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Fast and efficient visuotemporal attention requires the cerebellum

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Abstract

The presence, and nature, of any role of the cerebellum in complex, non-motor behaviors is only beginning to be uncovered. We investigated the non-spatial temporal dynamics of attention in 11 patients with chronic focal lesions to the cerebellum using a rapid serial visual presentation task known as the attentional blink paradigm. In this task two targets are embedded in a letter stream presented at central fixation for identification and the delay between the targets is manipulated. Patients demonstrated an unequivocal disturbance in rapid visual attention as indicated by an increased magnitude of the attentional blink (i.e., more impaired at detecting target 2 when presented in close contiguity to target 1) compared to 13 healthy controls. The attentional blink effect was not significantly protracted in our patients, suggesting a time-limited deficit in resource allocation during temporally demanding stimulus processing conditions. Recovery rate from the attentional blink was the same for our patients and controls implying intact selective attention following cerebellar damage. Because of the experimental design, the results of the present study could not be accounted for by motor dysfunction or saccadic dysmetria. These data provide evidence implicating the cerebellum as a critical node in the neuroanatomical network underlying visuotemporal attention and provide further evidence for the role of the cerebellum in non-motor behaviors.

Keywords: Cerebellum; Cognition; Visual attention; Rapid serial visual presentation; Attentional blink

1. Introduction

Cognitive impairment as a consequence of cerebellar damage was first implied in 1831 (Combettes, 1831), but only in the last 20 years has evidence converged from lesion studies and functional neuroimaging research to start to specify roles for the cerebellum in cognition, particularly in attention (Allen, Buxton, Wong, & Courchesne, 1997; Berger et al., 2005; Courchesne et al., 1994; Fiez, Petersen, Cheney, & Raichle, 1992; Gottwald, Wilde, Mihajlovic, & Mehdorn, 2004; Leiner, Leiner, & Dow, 1986; Schmahmann, 2004; Townsend et al., 1999). Lesion studies define whether the cerebellum plays a necessary role in these processes. However, for the clinical/lesion studies there has been no dimension of the role of the cerebellum in attention function that has escaped controversy: working definitions of attention, the demands of attention tasks, the patient populations appropriate for study, the extent to which apparently cognitive deficits can be explained as motor phenomena (Bischoff-Grethe, Ivry, & Grafton, 2002; Ravizza & Ivry, 2001) and even agreement about whether results are meaningful or not (Glickstein, 2006; Schmahmann & Caplan, 2006). With such broad disagreements about the methodology and the subjects, it is not surprising that there are failures to replicate findings (Daum et al., 1993; Helmuth, Ivry, & Shimizu, 1997; Schoch et al., 2004), leaving many issues unresolved.

The attentional blink (AB) paradigm has been used extensively in cognitive science to investigate the temporal dynamics of attention (Raymond, Shapiro, & Arnell, 1992) and may offer a clearer window about the role of the cerebellum in attention. The AB paradigm is a rapid serial visual presentation (RSVP) task requiring the identification of two targets in a stream of letters that appear in one location. The "blink" provides an index of our insensitivity at detecting the second of two targets when they are presented in rapid succession. This effect is thought to reflect a capacity-limited process. One explanation of the AB effect is that attention is captured by the first stimulus followed

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by a brief period (typically for ∼500 ms) before attention can be re-allocated to process the second target (Nieuwenstein & Potter, 2006). Importantly for research on cognitive functions associated with the cerebellum, unlike many other attention paradigms, the AB paradigm places no demands on control of eye movements or on any other motor control.

There are two features of AB performance that address different aspects of attentional processing. *Magnitude* is the accuracy at detecting the second stimuli during the period of the "blink". *Duration* is how long the "blink" lasts. If damage to the cerebellum affects magnitude then we should observe a greater reduction in the accurate detection of the second target when presented in close contiguity to the first target compared to controls with equivalence between patients and controls at the longer temporal intervals. This pattern of impairment suggests that damage to the cerebellum may compromise the efficient use of attentional resources particularly during temporally demanding stimulus processing conditions. If damage to the cerebellum affects duration, the AB effect will be prolonged beyond the usual 500 ms. This pattern of impairment suggests a deficit in the overall mechanisms of selective attention. Finally, if cerebellar damage generally diminishes sustained attention, both patterns might be observed but detection of the "second" target when there is no first target (see Section 2 for single-task condition) should be erratic compared to controls as the test then becomes an unpredictable target detection/vigilance task. None of these predictions are influenced in the slightest way by motor control or eye movement function.

