects was associated with larger lesions to the inferior cerebellum [ $r(1,5) = -0.75$ ,  $P = 0.053$ ].

## Discussion

Consistent with Experiments 1 and 2, verbal spans were reduced for cerebellar patients. However, the patients with cerebellar lesions exhibited normal articulatory suppression and word-length effects without any tendency to be reduced in magnitude. Moreover, the results suggest that the locus of interference between rehearsal mechanisms and articulatory suppression does not reside in the cerebellum; if this were the case, damage to the cerebellum should selectively impair verbal working memory and reduce the effect of concurrent articulation (rehearsal suppression). While the cerebellum is activated in imaging studies of covert and overt speech, it does not appear to support the type of speech production processes affected by articulatory suppression.

While caution is warranted given that this is a null result, we do note that the means for the patients are equal or even larger than the controls on these two measures, and there is considerable overlap in the distribution of the magnitude of the various effects (Fig. 9). In sum, the results indicate that damage to the cerebellum did not alter any of the markers of rehearsal in this experiment. These null results also stand in contrast to those reported in studies of patients with prefrontal lesions (Goerlich *et al.*, 1995; Vallar *et al.*, 1997)



**Fig. 9** The magnitude of (A) length and (B) articulatory suppression effects for patients and controls in Experiment 3.

showing attenuated word-length and articulatory suppression effects.

Our predictions concerning cerebellar damage on the size of the word-length and articulatory suppression effects assume that patients are impaired in their ability to engage in covert rehearsal. A strong form of such an impairment would be that the patients simply fail to rehearse. Alternatively, an impairment might result in the slowing of covert rehearsal. In this case, one might predict the word-length effect to be normal for patients if slowing affects short and long words equally. However, our predictions regarding articulatory suppression remain the same, regardless of whether the impairment results in the absence of rehearsal or the slowing of rehearsal. Articulatory suppression should prevent rehearsal in both control and patient groups. When released from articulatory suppression, rehearsal speed would be slower (or non-existent) for the patients, resulting in lower spans compared to controls. Thus, we would still predict a smaller difference between silent and articulatory suppression conditions for the patients compared to the controls.

Perhaps the simple articulatory task (repeat 'the') can be accomplished through other neural regions such as the SMA or insula. Indeed, Gruber (2001) reported that the SMA and the insula were engaged during silent rehearsal and articulatory suppression (Gruber, 2001; Gruber and von Cramon, 2003). In contrast, the cerebellum was actively disengaged during concurrent articulation (Gruber, 2001; but see Gruber and von Cramon, 2003). We had speculated that

the cerebellum was the most probable locus of articulatory suppression effects (Chein *et al.*, 2003). The current results challenge this idea (see also Silveri *et al.*, 1998; Gruber, 2001; Justus *et al.*, 2005).

The present results stand in contrast with a previous study of four patients with cerebellar lesions that found a trend for a reduced word-length effect relative to controls, despite significant effects in both groups (Justus, 2003; Experiment 1). One critical difference between the studies is the method of output. In our study, participants wrote numbers to indicate serial order whereas words were spoken aloud in the Justus (2003) experiment. A dependency on response format has important implications for understanding the role of the cerebellum in verbal working memory. Such a dissociation would be hard to reconcile with a rehearsal-based account of the patients' impairment. Instead, the explanation for reduced word-length effects would derive from the fact that overt articulation takes more time for cerebellar patients so that even lists of short words become subject to decay during retrieval. Consistent with this view, imaging studies reporting that cerebellar activity was influenced by word-length also implemented oral recall (Grasby *et al.*, 1994; Chein and Fiez, 2001). On the other hand, in Experiment 2 of this study we found that verbal working memory deficits persisted when the motor output demands were reduced, and length effects have also been observed during the encoding interval using fMRI (Chein and Fiez, 2001).

Although cerebellar patients did not exhibit deficits in the qualitative variables that we predicted (i.e. articulatory suppression and word-length effects), we correctly predicted variables that did not affect their performance. For example, cerebellar patients exhibited normal lexicality and recency effects. Thus, the cerebellum is not sensitive to any potential differences in the maintenance of words versus non-words, and is unimportant for recall that occurs immediately after encoding.

## General discussion

Our goal was to determine the extent of cerebellar involvement in verbal working memory tasks. We found that patients with cerebellar damage were moderately but consistently impaired on an immediate verbal recall task (Experiments 1 and 3) and that their deficits increased when a delay was introduced before recall (Experiment 2). These results correspond nicely to imaging studies reporting cerebellar involvement in verbal working memory tasks, and their subtle impairment on the standardized test of verbal working memory is consistent with the clinical observation that cerebellar damage is not typically associated with short-term memory deficits. Furthermore, we have provided evidence that the type of short-term memory deficit is more likely to be in the verbal rather than visuospatial domain. Verbal working memory impairments are still apparent when oral output is not required of cerebellar patients (Experiments 2 and 3).

Combined with previous findings in the patient and imaging literature, we can note some dissociations between

the roles of the cerebellum and Broca's area in verbal working memory tasks. The cerebellum is unaffected by manipulations of lexical status whereas Broca's area has been implicated to a greater degree in memory for non-words than real words (Chein and Fiez, 2001). These results suggest that Broca's area is more important for words that require more effortful articulatory/phonological processing whereas the cerebellum is similarly engaged for all types of verbal material. A second dissociation is observed with respect to the effect of articulatory suppression; patients with damage to prefrontal cortex show an attenuated suppression effect (Goerlich *et al.*, 1995; Vallar *et al.*, 1997), whereas the current results indicate that this effect is normal in patients with cerebellar damage. Given that SMA and insula activity are observed during articulatory suppression and silent rehearsal (Gruber, 2001; Gruber and von Cramon, 2003), the evidence suggests that the cerebellar contribution to verbal working memory is independent of processes affected by articulatory suppression, i.e. covert rehearsal. This view is further supported by event-related imaging evidence showing that cerebellar activation is pronounced during encoding for verbal working memory tasks with a long delay, but this activation does not persist through the delay interval (Chein and Fiez, 2001).

