Cerebellar lesions disrupt spatial and temporal visual attention.

Craig, B.T.[1-2], Morrill, A.[2], Anderson, B.[3], Danckert, J.[3], & Striemer C.L.[2,4*]

1. Cumming School of Medicine, University of Calgary, Calgary, AB, Canada
2. Department of Psychology, MacEwan University, Edmonton, AB, Canada
3. Department of Psychology, University of Waterloo, Waterloo, ON, Canada
4. Neuroscience and Mental Health Institute, University of Alberta, Edmonton, AB, Canada

* Corresponding Author
Christopher L. Striemer, PhD
Associate Professor
Department of Psychology
MacEwan University
10700 – 104 Avenue
Edmonton, Alberta, Canada, T5J 4S2
Email: striemerc@macewan.ca
Phone: 780-633-3467

Cerebellar contributions to spatial and non-spatial attention

Abstract

The current study represents the first comprehensive examination of spatial, temporal and sustained attention following cerebellar damage. Results indicated that, compared to controls, cerebellar damage slowed the onset of inhibition of return (IOR) during the reflexive covert attention task, and reduced the ability to detect successive targets during an attentional blink task. However, cerebellar damage had minimal influence on voluntary covert attention or the sustained attention to response task (SART). Lesion overlay analysis indicated that impaired performance on IOR and the attentional blink were associated with damage to Crus II and lobule VII (tuber) and VIII (pyramis) of the left posterior cerebellum. Critically, subsequent analyses indicated our results are not due to either general motor impairments or to damage to the deep cerebellar nuclei. Collectively these data demonstrate, for the first time, that the same cerebellar regions are involved in both spatial and temporal visual attention.

Keywords: cerebellum, cerebellar cognitive affective syndrome, inhibition of return, attentional blink, sustained attention

Cerebellar contributions to spatial and non-spatial attention

Traditionally, the cerebellum is considered to be important for the timing and coordination of motor outputs and motor learning (Glickstein, Strata, & Voogd, 2009; Glickstein, Sultan, & Voogd, 2011). However, more recent research has highlighted a role in a diverse array of cognitive, affective, and perceptual processes, including language, working memory, executive control, emotion and motion perception (Adamaszek et al., 2017; Baumann et al., 2015; Bellebaum & Daum, 2007; Marvel & Desmond, 2010; Sacchetti, Scelfo, & Strata, 2009; Schmahmann, Guell, Stoodley, & Halko, 2019; Schmahmann & Sherman, 1998; Stoodley, MacMore, Makris, Sherman, & Schmahmann, 2016; Stoodley & Schmahmann, 2009a; Stoodley & Stein, 2011).

The role of the cerebellum in cognition is supported by anatomical evidence demonstrating connections from the ventral dentate nucleus to non-motor regions of posterior parietal and prefrontal cortex (Clower, West, Lynch, & Strick, 2001; Dum & Strick, 2003; Strick, Dum, & Fiez, 2009). In addition, studies examining functional connectivity have revealed a number of distinct networks in the cerebellum that are functionally connected to different cortical networks known to be involved in a variety of cognitive functions (Buckner, Krienen, Castellanos, Diaz, & Yeo, 2011; Wang, Buckner, & Liu, 2013).

One area of contention regarding the cerebellum's role in cognition concerns its potential involvement in attention. Courchesne and colleagues first demonstrated that patients with cerebellar lesions were slower to shift attention between streams of auditory or visual events (Akshoomoff & Courchesne, 1992, 1994) and were slower to orient attention towards peripheral targets (Townsend et al., 1999). Subsequent studies, however, failed to identify clear attentional deficits in cerebellar patients (Dimitrov et al., 1996; Golla, Thier, & Haarmeier, 2005; Yamaguchi, Tsuchiya, & Kobayashi, 1998). This has led some to suggest that initial findings likely reflected

slowed motor responses (i.e., slowed button presses or eye movements) masquerading as attentional impairments (Glickstein et al., 2011; Haarmeier & Thier, 2007; Ravizza & Ivry, 2001).

There are a number of potential reasons for these conflicting findings. First and foremost, a majority of studies have included a mix of patients with cerebellar lesions, degeneration, and development disorders (Glickstein et al., 2011; Haarmeier & Thier, 2007; Ravizza & Ivry, 2001). Such an approach implicitly assumes that all regions of the cerebellum are *equally involved* in all aspects of the cognitive task being tested. That is, lesion location was, at least initially, not given serious consideration in the analysis or the model of cerebellar involvement in cognition. More recent neuroimaging research, and studies examining patients with circumscribed cerebellar lesions, suggest that distinct cerebellar regions are involved in spatial and non-spatial attention (Baier, Dieterich, Stoeter, Birklein, & Muller, 2010; Schweizer, Alexander, Cusimano, & Stuss, 2007; Striemer, Cantelmi, Cusimano, Danckert, & Schweizer, 2015; Striemer, Chouinard, Goodale, & de Ribaupierre, 2015; Townsend et al., 1999).

Specifically with respect to visuospatial attention, a number of studies have employed the well-known covert attention paradigm developed by Posner and colleagues (Posner, Rafal, Choate, & Vaughan, 1985; Posner, Snyder, & Davidson, 1980). In this task, participants fixate centrally while detecting peripheral targets that can appear at a previously cued location (i.e., a valid trial) or in the location opposite the cue (i.e., an invalid trial). Cues can be either predictive of the impending target location (presumably evincing voluntary allocation of attention) or non-predictive (evincing reflective orienting processes). Previous studies showed deficits in covert attention for predictive cues (Baier et al., 2010; Townsend et al., 1999). However, a recent patient study from our group (Striemer, Cantelmi, et al., 2015) demonstrated that patients with lateral cerebellar lesions showed deficits of reflexive covert orienting. Specifically, cerebellar patients

showed smaller cueing benefits at short SOAs (50ms) and a trend towards a diminished inhibition of return (IOR) – the typical reversal of the cueing effect found at a longer SOAs (Klein, 2000; Posner et al., 1985).

Functional neuroimaging work from our group supports the role of lateral cerebellar regions in reflexive covert orienting (Striemer, Chouinard, et al., 2015). We found significant BOLD activation in lobule VI of the left cerebellum for both reflexive and voluntary covert attention, with or without eye movements and controlling for manual responses. Importantly, activation in the cerebellar ROI was greater for reflexive compared to voluntary attention, and was significantly correlated with increased BOLD activity in superior and inferior parietal lobes, and the frontal eye fields – nodes of the fronto-parietal attention network.

Spatial orienting represents just one kind of attentional process. Attention must also be oriented in time and sustained over time (Husain & Nachev, 2007; Robertson, Manly, Andrade, Baddeley, & Yiend, 1997). Previous research demonstrated an impairment in temporal attention following cerebellar damage using the well-known attentional blink (AB) task (Schweizer et al., 2007). Here participants must identify two targets within a rapid stream of stimuli presented in central vision (Raymond, Shapiro, & Arnell, 1992). Schweizer and colleagues (2007) found that cerebellar patients had a larger AB. That is, they were less accurate at detecting the second target when it appeared shortly after the first. Importantly, patients achieved equivalent target two accuracy levels at about the same time as healthy controls. In other words, their AB was larger in magnitude, but was not prolonged in duration as it is for patients with inferior parietal or superior temporal damage (Husain, Shapiro, Martin, & Kennard, 1997; Shapiro, Hillstrom, & Husain, 2002).

Cerebellar contributions to spatial and non-spatial attention

To our knowledge, only one single case study has examined sustained attention following cerebellar damage (Schweizer et al., 2008). Fundamentally, sustained attention tasks require prolonged focus of attention over longer periods of time, without necessarily taxing the temporal allocation of attention itself (Esterman & Rothlein, 2019). Previous research demonstrated that the cerebellum is involved in executive functions (Bellebaum & Daum, 2007), as well as sequence detection (Leggio & Molinari, 2015; Molinari et al., 2008) and performance monitoring (Peterburs & Desmond, 2016). Thus, it is plausible that cerebellar damage could disrupt the ability to maintain performance on a task over time, which is a core component of sustained attention.

Here we contrasted performance on three standard attention tasks in patients with cerebellar damage and age-matched controls. Participants completed two versions of the Posner cuing paradigm to examine reflexive and voluntary orienting with a view to replicating and extending our prior work. Next, we examined temporal and sustained non-spatial attention by having participants complete versions of the AB task and the Sustained Attention to Response (SART) task (Manly, Robertson, Galloway, & Hawkins, 1999; Robertson et al., 1997). Lesion overlay analyses were used to identify specific regions of the cerebellum linked to observed deficits. The current study is the first comprehensive investigation of the effects of cerebellar lesions on spatial and non-spatial attention in the same patient group. Our results demonstrate, for the first time, that lesions to Crus II and Lobules VII and VIII of the left cerebellum disrupt reflexive spatial attention and the temporal allocation of attention (the AB), while showing minimal influence on voluntary spatial attention or sustained attention.

Methods

Participants

Fourteen patients with cerebellar lesions participated in the current study (mean age 63.57 years; SD=12.57; range 38-83; 6 females). All lesions were a result of cerebellar stroke classified as either a left (n=8), right (n=2), or bilateral (n=4) based on clinical notes and confirmed via MRI (n=8) or CT scans (n=6). All patients were right-handed and had no additional neurological deficits. Testing occurred in the chronic stages post-stroke (mean time post stroke= 4.5 years; Table 1). Patients were recruited form the Neurological Patient Database maintained by the University of Waterloo (Heart and Stroke Foundation funded) in which the patients had previously provided consent to be contacted for research studies.

For comparison, we tested 24 age-appropriate healthy controls (mean age=70.76 years; SD=8.83; range 54-82; 20 females). The two groups did not differ in mean age ($t(21)=1.88$, $p=.073$). Control participants were all right-handed and had no history of neurological impairment. Healthy age-appropriate controls were recruited from either the Waterloo Research in Aging Pool (WRAP; Waterloo, Ontario, Canada), or from the MINERVA Senior Studies Institute at MacEwan University (Edmonton, Alberta, Canada). All participants gave written consent prior to the first testing session.

This project was approved by the MacEwan University Research Ethics Board, the University of Waterloo Research Ethics Board, and the Tri-Hospital Research Ethics Board (Kitchener-Waterloo, Ontario). All participants were compensated $10 per hour for each session.

Table 1: Clinical data for the 14 cerebellar patients. Symbols: ǂ completed the covert attention tasks; ϕ completed the non-spatial attention tasks; * indicates that a new high-resolution MRI was acquired. † indicates damage to the cerebellar dentate nucleus.