Lesion studies have demonstrated an effect on the AB of lesions in frontal (Husain, Shapiro, Martin, & Kennard, 1997; Richer & Lepage, 1996) and parietal (Shapiro, Hillstrom, & Husain, 2002) regions, but there have been no studies of the AB in patients with isolated cerebellar lesions. Neuroimaging studies show consistent activation of prefrontal and parietal regions during AB tasks but have not always examined the cerebellum (Marois, Chun, & Gore, 2000; Marois, Yi, & Chun, 2004). One study has demonstrated bilateral activation associated with the AB (Marcantoni, Lepage, Beaudoin, Bourgouin, & Richer, 2003). A recent review of lesion and functional imaging studies of the AB (Hommel et al., 2006) made no mention of the cerebellum despite the extensive reciprocal connectivity between the cerebellum and putatively critical cortical areas (Allen et al., 2005; Middleton & Strick, 2001; Schmahmann, 2001).

Thus, the AB task offers information about attention capacity in a dual-task, single modality test that appears to explore, in a fairly novel manner, several possible contributions of the cerebellum.

2. Methods

2.1. Subjects

We tested 11 patients (age range, 24–74, mean, 48.3) with focal cerebellar damage at least 90 days post-injury (range, 90–980; mean, 451.9). Etiology of injury was restricted to vascular and benign tumour excision. Structural MRI (all patients received 1.5 T MRI scans), examined by an experienced neurologist (MPA), confirmed no evidence of extracerebellar damage. For a clinical summary of our patients see Table 1. Lesions were reconstructed using a cerebellar template developed by Ivry and colleagues from the Cognition and Action Laboratory at the University of California at Berkeley and is shown in Fig. 1. None of the patients had a previous stroke, traumatic brain injury, head radiation or chemotherapy, any other neurological disease, or any axis-I DSM diagnosis. Thirteen healthy adults without neurological or psychiatric disorder (age range, 23–73; mean, 53.7) matched for age ($t = 0.74$, n.s.) and education level ($t = 1.9$, n.s.) to the patients were also tested. As seen in Table 2, control participants and patients did not differ on standardized tests of working memory (Digit and Spatial Span), visuospatial abilities (Judgment of Line Orientation), frontal lobe function (Stroop, Trails A and B, and Verbal Fluency) and verbal comprehension (Token Test). All participants had normal or corrected to normal vision and colour perception. All participants provided written informed consent prior to commencing the experiment according to the Declaration of Helsinki. Ethical approval for this research was obtained by the Toronto Academic Health Sciences Council.

Table 1
Patient information

Patient	Gender	Age	Education	Etiology	Time post-lesion (days)
1	M	74	14	Vascular	260
2	M	70	18	Benign tumour	377
3	F	65	7	Vascular	328
4	M	40	21	Vascular	90
5	F	53	12	Benign tumour	472
6	M	30	12	Vascular	980
7	M	41	12	Benign tumour	443
8	F	42	12	Benign tumour	787
9	M	59	8	Benign tumour	674
10	M	33	14	Benign tumour	442
11	F	24	15	Benign tumour	118

2.2. Experimental stimuli and task

The experiment was conducted in a dark room. The stimuli were uppercase letters presented in white on a black background subtending a visual angle of 1.91° for height and 1.72° for width from a viewing distance of 60 cm. The letters were presented in RSVP (133 ms/letter), at the central fixation point of a computer screen. Each letter was exposed for 100 ms with no blank interstimulus interval (see Fig. 2).

Each trial consisted of a string of letters in which were embedded two defined targets. The non-targets were selected at random, without replacement, from the letters of the alphabet, excluding the target letters H, S, X, and Y and always presented in white. Participants were instructed to identify two targets (T1 and T2) that appeared in a fixed order. T2 was never presented prior to T1 in the letter stream. T1 was either a red H or S; T2 was either a white X or Y. There were 6–9 letters (selected randomly) presented prior to T1 and 9–12 letters (selected randomly) following T1. T2 could occur in any of the six positions (1, 2, 3, 4, 8, and 12) following the presentation of T1 with equal probability. There were always between 1 and 4 letters following the presentation of T2.

2.3. Procedure

A cross was presented at the center of the screen to act as a fixation point for the current trial. Each trial was initiated by the experimenter by pressing the spacebar on the computer keyboard, which removed the fixation cross and initiated the onset of the RSVP stream.