Note that the effect of delay on cerebellar performance in Experiment 2 does not entail that the cerebellum should be active during a delay period. If the cerebellum were involved in rehearsal, then one would expect activity of this region during a delay interval. However, other aspects of successful recall such as the initial strength of the memory trace, may be affected by a delay in the same way as the ability to rehearse. Thus, although the effect of a delay interval on cerebellar performance is consistent with a rehearsal explanation, the lack of its activity over a delay period (Chein and Fiez, 2001) and the absence of word-length and articulatory suppression effects in the patient data argues against this functional theory.

Assuming the cerebellum does not contribute to rehearsal, we must then consider alternative working memory functions that might recruit this subcortical structure. One possibility is phonological encoding. Patients with cerebellar lesions exhibit a reduced phonological similarity effect (Justus *et al.*, 2005); that is, the patients did not perform significantly worse for lists of words that contained the same vowel, as controls do. It is typically argued that the phonological similarity effect reflects processing within a short-term phonological store rather than rehearsal. While we are tempted to conclude that the cerebellum is not part of the rehearsal network, this dissociation relies on the assumption that word-length and phonological similarity effects are markers for rehearsal or storage, respectively. As noted previously, it has also been argued that the word-length effect reflects the time taken to produce words in the output stage rather than the demands on rehearsal processes during a delay interval (Avons *et al.*, 1994). Furthermore, phonological similarity effects have been argued to reflect rehearsal difficulty rather than interference in a phonological store (Jones *et al.*, 2004).

Our finding that performance on the verbal working memory tasks depended on the site of the lesion also argues that the cerebellum may contribute to both storage and rehearsal. Indeed, the current results can be considered within the model proposed by Desmond and colleagues (Desmond, *et al.*, 1997; Chen and Desmond, 2005) concerning functional compartmentalization within the cerebellum in terms of working memory. In this model, projections between the frontal cortex and superior cerebellum are hypothesized to support articulation, either overt or covert, projections between the temporal cortex and inferior cerebellum are associated with phonological storage. The inclusion of a delay period in Experiment 2 would increase the demands on rehearsal. Consistent with the Desmond model, we found that patients with superior and anterior cerebellar lesions were more affected than those with lesions of the inferior lobe. Moreover, lesions of the superior and anterior lobes were related to dysarthria. In contrast, the size of the recency effect, traditionally associated with storage rather than rehearsal, was associated with the extent of damage in the inferior lobe. While this pattern is intriguing, caution is warranted given the small sample size and the considerable variability observed between the site of damage and our assays of rehearsal and storage. For example, neither word-length nor articulatory suppression was reliably associated with lesions to the superior lobe, even though these variables are assumed to influence articulatory rehearsal.

Regardless of the precise nature of its contribution, however, it is clear that the cerebellum supports verbal working memory. Furthermore, an account emphasizing a role in covert rehearsal does not appear to be sufficient for explaining the role of the cerebellum in verbal working memory. The robust association between the cerebellum and articulation, either overt or covert (Price and Friston, 1997; Ackermann *et al.*, 1998; Mechelli *et al.*, 2003) was a primary motivation for the hypothesized role of the cerebellum in rehearsal. However, articulatory processes may influence verbal working memory in other ways. For example, cerebellar activity is observed at encoding as well as retrieval (Chein and Fiez, 2001) suggesting that the cerebellum may be important in the formation and retrieval of memory traces. The motor theory of speech emphasizes the intimate relationship between perception and action; specifically, perception relies on reference to articulatory representations (Liberman and Mattingly, 1985). Integrating acoustic/phonetic representations with articulatory representations may result in strengthened memory traces or may provide a mechanism for correcting degraded sensory information. Thus, instead of being important for rehearsal, the motor representations or processing provided by the cerebellum may be more important for creating an integrated memory trace that is less prone to decay or error.

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## Appendix I

|        | Short words  | Long words  | Short non-words  | Long non-words   |
|--------|--|---|--|--|
| List 1 | Baggage<br>Charcoal<br>Complaint<br>Lilac<br>Liver<br>Nutmeg<br>Pension<br>Princess<br>Razor<br>Sailor | Alligator<br>Auditorium<br>Cafeteria<br>Convertible<br>Decorator<br>Diagnosis<br>Fertilizer<br>Isolation<br>Monopoly<br>Radiator    | Drable<br>Dultom<br>Ebley<br>Megy<br>Menture<br>Piskel<br>Refance<br>Relper<br>Simal<br>Tapad  | Davitalist<br>Dondernation<br>Effility<br>Emmudition<br>Fedeloper<br>Fenicacy<br>Gelinity<br>Netromolis<br>Omilation<br>Seletary |
| List 2 | Bacon<br>Banker<br>Folly<br>Orange<br>Oven<br>Resin<br>Sewer<br>Shovel<br>Temper<br>Wallet             | Agriculture<br>Antiseptic<br>Biology<br>Elevator<br>Peninsula<br>Photographer<br>Seminary<br>Supervisor<br>Thermometer<br>Vegetable | Calter<br>Crangy<br>Edust<br>Glantel<br>Sefail<br>Tholen<br>Togar<br>Undush<br>Urdit<br>Valiff | Cenepactor<br>Ecalemy<br>Embivalent<br>Ipilemic<br>Ranotama<br>Romarity<br>Tepalation<br>Tolibitor<br>Velperment<br>Wolustary    |