Patient ID	Age	Sex	Handedness	Time post stroke (days)	Affected hemisphere	Lesion volume (cm^3)	Imaging notes
29ǂϕ	75	F	R	2955	Left	14.61	Infarct to left posterior inferior cerebellar arteries (PICA)
61ǂϕ*	78	M	R	2791	Left	46.80	Attenuation of left cerebellar hemisphere keeping with acute cerebral infarct
117ϕ*	73	F	R	3707	Bilateral	13.45	Large left cerebellar lesion with smaller lesion in right cerebellar hemisphere
182ǂ ϕ*	66	M	R	2398	Left	9.90	Subacute ischemic stroke in left cerebellar hemisphere
309ǂ ϕ*	73	M	R	2018	Left	34.30	Acute left cerebellar ischemic infarct resulting from thromboembolic event of the posterior inferior cerebellar artery (PICA)
378ǂ ϕ*	53	M	R	1857	Right	16.87	Infarct of right posterior cerebellar hemisphere
522ǂ	67	F	R	1343	Left	27.26	Subacute left cerebellar infarct
523ǂϕ*†	83	F	R	982	Right	12.30	Subacute ischemic stroke in right cerebellar hemisphere. Possible damage to right cerebellar dentate nucleus.
564ǂϕ*	54	F	R	1154	Left	2.28	Large central middle and inferior middle infarct of the left cerebellum
670ϕ	54	M	R	802	Bilateral	6.39	Subacute infarct of right cerebellar hemisphere and a small sub-centimeter hypodensity in the left cerebellar hemisphere
678ǂϕ	61	M	R	784	Left	47.99	Subacute infarct of left posterior inferior cerebellar artery (PICA)
867ǂϕ	65	M	R	139	Bilateral	55.94	Bilateral posterior inferior cerebellar artery (PICA) infarcts
909ϕ†	38	M	R	1742	Bilateral	24.08	Bilateral posterior inferior cerebellar artery (PICA) infarcts with extension into left visual cortex. Damage to left cerebellar dentate nucleus.
953ǂϕ*	50	F	R	536	Left	0.79	Infarct of the left superior cerebellar artery (SCA)

General procedures.

All participants completed four attention tests: reflexive and voluntary covert attention, the AB, and the SART. Each test is described in detail below. All participants completed the four tasks over two separate testing sessions. All button-press responses (when required) were made with the right (dominant) hand. One session tested covert spatial attention (reflexive followed by voluntary covert attention) and the other session tested non-spatial attention (AB and SART; counterbalanced). We always tested reflexive prior to voluntary covert attention in order to avoid any potential carryover effects of the predictive cue contingency in the voluntary task from influencing performance on the reflexive task. The order of the testing sessions (spatial vs. non-spatial attention) was counterbalanced between patients and controls. Some patients (n=8) attended a third session where we obtained a high-resolution T1 MRI anatomical scan of their brain. The remaining patients (n=6) were either unable to attend a third session or were precluded from having an MRI for medical reasons. To assess the presence of motor deficits in the cerebellar patients we administered a modified version of the International Cooperative Ataxia Rating Scale (ICARS Trouillas et al., 1997). The ICARS assessment was administered in the same session as the covert attention tasks in order to keep the two sessions roughly the same length.

International cooperative ataxia rating scale (ICARS).

The ICARS (Trouillas et al., 1997) examines a patient's walking capacity, gait speed, standing capacities and balance. The test also examines dysmetria and intention tremor in each of the upper and lower limbs using the finger-to-nose test, as well as the heel-to-toe test and the timing and coordination of limb movements using alternating pronation and supination of the

hands. Finally, the ICARS assesses oculomotor functions by searching for evidence of gaze-evoked nystagmus, deficits in oculomotor pursuit, or saccadic dysmetria. All tests were scored using the established ICARS scoring procedure (Trouillas et al., 1997). The modified ICARS had a total possible score of 56 (18 (posture and gait) + 32 (limb coordination) + 6 (oculomotor functions)) with higher scores indicative of greater impairment. All ICARS assessments were video recorded for offline analysis and confirmed by two separate raters to check for inter-rater reliability.

Apparatus.

All attention tasks were administered in a dimly lit room on a PC laptop computer with a 53cm x 30cm screen (1920 x 1080 resolution, 60 Hz refresh rate), while resting their head in a chin rest placed 57cm from the screen. All tasks were run using Superlab 5 software (https://www.cedrus.com/superlab/) with responses collected using a Cedrus RB-730 response pad with ± 2-3ms reaction time resolution.

Reflexive covert attention.

For the reflexive covert attention task (Figure 1) a 1cm x 1cm white fixation cross was presented centrally on a uniform black background. Two white boxes 2cm x 2cm size, located 10cm (10°) to the left and right, represented potential target locations. Box size was increased to 2.5cm x 2.5 cm to function as a cue. Targets were an asterisk ('*') 1 cm in diameter presented in the center of one of the two boxes. Participants were asked to fixate centrally while attending to the boxes to the left and right. Fixation was monitored (and recorded) using a Logitech 720 HD webcam zoomed in on the participant's eyes. Given that targets were located 10° to the left and

right, any eye movements toward a target were easily detectable on the webcam. In addition, participants were periodically reminded to maintain fixation. Overall, participants did an excellent job fixating with fewer than 5% of trials excluded due to fixation problems.

Each trial began with a 1000 Hz tone followed by a fixation period of between 1 to 3 seconds (randomly selected equally often at 500ms increments). Following fixation, one of the peripheral boxes appeared to brighten acting as a reflexive cue to attract attention to that location. Following a stimulus-onset-asynchrony (SOA) of either 50, 100, 300, or 600 ms the target appeared at either the cued location (i.e., "valid trials"), or the uncued location (i.e., "invalid trials"). The peripheral cue remained present until the target appeared and was not predictive of the target's location (i.e., 50% valid). Participants responded via button press as quickly and accurately as possible following target onset.

Participants completed two blocks of 190 trials, which consisted of 10 validly and invalidly cued left and right targets for each of the four SOAs, as well as 10 trials in which no cue was presented prior to target onset (i.e., 'no cue' trials). Each block also contained 10 'catch' trials in which the cue was presented but no target appeared. Catch trials were used to ensure that participants were reacting to the onset of the target and not the cue. Participants responded on 3% (or less) of catch trials. Participants completed 12 practice trials prior to completing the main experiment to ensure they understood the task.

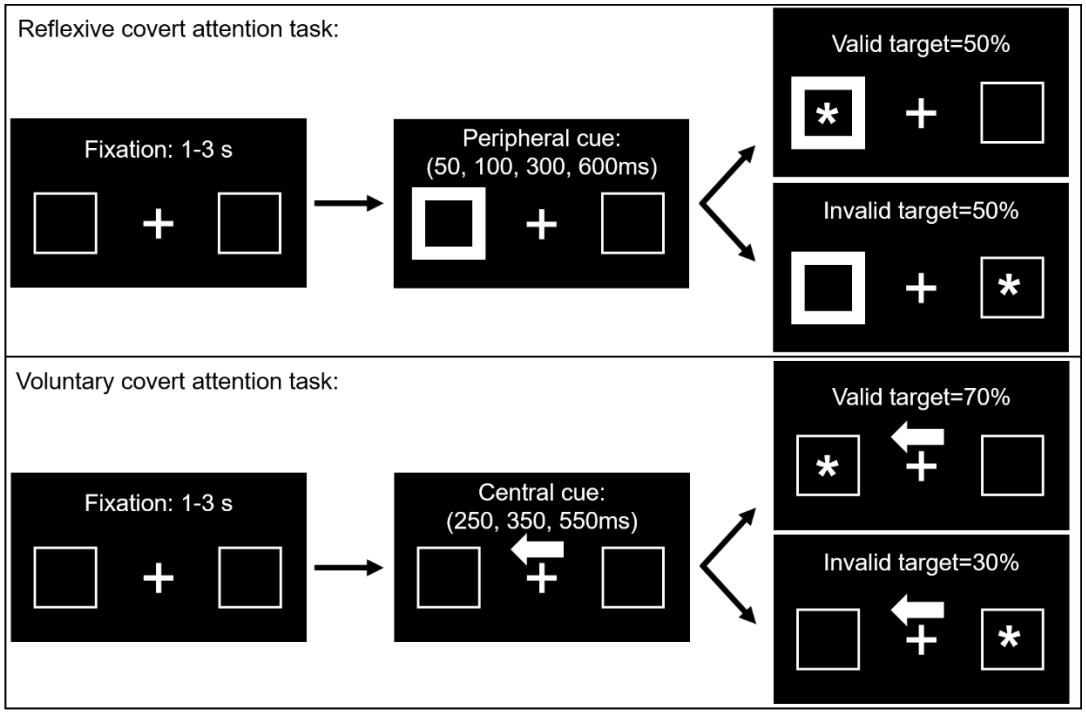
Voluntary covert attention (spatial attention).

The setup for the voluntary covert attention task (Figure 1) was similar to the reflexive covert attention task, with a few differences. First, target locations were cued via a central arrow symbol (1cm tall x 2cm wide) that accurately predicted the target location on 70% of trials. This

version of the covert attention task utilized SOAs of 250, 350, and 550 milliseconds. Participants completed two blocks of 130 trials with 12 invalid trials and 28 valid trials for each SOA for both left and right targets. In addition, we also included 10 no cue trials and 10 ‘catch’ trials. Participants were specifically told about the predictive nature of the cue.

For both covert attention tasks the primary dependent measure was the reaction time (RT) to respond to each trial type (valid vs. invalid) by SOA combination. In addition, to analyze the effect of cue validity as a function of SOA, we calculated cue effect sizes (CES) for each cue x SOA combination by subtracting the RT for valid trials from the RT for invalid trials. The CES represents the benefit of the cue while controlling for overall response speed such that a positive CES reflect a cueing benefit (i.e., faster RTs) for validly cued trials, whereas a negative CES reflects a cueing benefit for invalidly cued trials (i.e., IOR). Reaction times that were below 150ms or more than 2 SD above the participant’s mean for that cue-target-SOA combination were considered outliers and were removed from the analysis.

Figure 1. For the reflexive covert attention task (top panel) a single trial began with a fixation period of 1-3s followed by a peripheral cue on the left or right. Following a stimulus onset asynchrony (SOA) of 50, 100, 300 or 600ms a target (*) subsequently appeared either at the cued (i.e., valid) or uncued (i.e., invalid) location with equal probability. For the voluntary covert attention task (bottom panel) a central arrow cue was used, and the target appeared at the cued location on 70% of trials following an SOA of either 250, 350 or 550ms. Note that for both tasks the cue remained on the screen until the target appeared.



Attentional blink (AB) task.

For the attentional blink (AB) task (Figure 2) we adopted the procedure utilized by Schweizer and colleagues (2007). Participants were presented with an RSVP stream of individual uppercase white or red letters on a black background (67-point font) at a rate of 130ms per letter. Within the AB task there were two types of trials, 1-Target trials and 2-Target trials. 2-Target trials were essential for eliciting the AB effect, whereas 1-Target trials were used as a control condition.