Task 1 required subjects to discriminate between the letter H and S when a red letter was presented. T1 was presented for two-thirds of the trials and not presented for one-third of the trials. These two different conditions were randomly intermixed within each block of trials. Thus, performance on the T1-present trials was of prime importance for producing the AB effect and the T1-absent trials provided a control condition where no AB effect should be present due to the absence of a stimulus to trigger task 1. At the end of the RSVP sequence, the program requested a response for task 1 and the subjects

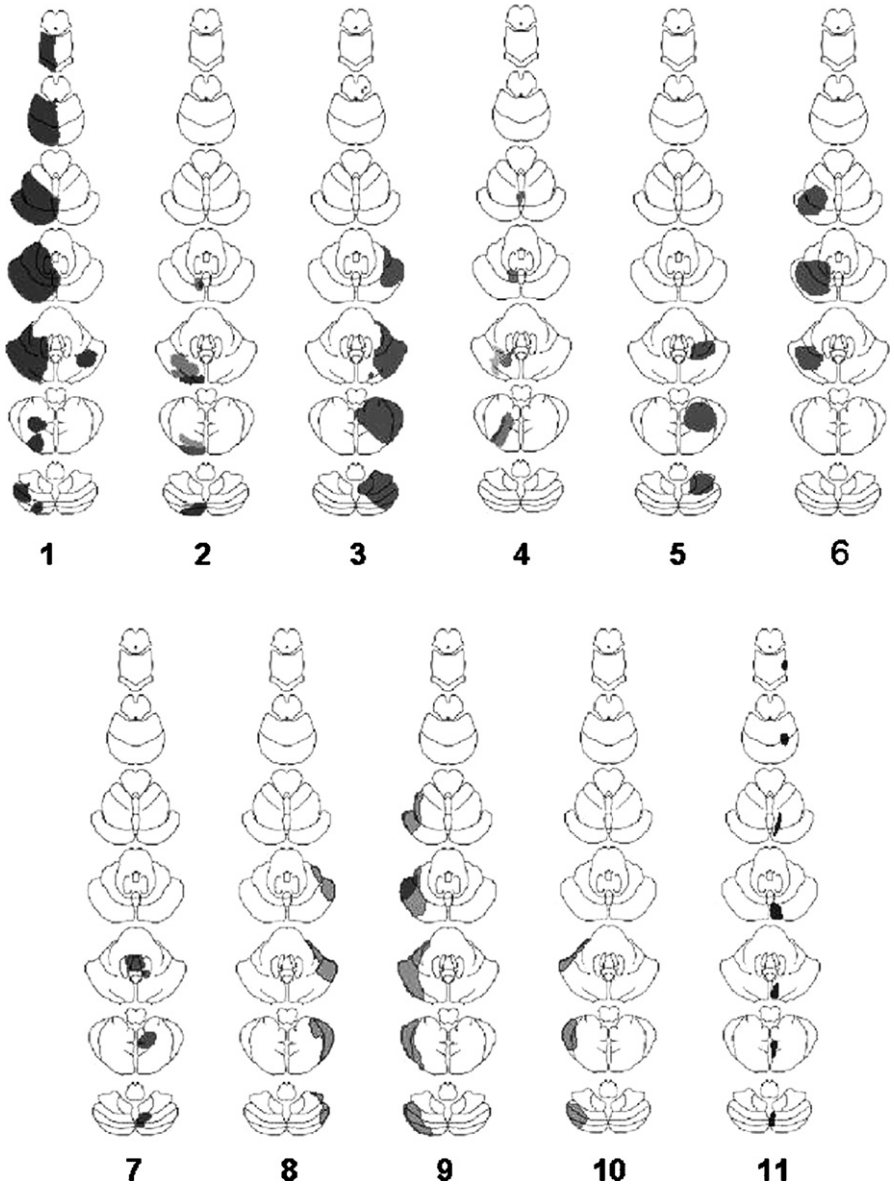

Fig. 1. Schematic reconstruction of the cerebellar lesions (in dark gray; light gray area represents secondary atrophy). In each column sections are arranged from superior (top) to inferior (bottom) for each patient. Cerebellar templates provided by Ivry and colleagues from the Cognition and Action Laboratory at the University of California, Berkeley.

reported the letter for the trial and the experimenter recorded this information using the computer keyboard. The "H" key was pressed for the H or the "S" key was pressed for the S. The "N" key was pressed during conditions where no red letter was presented.

Task 2 required subjects to discriminate between the letter X and Y presented in white and following Task 1. Subjects were made aware that the RSVP letter stream always contained either an X or a Y. T1-present and T1-absent trials were designed in the same way with the only exception being that in the T1-absent trials, T1 was replaced with a distractor letter selected at random and presented in white. At the end of the RSVP sequence and following the prompt for the response to Task 1, the program requested a response for Task 2 and the subjects reported the letter for the trial and the experimenter recorded this information using the computer keyboard. The "X" key was pressed for the X or the "Y" key was pressed for the Y.