For all 2-Target trials participants were instructed to report the presence of target letters which would always consist of a red H or S (T1) followed by a white X or Y (T2). T1 (red H or S) always appeared prior to T2 (white X or Y) in the letter stream. Non-target letters were drawn from the remaining letters of the alphabet (randomly selected) and were presented in white. In a single 2-Target trial 6-9 non-target letters (with equal probability) were presented prior to T1 (a red H or S), with equal probability. Then a total of 9-12 letters were presented (with equal probability) following T1 in which T2 (a white X or Y, with equal probability) could appear at each of 6 positions or 'lags' (1, 2, 3, 4, 8, or 12 letters after T1, with equal probability). In addition, there were always 1-4 letters presented after T2. Following the letter stream participants were asked to indicate whether any red target letters were presented (H or S, or "no"), and whether a white X or Y was presented. After each trial the researcher coded the participant's responses using the keyboard before pressing the spacebar to move to the next trial. For 2-Target trials the primary dependent measure was the participant's accuracy in identifying T2 after *correctly identifying* T1 as a function of lag.

1-Target trials were similar to 2-Target trials except that, for 1-Target trials, T1 (the red H or S) was replaced by a white non-target letter (randomly selected) which was then followed by T2 (a white X or Y) presented under the same constraints as in 2-Target trials (described above).

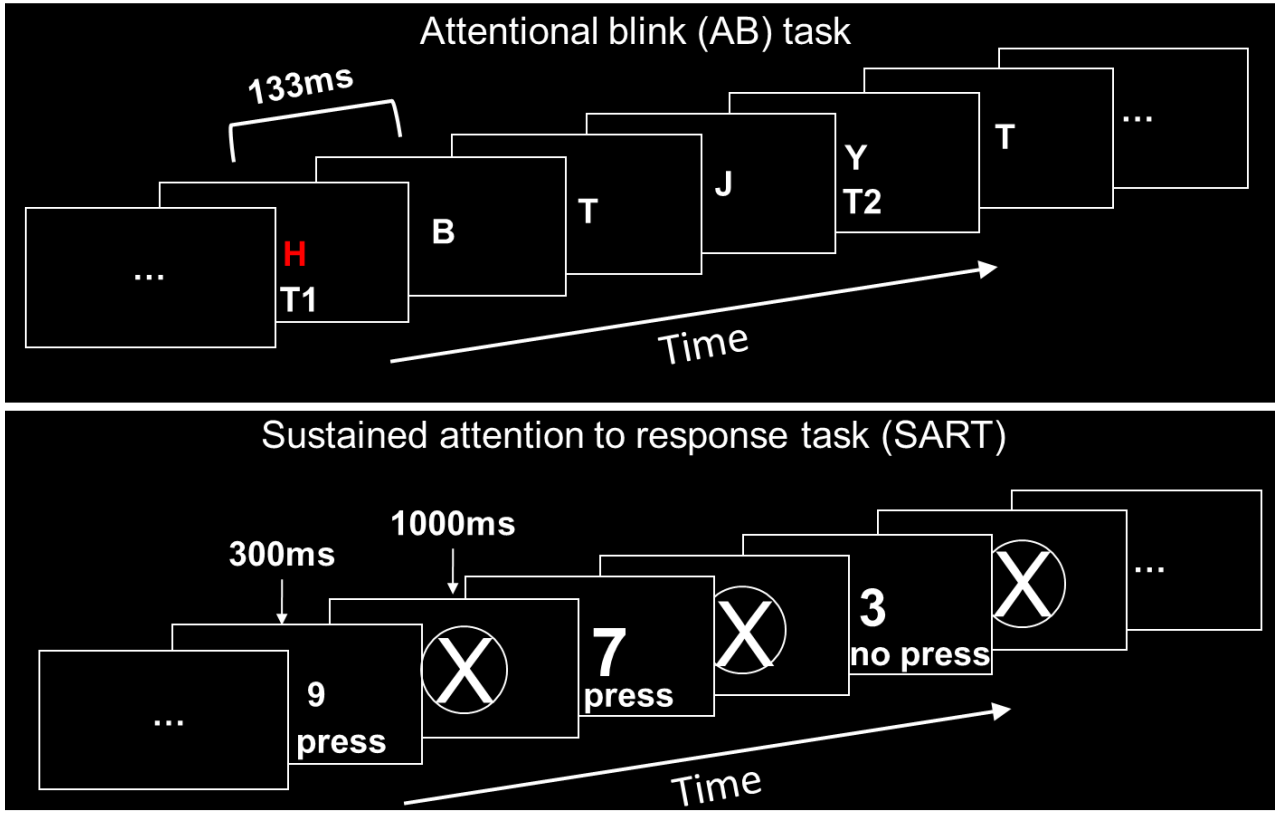
Following the letter stream participants were again asked to identify if any red letters were presented (H, S or "no") and whether a white X or Y was presented. The primary dependent measure on these trials was the accuracy in identifying T2 in the absence of T1.

Participants performed 180 trials with 1/3 being 1-Target trials, and 2/3 being 2-Target trials. 1-Target and 2-Target trials were intermixed and randomly presented. Participants were given 10 practice trials before completing the main task and were offered a short break halfway through. Control participants were removed as outliers if their accuracy for either the 1-Target or 2-Target tasks was more than 2SD outside of the overall group mean for a given Lag.

Sustained attention to response task (SART).

In the SART the digits 1-9 were presented one at a time in the center of the screen in random order. Each digit was presented for 300ms, followed by a 1000ms mask. Participants pressed the space bar of the computer keyboard as quickly and accurately as possible for every number except '3'. When a '3' appeared, participants were told to withhold their response. All digits were a standard white Arial font that varied in size (48, 72, 94, 100 or 120pt font) and were presented on a black background. The mask was a large white circle with an X in the center. There were 225 numbers presented in total with each number presented 25 times in random order (25 of them being 3). Participants were given 15 practice trials prior to the experimental trials to ensure they understood the task. Here, we measured the percentage of commission errors (i.e., presses for '3'), and misses (or omission errors – failing to press for numbers other than 3), as well as the RTs for errors and correct responses. Responses that were under 150ms were discarded as anticipatory.

Figure 2. During the attentional blink (AB; top panel) task participants were presented with a rapid serial visual presentation (RSVP; 133ms/letter) where they were asked to indicate the presence of a red H or S or a white X or Y. On 1-Target trials only a white X or Y was presented. On 2-Target trials a white X or Y was presented either 1, 2, 3, 4, 8, or 12 letters after the presentation of a red H or S. For the sustained attention to response task (SART; bottom panel) participants were to press a button each time a number appeared on the screen except for '3'. Each number appeared for 300ms followed by a mask period of 1000ms.



Lesion analysis.

To explore the association between lesion location and behaviour we acquired medical imaging data for each of the 14 patients and performed lesion overlay analyses. For 8 patients we acquired high resolution 160 slice 1mm ISO-voxel T1-weighted MRI scans collected on a 1.5T Philips scanner (Grand River Hospital, Kitchener, Ontario). The remaining six patients were either unable to undergo an MRI scan due to safety concerns (e.g., surgical implants, metal in their body), or elected not to do so for personal reasons. For these patients we acquired existing MRI or CT scan data from their medical records. For all patients lesions were traced by an experienced neurologist (B.A.) using MRIcron software (http://people.cas.sc.edu/rorden/mricron/index.html). All patient anatomicals and lesion masks were normalized onto a high-resolution CT template using the Clinical Toolbox for SPM 12 (Rorden, Bonilha, Fridriksson, Bender, & Karnath, 2012). The normalized individual lesion masks were then combined to make a group lesion mask in MRIcron which was overlaid onto the same high-resolution CT template. We then extracted the MNI coordinates for the regions of maximum lesion overlap and converted them into Talairach coordinates. These Talairach coordinates were used to localize the lesioned regions using the Talairach Daemon Atlas (http://www.talairach.org/). In addition to examining lesion overlap for the overall group, we also examined each patient's scan for damage to the cerebellar dentate output nuclei using the probabilistic dentate atlas developed by Dimitrova and colleagues (Dimitrova et al., 2006).

Statistical analyses.

Statistical analyses were carried out using SPSS 25. All within-subject ANOVAs were computed using a Greenhouse-Geisser correction when necessary (Greenhouse & Geisser, 1959),

and all post-hoc tests were carried out using the Tukey procedure to control for familywise error rate ($p<.05$).

Data availability statement.

The authors confirm that the data supporting the findings of this study are available within the article and its supplementary material.

Results

International Cooperative Ataxia Rating Scale (ICARS).

We acquired International Cooperative Ataxia Rating Scale (ICARS) scores for 12 of the 14 patients, with one patient dropping out of the study prior to completing this portion and data for a second patient lost due to experimenter error. ICARS data are shown in Table 2. There was high inter rater reliability for overall ratings on the ICARS ($r(11)=.98$, $p<.0001$).

Table 2. International cooperative ataxia rating scale (ICARS) data for the cerebellar patients (n=12). Total scores for each subtest and overall scores are listed for each patient (side of lesion in brackets). Larger numbers indicate greater motor impairment. Percentage impairment scores were calculated by dividing the patient's raw score by total possible score (Trouillas et al., 1997).

	Patient ID:																							
Test:	**29(L)**		**61(L)**		**117(B)**		**182(L)**		**309(L)**		**378(R)**		**522(L)**		**523(R)**		**564(L)**		**678(L)**		**867(B)**		**953(L)**	
Walking /8	6		0		5		0		0		0		0		5		0		0		2		0	
Gait /4	3		1		1		0		1		0		0		3		2		1		3		0	
Standing /6	5		1		2		2		2		1		2		3		2		2		2		0	
Total: Posture & gait /18	**14**		**2**		**8**		**2**		**3**		**1**		**2**		**11**		**4**		**3**		**7**		**0**	
	L	**R**	**L**	**R**	**L**	**R**	**L**	**R**	**L**	**R**	**L**	**R**	**L**	**R**	**L**	**R**	**L**	**R**	**L**	**R**	**L**	**R**	**L**	**R**
Heel-to-toe /4	1	1	0	0	2	1	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	3	0
Finger-to-nose /4	2	3	0	0	4	2	2	2	1	1	0	0	1	0	1	3	0	0	1	0	1	2	1	0
Pronation-supination /4	3	4	0	0	4	2	1	0	0	0	0	0	1	0	1	2	1	1	1	0	0	0	2	0
Total: Kinetic/ 24	**6**	**8**	**0**	**0**	**10**	**5**	**3**	**2**	**2**	**1**	**0**	**0**	**2**	**0**	**2**	**5**	**1**	**1**	**2**	**0**	**1**	**2**	**6**	**0**
Nystagmus /3	0		0		1		1		0		0		0		1		1		0		1		0	
Pursuit /2	1		0		2		1		0		0		0		1		0		1		1		1	
Saccade dysmetria /1	1		0		0		0		0		0		0		0		1		1		0		0	
Total: Oculomotor 6	**2**		**0**		**3**		**2**		**0**		**0**		**0**		**2**		**2**		**2**		**2**		**1**	
Total: Overall /48	**30**		**2**		**26**		**9**		**6**		**1**		**4**		**20**		**8**		**7**		**12**		**7**	
Percent impairment:	**63%**		**4%**		**54%**		**19%**		**13%**		**2%**		**8%**		**42%**		**17%**		**15%**		**25%**		**15%**	

Spatial attention tasks.

All RT data for the covert attention tasks for each patient and the controls are presented in Supplementary Tables 1 & 2. Of the 14 patients recruited, data was collected for the covert spatial attention tasks on only 11 as the remaining three patients had lesions extending into occipital cortex resulting in partial peripheral vision loss that made it difficult to view one of the peripheral boxes when fixating. Which patients completed each attention task is noted in Table 1.

Reflexive covert attention.

We analyzed non-cued trials using a mixed model ANOVA with side of target (left vs. right) as a within-subject factor and group (patients vs. controls) as a between-subject factor. This analysis revealed no significant main effects or interactions (all *p*'s >.07). Given that there was no effect of side of target we collapsed these data in subsequent analyses.

A mixed-model ANOVA with group (patients vs. controls) as a between-subject factor, and cue (valid vs. invalid) and SOA (50, 100, 300, 600) as within-subject factors, revealed main effects of cue ($F(1,34)=111.58$, $p<.0001$, $\eta^2=.77$), SOA ($F(2.3,78.8)=5.07$, $p=.006$, $\eta^2=.13$), and group ($F(1,34)=4.31$, $p=.045$, $\eta^2=.11$). Specifically, RTs were faster for valid (556ms) compared to invalid (596ms) trials, RTs were slower overall at the 50ms SOA (590ms) compared to all other SOAs (100=576ms; 300=568ms; 600=569ms; all *p's* <.05, Tukey corrected), and RTs were slower overall for patients (612ms) compared to controls (540ms).

There was a significant cue x SOA x group interaction ($F(2.7,90.4)=3.84$, $p=.016$, $\eta^2=.10$; Figure 3A). This was driven by a difference in CES at 600 ms SOA. Patients showed a large, positive CES (mean=49 ms), whereas for controls the CES was essentially not different from zero

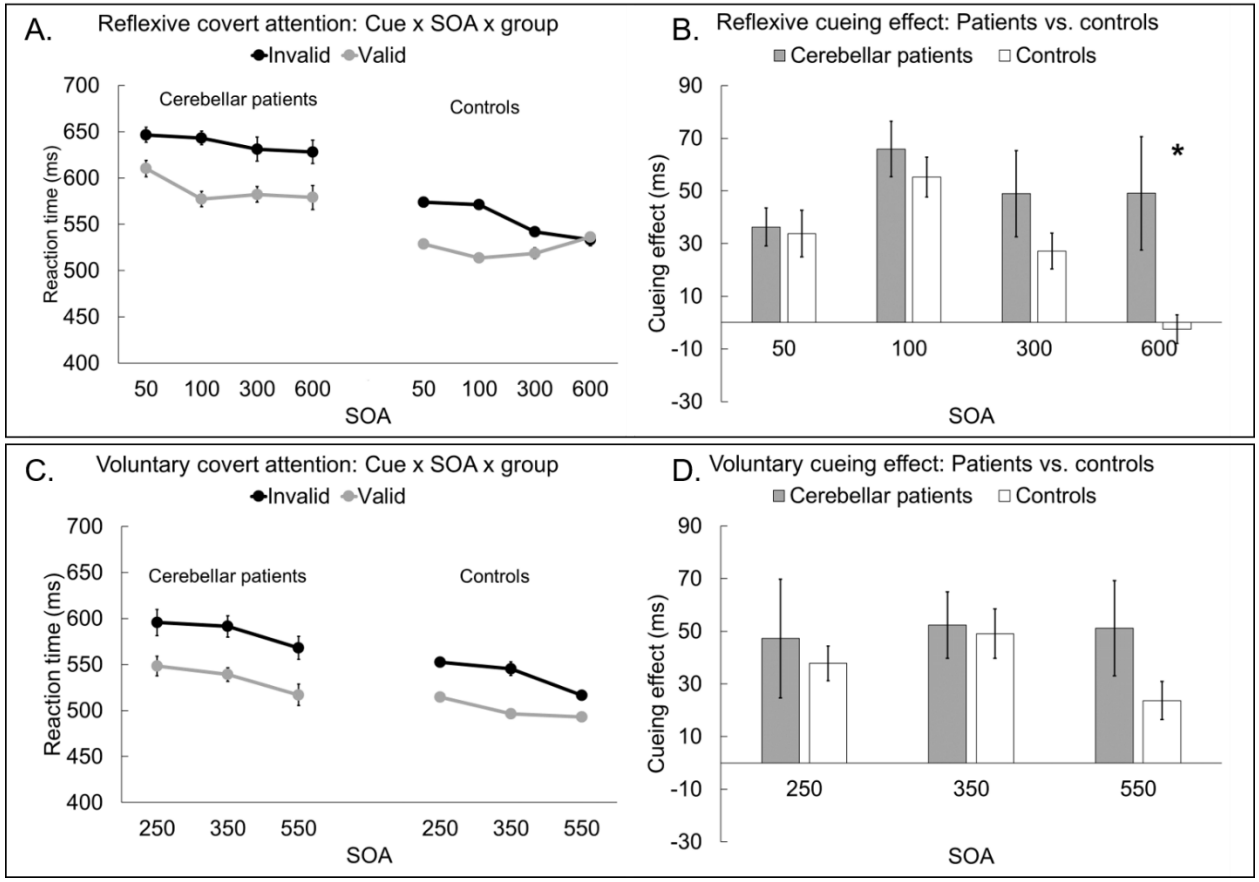
(mean=-3 ms; t(34)=3.18, $p<.05$, Tukey corrected). CES sizes at all other SOAs did not differ between groups (all p's >.15, uncorrected; Figure 3B).

Voluntary covert attention

A mixed-model ANOVA (group x side of target) analysis of the non-cued trial data revealed no significant main effects or interactions (all p's >.16). Again, given that there was no effect of side of target we collapsed these data in subsequent analyses.

For cued trials, a mixed-model ANOVA (group x cue x SOA (250, 350, 550)) revealed main effects of cue (F(1,33)=70.19, $p<.0001$, $\eta^2=.68$) and SOA (F(2,66)=15.19, $p<.0001$, $\eta^2=.32$), with no other main effects or interactions (Figure 3C). For comparison to the reflexive covert attention task, we also compared CES (invalid–valid RT) between the two groups at each SOA (Figure 3D). This analysis did not reveal any significant differences (all p's >.10, uncorrected).

Figure 3. Reaction time (RT) data for the reflexive (A) and voluntary (C) covert attention tasks are presented as a function of group (patients vs. controls) cue (valid vs. invalid) and stimulus onset asynchrony (SOA). Error bars represent the within-subject standard error (Loftus & Masson, 1994). **Cue effect size data** (i.e., invalid minus valid RTs) for the reflexive (B) and voluntary (D) covert attention tasks are presented as a function of group (patients vs. controls) and SOA. Error bars represent standard error. * indicates a statistically significant difference.



Non-spatial attention tasks.

All accuracy and RT data for the non-spatial attention tasks for each patient and controls are presented in Supplementary Tables 3 & 4. For the non-spatial attention tasks, we collected data from 13 of 14 patients as one dropped out of the study before completing this session.

Attentional blink (AB).

We first analyzed the percentage of errors for the 1-Target and 2-Target trials using a mixed-model ANOVA with group (patients vs. controls) as a between-subject factor and trial type (1-Target vs. 2-Target) and lag (1, 2, 3, 4, 8, 12) as within-subject factors (Figure 4A). This analysis revealed significant main effects of trial type ($F(1,33)=20.04$, $p<.0001$, $\eta^2=.38$), lag ($F(5,165)=16.82$, $p<.0001$, $\eta^2=.34$), and group ($F(1,33)=4.66$, $p=.038$, $\eta^2=.12$). Specifically, participants had lower accuracy for 2-Target trials (72%) compared to 1-Target trials (78%) and had higher accuracy in the longest 2 lags (8, 12) compared to the shortest 4 lags (1, 2, 3, 4; all t's >3.02, all *p*'s <.05, Tukey corrected). Patients also had lower accuracy overall (72%) compared to controls (78%). In addition, controls demonstrated the well-known lag-1 sparing effect (1-Target=76% vs. 2-Target=70%; $t(21)=1.54$, $p=.14$), an effect that was absent in patients (1-Target=72% vs. 2-Target=54%; $t(13)=3.27$, $p=.014$, Tukey corrected).

There was also a significant trial type x lag interaction ($F(5,165)=5.61$, $p<.0001$, $\eta2=.15$). This was driven by a significantly larger difference in performance for the 2-Target compared to 1-Target trials for first 3 lags (lag 1=11%, lag 2=15%, lag 3=12%) compared to the last 3 lags (lag 4=2%, lag 8=-3%, and lag 12=-5%; all t's > 3.02, all *p*'s <.05, Tukey corrected).