The main dependent variable of interest was the accuracy at detecting each target. A practice block of 24 trials at a slower rate (250 ms/letter) allowed participants to become familiar with the task prior to the experimental blocks. Each participant completed 432 experimental trials (6 blocks of 72 trials).

2.4. Criterion measures and data analyses

T2 detection accuracy following the correct identification of T1 and T2 detection accuracy on T1-absent trials were submitted to analyses of variance (ANOVAs) in which Lag (six positions) was a within-subject factor and Group (cerebellar versus controls) was a between-subject variable.

3. Results

3.1. Accuracy

3.1.1. Target 1 (T1)

Overall, identification of T1 for the cerebellar group was 96.3 and 97.5% for controls, indicating that both groups were actively engaged in identifying T1. A 2 Group × 6 Lag ANOVA of mean

Table 2
Subject characteristics

Demographics	Controls	Patients	
N	13	11	
Gender	7 M, 6 F	7 M, 4 F	
Age	53.7 (5.2)	48.3 (5.11)	
Education	15.36 (0.72)	13.18 (1.20)	
Neuropsychological tests	**Controls**	**Patients**	***P*-value (*t*-test)**
Digit span			
Forward score	10.64 (0.41)	9.64 (0.79)	0.274
Backward score	7.36 (0.70)	6.91 (0.77)	0.659
Total score	18.00 (0.77)	16.55 (1.44)	0.384
Spatial span (WMS III subtest)[a]			
Forward score	6.40 (0.54)	8.00 (0.63)	0.070
Backward score	6.60 (0.54)	7.73 (0.68)	0.209
Total score	13.00 (0.91)	15.73 (1.24)	0.093
Trails A and B[a]			
A time	37.00 (3.81)	34.18 (3.78)	0.605
B time	83.18 (11.22)	80.91 (14.61)	0.903
B errors	0.55 (0.37)	0.45(0.31)	0.852
B − A/A	1.29 (0.25)	1.28 (0.23)	0.973
Benton judgment of line orientation[a]			
Form H (out of 15)	12.50 (0.50)	11.91 (0.80)	0.541
Stroop[a] (Comalli version)			
Word reading total time (s)	47.60 (1.71)	51.64 (3.05)	0.266
Colour naming total time (s)	70.10 (2.54)	70.00 (6.26)	0.988
Incongruent total time (s)	125.70 (5.37)	122.09 (10.37)	0.762
Errors	1.20 (0.33)	2.64 (1.01)	0.201
Verbal fluency (FAS—first minute S)			
Total correct	44.73 (4.81)	38.82 (3.26)	0.323
Total perseverations	1.36 (0.58)	1.00 (0.36)	0.599
Error	1.91 (0.53)	2.27 (0.56)	0.642
Token test[a]			
Raw score	42.30 (0.63)	41.55 (0.98)	0.549

Standard errors of the mean are shown in parentheses.
[a] $N = 10$ for control group. Some controls were not available for all neuropsychological testing.

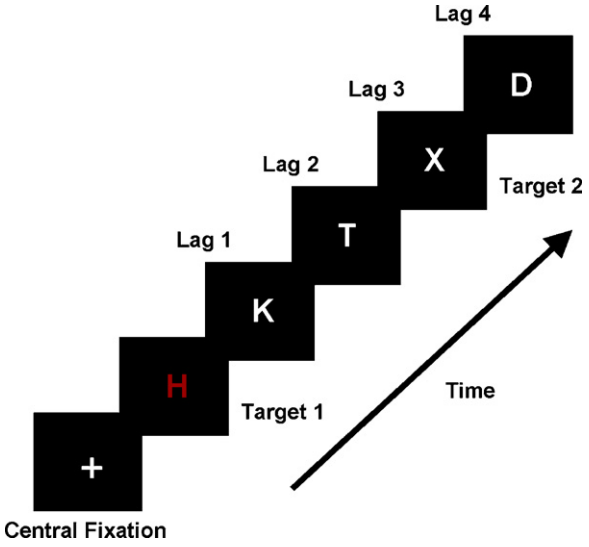

Fig. 2. The attentional blink paradigm.

% accuracy to detect T1 revealed no significant differences (Ps > 0.17).

3.1.2. Target 2 (T2): single-task condition (T1-absent)

A 2 Group (cerebellar, control) × 6 Lag (1, 2, 3, 4, 8, and 12) mixed model ANOVA of mean % accuracy to detect T2 in the absence of T1 was performed. The ANOVA produced no significant effects (Ps > 0.20). Overall, identification of T2 in the absence of T1 for the cerebellar group was 76.4 and 84.1% for controls.