A trial type x group interaction ($F(1,33)=5.66$, $p=.023$, $\eta^2=.15$) indicated that, overall, the two groups had similar accuracy for 1-Target trials (patients=77% vs. controls=80%; $t(33)=1.02$,

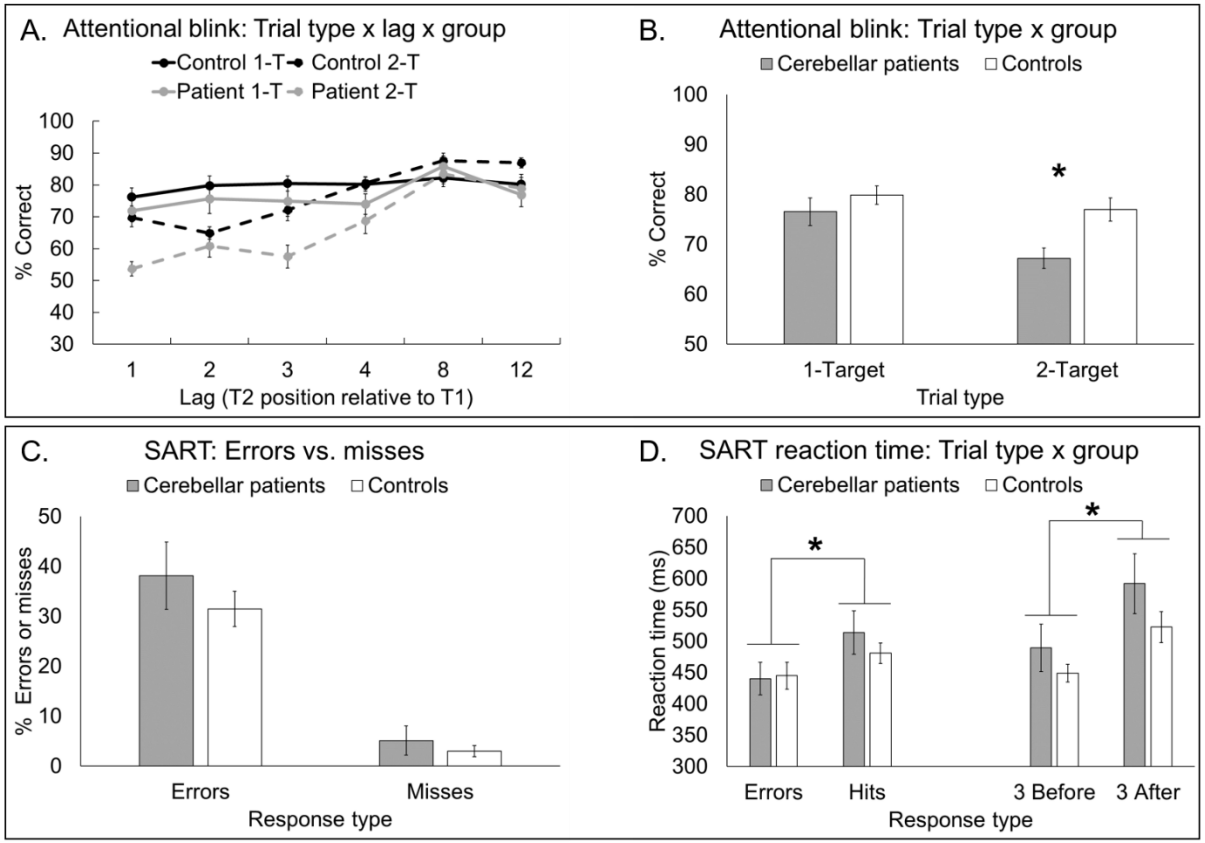
p=.32, uncorrected). However, patients had significantly lower accuracy on 2-Target trials (67%) compared to controls (77%; t(33)=2.89, *p*<.05, Tukey corrected; Figure 4B).

Sustained attention to response task (SART).

For the SART we first compared the percentage of commission errors (i.e., presses for "3") and misses (i.e., omission errors) across the two groups (Figure 4C). This analysis revealed no significant differences (commission errors; patients=38% vs. controls=31%; t(34)=1.06, *p*=.30; omission errors; patients=5% vs. controls=3%; t(34)=0.78, *p*=.44)[1]. Next, we examined RTs by comparing the average RTs for errors of commission vs. correct responses between the two groups using a mixed-model ANOVA with trial type (errors vs. hits) as a within-subject factor and group (patients vs. controls) as a between-subject factor (Figure 4D). This analysis demonstrated a significant main effect of trial type with RTs for error trials (443ms) being faster than RTs for hits (498ms; F(1,34)=15.07, *p*<.0001; η^2=.31). No other effects were significant. We used a similar ANOVA to compare the average RT for the three trials immediately preceding an error of commission compared to the average RTs for the three trials immediately following an error. This analysis also revealed a significant main effect of trial type such that participants were faster to respond in trials immediately preceding an error (469ms) compared to trials following an error (557ms; F(1,34)=30.23, *p*<.0001; η^2=.47). No other effects were significant. Finally, to examine RT variability we conducted a trial type x group ANOVA on the SD of RTs for errors of commission, as well as RTs for correct trials. This analysis revealed no main effects or interactions, indicating that RT variability was not different between cerebellar patients and controls (all *p*'s >.21).

[1] A subsequent analysis using d' also revealed no significant group differences in sensitivity (controls d'=2.75 vs. patients d'=2.36; t(33)=1.06, *p*=.29).

Figure 4. Accuracy data for the attention blink task (panels A & B) are presented as a function of group (patients vs. controls), trial type (1-Target vs. 2-Target) and lag (1, 2, 3, 4, 8, 12). Percentage of errors or misses (panel C) and reaction time (RT) data (panel D) for the sustained attention to response task (SART) are presented as a function of response type (hit, miss or error). Error bars for panel A represent the within-subject standard error (Loftus & Masson, 1994). Error bars for the remaining panels (B-D) represent the standard error. * indicates a statistically significant difference.



Correlation analysis

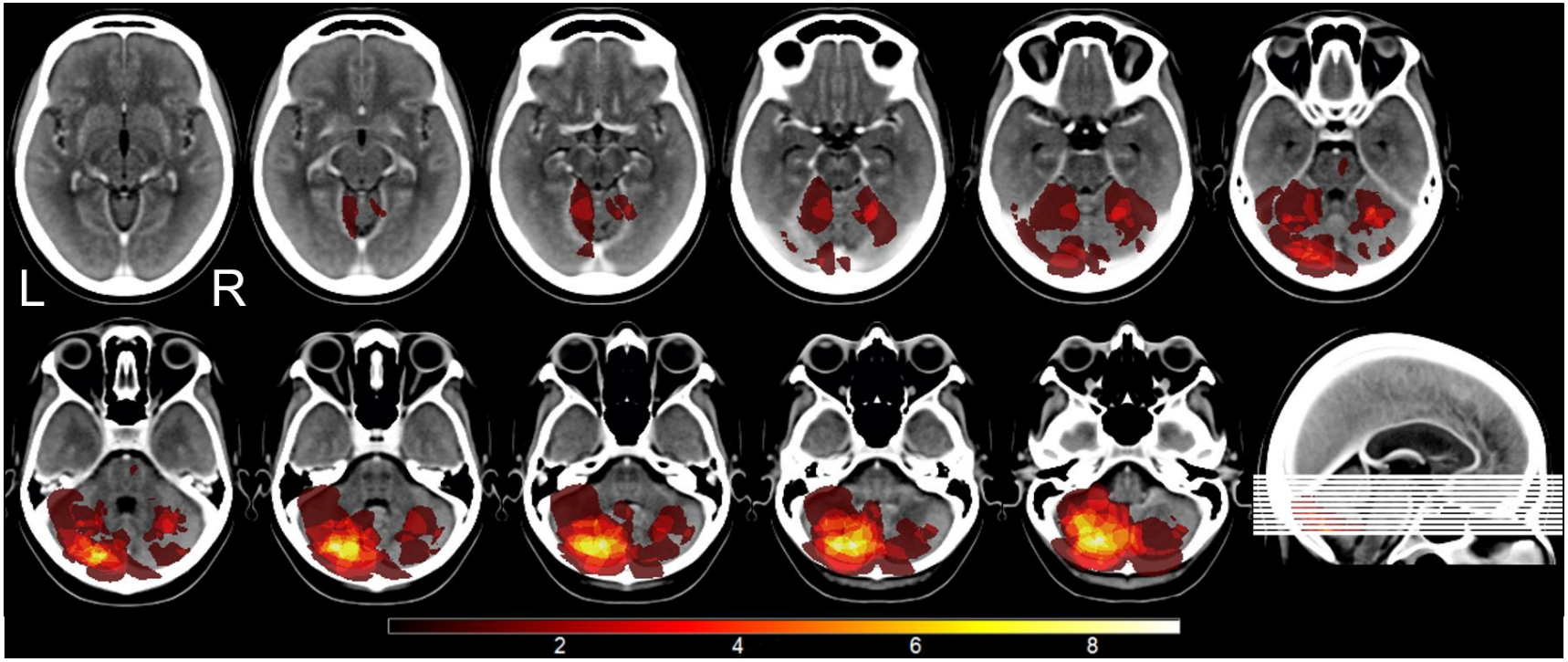
In addition to analyzing data from the attention tasks, we also wanted to examine whether performance on any of the attention tasks was correlated with any relevant clinical measures such as time post stroke, lesion volume, or motor impairment (i.e., total ICARS score). No significant correlations emerged between overall CES for the two covert attention tasks or performance on the AB (2-Target – 1-Target accuracy) or SART (errors of commission) tasks with either lesion volume, time post stroke, or the overall score on the ICARS (all *p*'s >.15, uncorrected).

Lesion analysis

Lesion maps for each patient are available online (Supplementary Figure 1). The areas of maximum lesion overlap in our group are the inferior semilunar lobule (i.e., Crus II; x= -16, y= -78, z= -35 and x= -19, y=-74, z= -38), tuber (lobule VII; x= -29, y=-79, z=-28), and pyramis (lobule VIII; x= -16, y= -76, z=-30) of the posterior lobe of the left cerebellum (Figure 5). Note that subsequent analyses of the group lesion masks for patients who completed the reflexive covert attention tasks (n=11) and the AB task (n=13) revealed largely the same results (Supplementary Figure 2; Supplementary Table 5).

To check for evidence of damage to the dentate nucleus we localized the dentate using the probabilistic 3D atlas developed by Dimitrova and colleagues (2006). Based on their maximal MNI coordinates for the left (X= -15, Y= -57, Z= -36) and right (X= 19, Y= -55, Z= -36) dentate, only two patients had damage to the dentate nucleus (patient 909, left dentate; patient 523, probable right dentate). Note that the absence of an IOR effect and the increased AB effect are still apparent in our cerebellar patients even when the two patients with dentate damage are excluded from the analyses (see Supplementary Material).

Figure 5. Lesion overlap for the cerebellar patient group (n=14). The regions of maximum lesion overlap are the inferior semilunar lobule (i.e., Crus II; x= -16, y= -78, z= -35 and x= -19, y=-74, z= -38), tuber (lobule VII; x= -29, y=-79, z=-28), and pyramis (lobule VIII; x= -16, y= -76, z=-30) of the posterior lobe of the left cerebellum.



Discussion

Cerebellar damage impairs the ability to orient attention in both time and space. Specifically, for reflexive orienting of spatial attention we demonstrated clear evidence that cerebellar damage results in the slowed onset of IOR (Figure 3A&B), such that the cueing effect for cerebellar patients was still quite large (49ms) compared to a non-significant cueing effect for controls (-3ms; Figure 3B). The increased cueing effect in cerebellar patients at the longest (600ms) SOA provides a conceptual replication and extension of our earlier work (Striemer, Cantelmi, et al., 2015) in which patients with cerebellar injury showed a trend towards a slowed onset of IOR at a shorter (300ms) SOA. However, in contrast to our previous study (Striemer, Cantelmi, et al., 2015), we did not see any evidence of a slower orienting response at the earliest (50ms) SOA. Although it is unclear why this effect at the earliest SOA was not observed here, the slowed onset of IOR appears to be a consistent impairment observed following damage to the lateral cerebellum as this effect has now been observed in two separate studies with different patient groups. Previous studies suggest that IOR may be related to an inhibition of response to a previously attended location (for a review see Chica, Martin-Arevalo, Botta, & Lupianez, 2014). Thus, damage to the posterior-lateral cerebellum may impair the ability to inhibit responding at previously attended locations. Confirmation of this hypothesis will require future investigation.