3.1.3. Target 2 (T2): dual-task condition (T1-present)

To address our main hypotheses we performed a 2 Group (cerebellar, control) × 6 Lag (1, 2, 3, 4, 8, and 12) mixed model ANOVA of mean % accuracy to detect T2 given correct identification of T1. The ANOVA produced a main effect of Lag [$F_{5,18} = 24.8$, $P < 0.0001$] and a main effect of Group [$F_{1,22} = 6.0$, $P = 0.022$]. A significant Lag × Group interaction was also obtained [$F_{5,18} = 2.3$, $P = 0.046$]. The significant main effect of Lag indicates the typical AB effect as shown by reduced

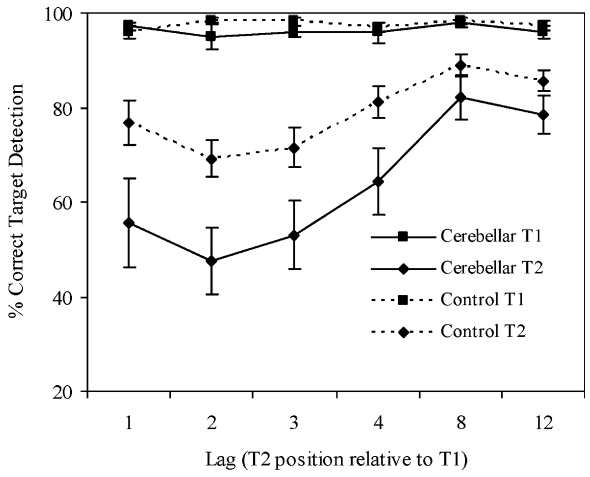

Fig. 3. Performance (mean % accuracy ± S.E.M.) in detecting T1 and T2 for both cerebellar and control participants at six different temporal lags (T2 position relative to T1).

accuracy in detecting T2 when presented in close temporal proximity to T1 and the increased accuracy as the delay between T1 and T2 increased. The main effect of group arose due to an increased magnitude of the AB (reduced accuracy to detect T2) in patients with cerebellar lesions compared to healthy controls (see Fig. 3). The significant interaction arose due to our cerebellar patient's greater reduction in the accurate detection of T2 at the shorter lags, compared to controls, with an improvement in the detection of T2 when there was a longer temporal gap to process the first target (e.g., lags 8 and 12). Planned group comparisons revealed a significant difference between cerebellar and control participants at lags 1–4 ($Ps < 0.05$) and no significant differences between groups at lags 8 and 12 ($Ps > 0.11$).

We examined whether the magnitude of the AB in our patients was influenced by lesion lateralization or lesion chronicity. Planned contrasts of control performance compared to patients with left versus right-sided lesions revealed no significant differences ($Ps > 0.06$). The correlation between lesion chronicity (time post-lesion) and our dependent variable was also non-significant.

4. Discussion

There were four main results from this study:

i. The healthy controls produced the typical AB effect as indicated by an impaired ability to detect a second target when presented in close temporal proximity to the first target (Raymond et al., 1992). The paradigm as designed was effective.
ii. The identification of T1 for the cerebellar group was 96.3 and 97.5% for controls, indicating that both groups were actively engaged in identifying T1.
iii. Cerebellar patients had an increased magnitude of the AB effect that was not significantly prolonged. To the best of our knowledge, this is the first demonstration of impaired attentional blink in patients with isolated cerebellar lesions. This adds to the investigations of the AB in patients with frontal and parietal lesions, in whom variable impairments have been demonstrated.
iv. Identification performance of T2 at the longer temporal lags (8 and 12) did not significantly differ between our groups and a rapid recovery following lag 3 was observed in our patients and controls. Detection accuracy of T2 when it appeared without T1 was slightly, but nonsignificantly, reduced (7.7% relative to controls) in the cerebellar patients.

There are two implications of these results. First, there is an effect of cerebellar lesions on the AB, and the time frame for a cerebellar role in visual attention, is limited to a few hundred (~400–500) milliseconds. Second, the rate of recovery following the "blink" was similar between our groups suggesting that the overall mechanisms of selective attention are unimpaired following damage to the cerebellum.

One commonly held account for the AB effect is that it is a measure of the time required to encode stimuli into visual short-term memory for later conscious report (Chun & Potter, 1995; Jolicoeur & Dell'Acqua, 1998). During this very brief (apparently < 500 ms) process, there is insufficient perceptual or working memory to capture a second target for encoding and T2 representations are "overwritten" by items following in the letter stream (Brehaut, Enns, & Di Lollo, 1999; Dell'Acqua, Pascali, Jolicoeur, & Sessa, 2003; Giesbrecht & Di Lollo, 1998; Seiffert & DiLollo, 1997). We have demonstrated that damage to the cerebellum produces an exaggerated magnitude of the AB effect without extending the time over which the effect is seen or generally reducing vigilance for rapidly appearing stimuli.

The fact, that detection of T1 in the dual-task condition and detection of T2 in the single-task condition were both normal makes a general impairment in sustained attention or vigilance unlikely. Preserved selective attention after cerebellar damage has been previously reported (Gottwald, Mihajlovic, Wilde, & Mehdorn, 2003).

There are three findings that argue that the abnormal AB effect in our patients is not due to a reduced working memory store. First, patients and controls performed similarly on standardized measures of working memory (see Table 2). Second, there was no extension of the duration of the AB effect. Third, detection of T2 in the single-task condition should be susceptible to decay of the expected T2 stimulus properties over time leaving detection influenced by position in the letter stream, but detection accuracy for T1-absent trials was not significantly different between patients and controls and remained consistent across all positions in the letter stream. Recently, Akyurek and Hommel (2005) tested healthy controls on a RSVP task in conjunction with a short-term memory load task. This manipulation effectively reduces working memory capacity. Target detection of both T1 and T2 deteriorated with increased memory load across the entire RSVP stream. Given the fact that our patients identified T1 with near perfect accuracy it is unlikely that impaired working memory could account for our current findings.

If there is an effect of cerebellar lesions on visual attention in the AB paradigm that is not reducible to a deficit in working memory or vigilance or sustained attention, what is the nature of the deficit? In the past 15 years, several explanations for attention

impairments after cerebellar damage have been proposed, and it is likely that there is more than one attentional impairment due to cerebellar lesions. Courchesne and colleagues (Akshoomoff & Courchesne, 1992; Courchesne et al., 1994) demonstrated impairments in the rapid shifting of spatial attention in a small group of patients with a variety of cerebellar pathologies. The authors were the first to propose a specific role of the cerebellum in the rapid deployment of attentional resources required to maintain a large enough attentional space to coordinate cognition (Courchesne et al., 1994). This deployment could be a rapid activation of attentional systems or a "cleansing" inhibition of just completed operations allowing sufficient attentional space for subsequent operations. If arguing by analogy from the role of the cerebellum in motor functioning, one might suppose that the cerebellum completes an open collateral pathway that focuses an intended movement (or cognitive operations) through inhibition of possible divergence from intention. The patient population studied in this work was, however, not optimal for identifying specific roles of cerebellar regions: young adults with autism who are presumed to have developmental cerebellar pathology, children who had undergone surgical resection of astrocytomas (two received radiation) and one adult with idiopathic cerebellar degeneration plus, as the authors point out, extracerebellar atrophy.

Ivry and colleagues (Helmuth et al., 1997; Ravizza & Ivry, 2001; Shin & Ivry, 2003) have also demonstrated that cerebellar damage impairs attention, a result they interpreted as reduced attentional resource capacity when monitoring motor demands consumed resources available to coordinate complex cognitive operations. Using an alternating attention task similar to the task introduced by Courchesne and colleagues (Akshoomoff & Courchesne, 1992; Courchesne et al., 1994), they compared performance of adult patients with cerebellar lesions to a group of patients with Parkinson's disease (Ravizza & Ivry, 2001). The groups showed similar levels of impairment until the investigators varied the motor demands of the task and held the attentional requirements constant. When motor demands were decreased, only the group of patients with cerebellar lesions improved. These results reinforce the possibility that there may be more than one effect of cerebellar lesions on attention, some embedded in motor or action planning—the efferent component of a task, and others independent of motor function or action planning.

The results of the current AB experiment provide evidence for a role of the cerebellum in attention independent of motor impairment at any level. The dependent variable in this paradigm is detection accuracy, not speeded motor responses. Although there is a small motor component—spoken identification of the targets, it is an unspeeded action after the fact of the task. Thus, impairment cannot easily be attributed to demands for simultaneous motor or action planning. For this AB paradigm, the control for possible gaze instability is the nearly perfectly accurate detection of the first target which appeared at varying positions in the letter stream. All stimuli were presented in central fixation, thus impairment cannot be due to a dysmetria of saccades (Golla, Their, & Haarmeier, 2005). None of the patients had any extracerebellar lesion.