In the current study voluntary covert attention was relatively unaffected by cerebellar damage. This provides support for our earlier hypothesis that the cerebellum plays a larger role in reflexive than voluntary covert attention (Striemer, Cantelmi, et al., 2015; Striemer, Chouinard, et al., 2015). This greater impairment in reflexive attention may be due to the fact that peripheral abrupt onset cues (like the ones used in the reflexive attention task in the current study) are effective in attracting both shifts of attention (Jonides & Yantis, 1988), as well as eye movements

(Mulckhuyse, van Zoest, & Theeuwes, 2008; Schreij, Owens, & Theeuwes, 2008). In contrast, central arrow cues require more time to be interpreted before a saccade plan can be generated (Jonides, 1981; Muller & Rabbitt, 1989). Therefore, it is likely that abrupt onset peripheral cues result in a stronger engagement of oculomotor structures in both the cerebral cortex and cerebellum. Furthermore, damage to cerebellar regions involved in saccadic eye movements such as lobule VII (Robinson & Fuchs, 2001), which is also part of the region of maximum lesion overlap in the current study (Figure 5), would be more likely to impair performance on reflexive compared to voluntary covert attention tasks in general.

In addition to the slowed onset of IOR, cerebellar damage also resulted in a larger overall AB effect (Schweizer et al., 2007); (Figure 4 A&B) and the absence of the well-known lag-1 sparing effect (Raymond et al., 1992). Previous research suggests that lag-1 sparing is associated with the binding of the two targets into a single event (Hommel & Akyurek, 2005; Raymond et al., 1992). Thus, the absence of lag-1 sparing and a larger overall AB effect implies a slowed rate of processing each item in the RSVP stream. This is consistent with the notion that the cerebellum plays an important role in the detection and processing of sequences (Baumann et al., 2015; Leggio & Molinari, 2015), as well as anticipating the timing of sensory events in order to reduce performance variability (Ghajar & Ivry, 2009). Damage to such a mechanism would make it difficult to process a rapid series of events over a short time as is required during a task like the AB.

Although cerebellar damage resulted in problems with the processing of a rapid series of visual events over time as quantified by the AB, there was no problem with sustained attention per se as cerebellar patients performed similarly to controls on the SART. Specifically, patient's error rates, sensitivity, RTs, RT variability and post-error slowing on the SART were statistically

indistinguishable from controls, suggesting that the cerebellar regions damaged in our patients are not involved in updating performance following attentional errors. If one assumes a vigilance task like the SART requires some degree of performance monitoring (an executive function, Jurado & Rosselli, 2007) then the absence of any impairment on the SART following cerebellar injury is somewhat surprising given that previous research has argued that the cerebellum plays an important role in performance monitoring (Peterburs & Desmond, 2016) and executive functions more generally (Bellebaum & Daum, 2007). Consistent with the present findings, two previous patient studies observed that cerebellar lesions did not impair performance on a Go/NoGo attention task that is similar in nature to the SART (Gottwald, Mihajlovic, Wilde, & Mehdorn, 2003; Gottwald, Wilde, Mihajlovic, & Mehdorn, 2004). However, neuroimaging studies tell a different story. Specifically, neuroimaging studies suggest that Crus I in the left cerebellum is consistently activated during tasks that measure executive functions (see Stoodley & Schmahmann, 2009b for a meta-analysis). It is worth noting that the regions of maximum lesion overlap in the current study were Crus II and lobule VII and VIII (Figure 5). Thus, the fact that we did not observe deficits in SART task performance might be related to the fact that, on average, Crus I was largely spared in our patients.

As mentioned previously, our lesion analysis revealed that the regions most consistently damaged in our patients were lobule VII (tuber), lobule VIII (pyramis) and Crus II (inferior semi-lunar lobule) of the left posterior cerebellum (Figure 5). We also examined each patient's scan for any evidence of damage to the cerebellar dentate nucleus with only two patients showing evidence of damage to this region (in opposing hemispheres). This result is important given that damage to the dentate nucleus would effectively disconnect the entire lateral cerebellar hemisphere from the cerebral cortex. Thus, the deficits observed here are due to damage to lobule VII, VIII and Crus II

of the left cerebellum, and not the disruption of an entire cerebellar hemisphere. This was further reinforced by follow-up analyses (see Supplementary Material) where the reduced IOR effect and the increased AB effect were still present in our cerebellar patients even when the data from the two patients with dentate damage were removed. Resting state functional connectivity data indicate that the same cerebellar regions damaged in our patients are functionally connected with the dorsal and ventral attention networks (Brissenden, Levin, Osher, Halko, & Somers, 2016; Buckner et al., 2011; Guell, Schmahmann, Gabrieli, & Ghosh, 2018; Wang et al., 2013) and are active during divided attention tasks (King, Hernandez-Castillo, Poldrack, Ivry, & Diedrichsen, 2018).

It is noteworthy that the attentional deficits we observed following cerebellar lesions were most apparent after damage to the *left* cerebellum. This is consistent with our previous fMRI study (Striemer, Chouinard, et al., 2015) demonstrating significant BOLD activity in the left cerebellum during covert attention tasks, and with the fact that regions of the left cerebellum such as lobule VI, VIII and Crus II are functionally connected to the ventral attention network in the right hemisphere (Buckner et al., 2011; Wang et al., 2013).

It is also important to emphasize that the attentional deficits observed in our cerebellar patients are not simply due to motor impairments. Specifically, there were no significant correlations between the total score on the ICARS and performance measures on any of the attention tasks that we used.

What role does the cerebellum play in attention?

Our findings support the notion that cerebellar damage disrupts the allocation of attention across spatial and temporal domains resulting in a form of “attentional dysmetria.” One prominent

theory suggests that the cerebellum coordinates motor output by comparing it to predicted sensory consequences (Ghajar & Ivry, 2009; Sokolov, Miall, & Ivry, 2017). The cerebellum may then "generate time-based expectancies of sensory information" in order to more efficiently synchronize predicted with actual sensory input in order to help reduce performance variability (Ghajar & Ivry, 2009). This prediction is similar to suggestions that lesions to the cerebellum produce "dysmetria of thought" through disrupting the timing and coordination of cognitive processing (Schmahmann et al., 2019; Schmahmann & Sherman, 1998). Our data directly support these theories by demonstrating that lesions to these same cerebellar regions (left lobules VII, VIII and Crus II) lead to an impairment on the AB (see also Schweizer et al., 2007), as well as the slowed onset of IOR.

In summary, we have demonstrated, for the first time, that damage to Crus II and lobule VII (tuber) and VIII (pyramis) disrupt the onset of IOR and reduces the capacity to detect successive targets during a rapid serial visual presentation (i.e., an increased AB effect). Importantly, anatomical studies in non-human primates, as well as connectivity and task-based functional neuroimaging studies in humans, have demonstrated that these same cerebellar regions are linked with cortical networks that are known to be involved in attention. These data therefore provide direct evidence that 1) the cerebellum plays a critical role in attention, and 2) that the same cerebellar regions are implicated in both spatial and temporal attention.

Acknowledgements:

The authors would like to thank Nadia Botha and Hanbin Go for their assistance with data collection and analysis and Drs. Jörn Diedrichsen and Carlos Hernandez-Castillo (Brain and Mind Institute, University of Western Ontario) for their assistance with the cerebellar lesion analysis.

The authors would also like to thank Nadine Quehl and Karly Neath-Tavares for their assistance with patient recruitment. This research was supported by Natural Sciences and Engineering Research Council of Canada (NSERC) Discovery Grants to C.S., J.D. and B.A., a Canadian Institutes of Health Research (CIHR) grant awarded to J.D. and B.A, Canadian Foundation for Innovation (CFI) and Heart and Stroke Foundation grants to J.D., a Strategic Research Grant from MacEwan University to C.S, and NSERC Undergraduate Student Research and CIHR Vanier Awards to B.T.C.

References:

Adamaszek, M., D'Agata, F., Ferrucci, R., Habas, C., Keulen, S., Kirkby, K. C., . . . Verhoeven, J. (2017). Consensus Paper: Cerebellum and Emotion. *Cerebellum, 16*(2), 552-576. doi:10.1007/s12311-016-0815-8

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

Akshoomoff, N. A., & Courchesne, E. (1994). ERP Evidence for a Shifting Attention Deficit in Patients with Damage to the Cerebellum. *Journal of Cognitive Neuroscience, 6*(4), 388-399. doi:10.1162/jocn.1994.6.4.388

Baier, B., Dieterich, M., Stoeter, P., Birklein, F., & Muller, N. G. (2010). Anatomical correlate of impaired covert visual attentional processes in patients with cerebellar lesions. *Journal of Neuroscience, 30*(10), 3770-3776. doi:30/10/3770 [pii]
10.1523/JNEUROSCI.0487-09.2010

Baumann, O., Borra, R. J., Bower, J. M., Cullen, K. E., Habas, C., Ivry, R. B., . . . Sokolov, A. A. (2015). Consensus paper: the role of the cerebellum in perceptual processes. *Cerebellum, 14*(2), 197-220. doi:10.1007/s12311-014-0627-7

Bellebaum, C., & Daum, I. (2007). Cerebellar involvement in executive control. *Cerebellum, 6*(3), 184-192. doi:10.1080/14734220601169707

Brissenden, J. A., Levin, E. J., Osher, D. E., Halko, M. A., & Somers, D. C. (2016). Functional Evidence for a Cerebellar Node of the Dorsal Attention Network. *Journal of Neuroscience, 36*(22), 6083-6096. doi:10.1523/JNEUROSCI.0344-16.2016

Buckner, R. L., Krienen, F. M., Castellanos, A., Diaz, J. C., & Yeo, B. T. (2011). The organization of the human cerebellum estimated by intrinsic functional connectivity. *Journal of Neurophysiology, 106*(5), 2322-2345. doi:10.1152/jn.00339.2011

Chica, A. B., Martin-Arevalo, E., Botta, F., & Lupianez, J. (2014). The Spatial Orienting paradigm: how to design and interpret spatial attention experiments. *Neuroscience and Biobehavioral Reviews, 40*, 35-51. doi:10.1016/j.neubiorev.2014.01.002

Clower, D. M., West, R. A., Lynch, J. C., & Strick, P. L. (2001). The inferior parietal lobule is the target of output from the superior colliculus, hippocampus, and cerebellum. *Journal of Neuroscience, 21*(16), 6283-6291.

Dimitrov, M., Grafman, J., Kosseff, P., Wachs, J., Alway, D., Higgins, J., . . . Hallett, M. (1996). Preserved cognitive processes in cerebellar degeneration. *Behavioral Brain Research, 79*(1-2), 131-135.