The AB effect is sensitive to lesions in a wide range of cerebral regions. The majority of this research does, however, implicate the frontal and parietal lobes. Richer and Lepage (1996) demonstrated an increased magnitude of the AB in patients with large prefrontal excisions but not in patients with temporal lobe resections. Prolonged AB duration has been reported in patients with right inferior parietal or right inferior frontal lesions, either with unilateral neglect (Husain et al., 1997) or without neglect (Shapiro et al., 2002). Rizzo, Akutsu, and Dawson (2001) have reported increased and prolonged AB effects in patients with lesions of either hemisphere and of any region within the hemisphere. Observations of similar effects of widely separated lesions on complex operations are often observed (e.g., spatial attention/neglect and motor control/apraxia) and are interpreted as evidence for an integrated network in which the different regions make distinct contributions to the overall behavior. Based on our results, the network for the temporal control of rapid visual attention should include the cerebellum. Whether there are regionally specific attentional functions within the cerebellum remains to be determined.

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References

Akshoomoff, N. A., & Courchesne, E. (1992). A new role for the cerebellum in cognitive operations. *Behavioral Neuroscience*, *106*(5), 731–738.

Akyurek, E. G., & Hommel, B. (2005). Short-term memory and the attentional blink: Capacity versus content. *Memory and Cognition*, *33*(4), 654–663.

Allen, G., Buxton, R., Wong, E., & Courchesne, E. (1997). Attentional activation of the cerebellum independent of motor involvement. *Science*, *275*, 1940–1943.

Allen, G., McColl, R., Barnard, H., Ringe, W. K., Fleckenstein, J., & Cullum, C. M. (2005). Magnetic resonance imaging of cerebellar-prefrontal and cerebellar-parietal functional connectivity. *Neuroimage*, *28*, 39–48.

Berger, A., Sadeh, M., Tzur, G., Shuper, A., Kornreich, L., Inbar, D., et al. (2005). Task switching after cerebellar damage. *Neuropsychology*, *19*, 362–370.

Bischoff-Grethe, A., Ivry, R. B., & Grafton, S. T. (2002). Cerebellar involvement in response reassignment rather than attention. *The Journal of Neuroscience*, *22*(2), 546–553.

Brehaut, J. C., Enns, J. T., & Di Lollo, V. (1999). Visual masking plays two roles in the attentional blink. *Perception and Psychophysics*, *61*(7), 1436–1448.

Chun, M. M., & Potter, M. C. (1995). A two-stage model for multiple target detection in rapid serial visual presentation. *Journal of Experimental Psychology: Human Perception and Performance*, *21*, 109–127.

Combettes. (1831). Absence compléte du cervelet, des pédoncules postérieurs et de la protubérance cérébrale chez une jeune fille morte dans sa onziéme année. *Bulletin of the Society of Anatomy Paris*, *5*, 148–157.

Courchesne, E., Townsend, J., Akshoomoff, N. A., Saitoh, O., Yeung-Courchesne, R., Lincoln, A. J., et al. (1994). Impairment shifting attention

in autistic and cerebellar patients. *Behavioural Neuroscience*, *108*, 848–865.

Daum, I., Ackermann, H., Schugens, M. M., Reimold, C., Dichgans, J., & Birbaumer, N. (1993). The cerebellum and cognitive functions in humans. *Behavioral Neuroscience*, *107*, 411–419.

Dell'Acqua, R., Pascali, A., Jolicoeur, P., & Sessa, P. (2003). Fourdot masking produces the attentional blink. *Vision Research*, *43*, 1907–1913.

Fiez, J., Petersen, S., Cheney, M., & Raichle, M. (1992). Impaired non-motor learning and error detection associated with cerebellar damage. *Brain*, *115*, 155–178.

Giesbrecht, B., & Di Lollo, V. (1998). Beyond the attentional blink: Visual masking by object substitution. *Journal of Experimental Psychology: Human Perception and Performance*, *24*(5), 1454–1466.

Glickstein, M. (2006). The cerebellum—motor control or more? *Brain*, *129*, 288–289.

Golla, H., Their, P., & Haarmeier, T. (2005). Disturbed overt but normal covert shifts of attention in adult cerebellar patients. *Brain*, *128*, 1525–1535.

Gottwald, B., Mihajlovic, Z., Wilde, B., & Mehdorn, H. (2003). Does the cerebellum contribute to specific aspects of attention? *Neuropsychologia*, *41*, 1452–1460.

Gottwald, B., Wilde, B., Mihajlovic, Z., & Mehdorn, H. (2004). Evidence for distinct cognitive deficits after focal cerebellar lesions. *Journal of Neurology, Neurosurgery and Psychiatry*, *75*, 1524–1531.

Helmuth, L., Ivry, R., & Shimizu, N. (1997). Preserved performance by cerebellar patients on tests of word generation, discrimination learning, and attention. *Learning and Memory*, *3*, 456–474.

Hommel, B., Kessler, K., Schmitz, F., Gross, J., Akyurek, E., Shapiro, K., et al. (2006). How the brain blinks: Towards a neurocognitive model of the attentional blink. *Psychological Research*, *70*(6), 425–435.

Husain, M., Shapiro, K., Martin, J., & Kennard, C. (1997). Abnormal temporal dynamics of visual attention in spatial neglect patients. *Nature*, *385*, 154–156.

Jolicoeur, P., & Dell'Acqua, R. (1998). The demonstration of short-term consolidation. *Cognitive Psychology*, *36*(2), 138–202.

Leiner, H. C., Leiner, A. L., & Dow, R. S. (1986). Does the cerebellum contribute to mental skills? *Behavioral Neuroscience*, *100*, 443–454.

Marcantoni, W. S., Lepage, M., Beaudoin, G., Bourgouin, P., & Richer, F. (2003). Neural correlates of dual task interference in rapid visual streams: An fMRI study. *Brain and Cognition*, *53*, 318–321.

Marois, R., Chun, M. M., & Gore, J. C. (2000). Neural correlates of the attentional blink. *Neuron*, *28*, 299–308.

Marois, R., Yi, D. J., & Chun, M. M. (2004). The neural fate of consciously perceived and missed events in the attentional blink. *Neuron*, *41*, 465–472.

Middleton, F. A., & Strick, P. L. (2001). Cerebellar projections to the prefrontal cortex of the primate. *The Journal of Neuroscience*, *21*, 700–712.

Nieuwenstein, M. R., & Potter, M. C. (2006). Temporal limits of selection and memory encoding: A comparison of whole versus partial report in rapid serial visual presentation. *Psychological Science*, *17*(6), 471–475.

Ravizza, S., & Ivry, R. (2001). Comparison of the basal ganglia and cerebellum in shifting attention. *Journal of Cognitive Neuroscience*, *13*, 285–297.

Raymond, J. E., Shapiro, K. L., & Arnell, K. M. (1992). Temporary suppression of visual processing in an RSVP task: An attentional blink? *Journal of Experimental Psychology: Human Perception and Performance*, *18*, 849–860.

Richer, F., & Lepage, M. (1996). Frontal lesions increase post-target interference in rapid stimulus streams. *Neuropsychologia*, *34*, 509–514.

Rizzo, M., Akutsu, H., & Dawson, J. (2001). Increased attentional blink after focal cerebral lesions. *Neurology*, *57*(5), 795–800.

Schmahmann, J. D. (2001). The cerebrocerebellar system: Anatomic substrates of the cerebellar contribution to cognition and emotion. *International Review of Psychiatry*, *13*, 247–260.

Schmahmann, J. D. (2004). Disorders of the cerebellum: Ataxia, dysmetria of thought, and the cerebellar cognitive affective syndrome. *Journal of Neuropsychiatry and Clinical Neuroscience*, *16*, 367–378.

Schmahmann, J. D., & Caplan, D. (2006). Cognition, emotion and the cerebellum. *Brain*, *129*, 290–291.

Schoch, B., Gorissen, B., Richter, S., Ozimek, A., Kaiser, O., Dimitrova, A., et al. (2004). Do children with focal cerebellar lesions show deficits in shifting attention? *Journal of Neurophysiology*, *92*, 1856–1866.

Seiffert, A. E., & DiLollo, V. (1997). Low-level masking in the attentional blink. *Journal of Experimental Psychology: Human Perception and Performance*, *23*(4), 1061–1073.

Shapiro, K., Hillstrom, A. P., & Husain, M. (2002). Control of visuotemporal attention by inferior parietal and superior temporal cortex. *Current Biology*, *12*, 320–1325.

Shin, J. C., & Ivry, R. B. (2003). Spatial and temporal sequence learning in patients with Parkinson's disease or cerebellar lesions. *Journal of Cognitive Neuroscience*, *15*(8), 1232–1243.

Townsend, J., Courchesne, E., Covington, J., Westerfield, M., Harris, N. S., Lyden, P., et al. (1999). Spatial attention deficits in patients with acquired or developmental cerebellar abnormality. *The Journal of Neuroscience*, *19*, 5632–5643.