Dimitrova, A., Zeljko, D., Schwarze, F., Maschke, M., Gerwig, M., Frings, M., . . . Timmann, D. (2006). Probabilistic 3D MRI atlas of the human cerebellar dentate/interposed nuclei. *Neuroimage, 30*(1), 12-25. doi:10.1016/j.neuroimage.2005.09.020

Dum, R. P., & Strick, P. L. (2003). An unfolded map of the cerebellar dentate nucleus and its projections to the cerebral cortex. *Journal of Neurophysiology, 89*(1), 634-639.

Esterman, M., & Rothlein, D. (2019). Models of sustained attention. *Curr Opin Psychol, 29*, 174-180. doi:10.1016/j.copsyc.2019.03.005

Ghajar, J., & Ivry, R. B. (2009). The predictive brain state: asynchrony in disorders of attention? *Neuroscientist, 15*(3), 232-242. doi:10.1177/1073858408326429

Glickstein, M., Strata, P., & Voogd, J. (2009). Cerebellum: history. *Neuroscience, 162*(3), 549-559. doi:S0306-4522(09)00271-1 [pii]

10.1016/j.neuroscience.2009.02.054
Glickstein, M., Sultan, F., & Voogd, J. (2011). Functional localization in the cerebellum. *Cortex, 47*(1), 59-80. doi:S0010-9452(09)00263-9 [pii]
10.1016/j.cortex.2009.09.001
Golla, H., Thier, P., & Haarmeier, T. (2005). Disturbed overt but normal covert shifts of attention in adult cerebellar patients. *Brain, 128*(Pt 7), 1525-1535. doi:awh523 [pii]
10.1093/brain/awh523
Gottwald, B., Mihajlovic, Z., Wilde, B., & Mehdorn, H. M. (2003). Does the cerebellum contribute to specific aspects of attention? *Neuropsychologia, 41*(11), 1452-1460.
Gottwald, B., Wilde, B., Mihajlovic, Z., & Mehdorn, H. M. (2004). Evidence for distinct cognitive deficits after focal cerebellar lesions. *Journal of Neurology, Neurosurgery, and Psychiatry, 75*(11), 1524-1531. doi:10.1136/jnnp.2003.018093
Greenhouse, S. W., & Geisser, S. (1959). On methods in the analysis of profile data. *Psychometrika, 24*, 95-112.
Guell, X., Schmahmann, J. D., Gabrieli, J., & Ghosh, S. S. (2018). Functional gradients of the cerebellum. *Elife, 7*. doi:10.7554/eLife.36652
Haarmeier, T., & Thier, P. (2007). The attentive cerebellum - myth or reality? *Cerebellum, 6*(3), 177-183. doi:777346475 [pii]
10.1080/14734220701286187
Hommel, B., & Akyurek, E. G. (2005). Lag-1 sparing in the attentional blink: benefits and costs of integrating two events into a single episode. *Q J Exp Psychol A, 58*(8), 1415-1433. doi:10.1080/02724980443000647
Husain, M., & Nachev, P. (2007). Space and the parietal cortex. *Trends in Cognitive Science, 11*(1), 30-36.
Husain, M., Shapiro, K., Martin, J., & Kennard, C. (1997). Abnormal temporal dynamics of visual attention in spatial neglect patients. *Nature, 385*(6612), 154-156.
Jonides, J. (1981). Voluntary versus automatic control over the mind's eye's movement. In J. B. Long & A. Baddeley (Eds.), *Attention & Performance IX* (pp. 187-203). New Jersey: Erlbaum.
Jonides, J., & Yantis, S. (1988). Uniqueness of abrupt visual onset in capturing attention. *Perception and Psychophysics, 43*(4), 346-354.
Jurado, M. B., & Rosselli, M. (2007). The elusive nature of executive functions: a review of our current understanding. *Neuropsychol Rev, 17*(3), 213-233. doi:10.1007/s11065-007-9040-z
King, M., Hernandez-Castillo, C., Poldrack, R. A., Ivry, R. B., & Diedrichsen, J. (2018). A multi-domain task battery reveals functional boundaries in the human cerebellum. *BioRxiv*. doi:http://dx.doi.org/10.1101/423509
Klein, R. M. (2000). Inhibition of return. *Trends in Cognitive Science, 4*(4), 138-147.
Leggio, M., & Molinari, M. (2015). Cerebellar sequencing: a trick for predicting the future. *Cerebellum, 14*(1), 35-38. doi:10.1007/s12311-014-0616-x
Loftus, G. R., & Masson, M. E. J. (1994). Using confidence intervals in within-subject designs. *Psychonomic Bulletin & Review, 1*, 476-490.
Manly, T., Robertson, I. H., Galloway, M., & Hawkins, K. (1999). The absent mind: further investigations of sustained attention to response. *Neuropsychologia, 37*(6), 661-670.
Marvel, C. L., & Desmond, J. E. (2010). Functional topography of the cerebellum in verbal working memory. *Neuropsychol Rev, 20*(3), 271-279. doi:10.1007/s11065-010-9137-7

Molinari, M., Chiricozzi, F. R., Clausi, S., Tedesco, A. M., De Lisa, M., & Leggio, M. G. (2008). Cerebellum and detection of sequences, from perception to cognition. *Cerebellum, 7*(4), 611-615. doi:10.1007/s12311-008-0060-x

Mulckhuyse, M., van Zoest, W., & Theeuwes, J. (2008). Capture of the eyes by relevant and irrelevant onsets. *Experimental Brain Research, 186*(2), 225-235. doi:10.1007/s00221-007-1226-3

Muller, H. J., & Rabbitt, P. M. (1989). Reflexive and voluntary orienting of visual attention: time course of activation and resistance to interruption. *Journal of Experimental Psychology, Human Perception and Performance, 15*(2), 315-330.

Peterburs, J., & Desmond, J. E. (2016). The role of the human cerebellum in performance monitoring. *Current Opinion Neurobiology, 40*, 38-44. doi:10.1016/j.conb.2016.06.011

Posner, M. I., Rafal, R. D., Choate, L. S., & Vaughan, J. (1985). Inhibition of return: Neural basis and function. *Cognitive Neuropsychology, 2*, 211-228.

Posner, M. I., Snyder, C. R., & Davidson, B. J. (1980). Attention and the detection of signals. *Journal of Experimental Psychology, 109*(2), 160-174.

Ravizza, S. M., & Ivry, R. B. (2001). Comparison of the basal ganglia and cerebellum in shifting attention. *Journal of Cognitive Neuroscience, 13*(3), 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*(3), 849-860.

Robertson, I. H., Manly, T., Andrade, J., Baddeley, B. T., & Yiend, J. (1997). 'Oops!': performance correlates of everyday attentional failures in traumatic brain injured and normal subjects. *Neuropsychologia, 35*(6), 747-758.

Robinson, F. R., & Fuchs, A. F. (2001). The role of the cerebellum in voluntary eye movements. *Annual Review Neuroscience, 24*, 981-1004. doi:10.1146/annurev.neuro.24.1.981

Rorden, C., Bonilha, L., Fridriksson, J., Bender, B., & Karnath, H. O. (2012). Age-specific CT and MRI templates for spatial normalization. *Neuroimage, 61*(4), 957-965. doi:10.1016/j.neuroimage.2012.03.020

Sacchetti, B., Scelfo, B., & Strata, P. (2009). Cerebellum and emotional behavior. *Neuroscience, 162*(3), 756-762. doi:S0306-4522(09)00127-4 [pii]
10.1016/j.neuroscience.2009.01.064

Schmahmann, J. D., Guell, X., Stoodley, C. J., & Halko, M. A. (2019). The Theory and Neuroscience of Cerebellar Cognition. *Annual Review Neuroscience*. doi:10.1146/annurev-neuro-070918-050258

Schmahmann, J. D., & Sherman, J. C. (1998). The cerebellar cognitive affective syndrome. *Brain, 121 (Pt 4)*, 561-579.

Schreij, D., Owens, C., & Theeuwes, J. (2008). Abrupt onsets capture attention independent of top-down control settings. *Perception and Psychophysics, 70*(2), 208-218.

Schweizer, T. A., Alexander, M. P., Cusimano, M., & Stuss, D. T. (2007). Fast and efficient visuotemporal attention requires the cerebellum. *Neuropsychologia, 45*(13), 3068-3074.

Schweizer, T. A., Levine, B., Rewilak, D., O'Connor, C., Turner, G., Alexander, M. P., . . . Stuss, D. T. (2008). Rehabilitation of executive functioning after focal damage to the cerebellum. *Neurorehabil Neural Repair, 22*(1), 72-77. doi:10.1177/1545968307305303

Shapiro, K., Hillstrom, A. P., & Husain, M. (2002). Control of visuotemporal attention by inferior parietal and superior temporal cortex. *Current Biology, 12*(15), 1320-1325.

Sokolov, A. A., Miall, R. C., & Ivry, R. B. (2017). The Cerebellum: Adaptive Prediction for Movement and Cognition. *Trends in Cognitive Science, 21*(5), 313-332. doi:10.1016/j.tics.2017.02.005

Stoodley, C. J., MacMore, J. P., Makris, N., Sherman, J. C., & Schmahmann, J. D. (2016). Location of lesion determines motor vs. cognitive consequences in patients with cerebellar stroke. *Neuroimage Clin, 12*, 765-775. doi:10.1016/j.nicl.2016.10.013

Stoodley, C. J., & Schmahmann, J. D. (2009a). The cerebellum and language: evidence from patients with cerebellar degeneration. *Brain and Language, 110*(3), 149-153. doi:S0093-934X(09)00084-4 [pii]
10.1016/j.bandl.2009.07.006

Stoodley, C. J., & Schmahmann, J. D. (2009b). Functional topography in the human cerebellum: a meta-analysis of neuroimaging studies. *Neuroimage, 44*(2), 489-501. doi:S1053-8119(08)00972-5 [pii]
10.1016/j.neuroimage.2008.08.039

Stoodley, C. J., & Stein, J. F. (2011). The cerebellum and dyslexia. *Cortex, 47*(1), 101-116. doi:S0010-9452(09)00280-9 [pii]
10.1016/j.cortex.2009.10.005

Strick, P. L., Dum, R. P., & Fiez, J. A. (2009). Cerebellum and nonmotor function. *Annual Review Neuroscience, 32*, 413-434. doi:10.1146/annurev.neuro.31.060407.125606

Striemer, C. L., Cantelmi, D., Cusimano, M. D., Danckert, J. A., & Schweizer, T. A. (2015). Deficits in reflexive covert attention following cerebellar injury. *Front Hum Neurosci, 9*, 428. doi:10.3389/fnhum.2015.00428

Striemer, C. L., Chouinard, P. A., Goodale, M. A., & de Ribaupierre, S. (2015). Overlapping neural circuits for visual attention and eye movements in the human cerebellum. *Neuropsychologia, 69*, 9-21. doi:10.1016/j.neuropsychologia.2015.01.024

Townsend, J., Courchesne, E., Covington, J., Westerfield, M., Harris, N. S., Lyden, P., . . . Press, G. A. (1999). Spatial attention deficits in patients with acquired or developmental cerebellar abnormality. *Journal of Neuroscience, 19*(13), 5632-5643.

Trouillas, P., Takayanagi, T., Hallett, M., Currier, R. D., Subramony, S. H., Wessel, K., . . . Manyam, B. (1997). International Cooperative Ataxia Rating Scale for pharmacological assessment of the cerebellar syndrome. The Ataxia Neuropharmacology Committee of the World Federation of Neurology. *J Neurol Sci, 145*(2), 205-211.

Wang, D., Buckner, R. L., & Liu, H. (2013). Cerebellar asymmetry and its relation to cerebral asymmetry estimated by intrinsic functional connectivity. *Journal of Neurophysiology, 109*(1), 46-57. doi:10.1152/jn.00598.2012

Yamaguchi, S., Tsuchiya, H., & Kobayashi, S. (1998). Visuospatial attention shift and motor responses in cerebellar disorders. *Journal of Cognitive Neuroscience, 10*(1), 95-107.

Supplementary material:

Attentional deficits in cerebellar patients are not the result of damage to the dentate nucleus.

To ensure that the attentional deficits we observed in the reflexive covert attention task and the AB task were not related to damage to the dentate nucleus we re-ran the same analyses again not including the data from the two patients with probable dentate damage (patients 523 and 909; see Table 2 in the main manuscript).

Re-analysis of the data from the reflexive covert attention task revealed identical results. Specifically, there was a significant cue x SOA x group interaction ($F(3,99)=3.09$, $p=.03$; $\eta^2=.09$) such that CES for the cerebellar patients was larger at the 600ms SOA (patients=48ms vs. controls=-3ms; ($t(33)=2.98$, $p<.05$, Tukey corrected). However, there were no significant differences at any of the other SOAs (all p's >.21, uncorrected).

In addition, we also re-analyzed the AB data removing the two patients with dentate damage. This analysis produced identical results with a significantly larger AB effect (i.e., the difference in accuracy for the 1-Target compared to the 2-Target task) in patients without dentate damage (9.46%) compared to controls (2.86%; $t(21)=2.30$, $p=.032$).

Supplementary Table 1: Mean reaction time (RT) data for the reflexive covert attention task for patients (n=11) and controls (n=24) as a function of cue (valid vs. invalid) and stimulus onset asynchrony (50, 100, 300, 600ms).

Patient (lesion):	**Valid 50**	**Valid 100**	**Valid 300**	**Valid 600**	**Invalid 50**	**Invalid 100**	**Invalid 300**	**Invalid 600**
29 (L)	991	925	967	859	998	955	1080	1057
61 (L)	507	516	512	565	584	553	583	575
182 (L)	592	514	607	542	605	613	589	635
309 (L)	587	545	482	490	627	584	481	471
378 (R)	454	463	493	490	497	507	470	480
522 (L)	563	494	501	485	613	591	567	605
523 (R)	606	566	528	494	621	596	595	551
564 (L)	610	593	592	600	677	699	652	699
678 (L)	458	457	453	461	484	490	448	442
867 (B)	626	604	596	656	678	702	663	644
953 (L)	719	676	674	729	730	785	816	750
Patient mean (SD), n=11	**610 (148)**	**577 (132)**	**582 (144)**	**579 (125)**	**647 (137)**	**643 (135)**	**631 (181)**	**628 (172)**
Control mean (SD), n=24	**528 (68)**	**513 (69)**	**519 (73)**	**536 (69)**	**574 (69)**	**571 (77)**	**542 (76)**	**533 (78)**

Supplementary Table 2: Mean reaction time (RT) data for the voluntary covert attention task for patients (n=11) and controls (n=24) as a function of cue (valid vs. invalid) and stimulus onset asynchrony (250, 350, 550ms).

Patient (lesion):	**Valid 250**	**Valid 350**	**Valid 550**	**Invalid 250**	**Invalid 350**	**Invalid 550**
29 (L)	738	851	745	976	828	797
61 (L)	442	437	432	479	488	493
182 (L)	556	543	591	586	615	584
309 (L)	397	352	319	425	382	395
378 (R)	445	440	453	469	469	462
522 (L)	532	566	464	556	620	579
523 (R)	598	530	569	592	590	556
564 (L)	587	579	563	724	640	745
678 (L)	408	361	321	411	412	313
867 (B)	671	645	669	693	682	692
953 (L)	656	627	561	640	779	633
Patient mean (SD), n=11	**548 (115)**	**539 (143)**	**517 (134)**	**596 (163)**	**591 (143)**	**568 (147)**
Control mean (SD), n=24	**515 (81)**	**496 (80)**	**493 (80)**	**553 (86)**	**545 (98)**	**517 (89)**

Supplementary Table 3: Mean percent accuracy on the attentional blink task for patients (n=13) and controls (n=24) as a function of trial type (1-Target vs. 2-Target) and Lag (1-4, 6 or 12).

Patient (lesion):	**1T-L1**	**1T-L2**	**1T-L3**	**1T-L4**	**1T-L6**	**1T-L12**	**2T-L1**	**2T-L2**	**2T-L3**	**2T-L4**	**2T-L6**	**2T-L12**
29 (L)	85	100	50	92	69	60	47	37	50	63	88	85
61 (L)	43	67	75	62	40	83	30	72	63	20	100	76
117 (B)	67	50	100	70	87	85	65	71	69	77	84	63
182 (L)	92	100	100	81	75	67	55	75	65	77	92	89
309 (L)	67	43	67	85	100	60	47	38	73	70	100	80
378 (R)	71	90	20	88	100	79	68	60	63	80	65	94
523 (R)	44	63	50	56	78	33	44	44	69	56	65	38
564 (L)	82	75	64	57	89	89	46	48	39	78	76	84
670 (B)	86	73	86	77	100	83	61	65	63	83	84	88
678 (L)	50	100	100	71	92	88	79	72	57	76	100	92
867 (B)	91	88	71	89	100	91	53	54	47	54	80	88
909 (B)	70	75	91	65	100	100	50	77	58	80	74	75
953 (L)	88	62	100	71	86	83	52	78	33	80	78	75
Patient mean (SD), n=13	**72 (17)**	**76 (19)**	**75 (25)**	**74 (12)**	**86 (17)**	**77 (18)**	**54 (12)**	**61 (15)**	**58 (12)**	**69 (18)**	**84 (12)**	**79 (15)**
Control mean (SD), n=24	**76 (16)**	**80 (17)**	**80 (15)**	**80 (13)**	**82 (17)**	**80 (16)**	**70 (18)**	**65 (16)**	**72 (15)**	**81 (15)**	**88 (9)**	**87 (11)**

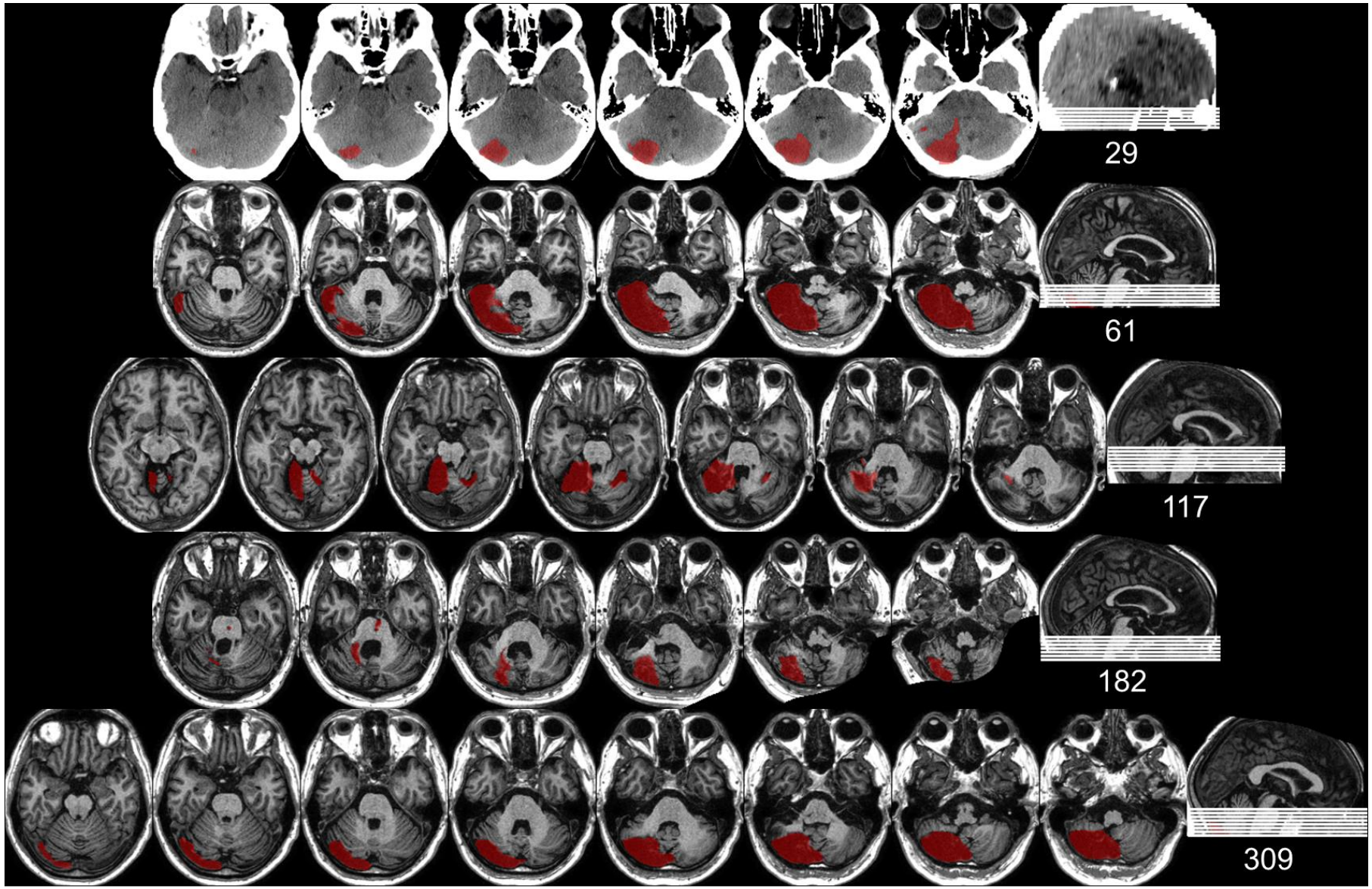
Supplementary Table 4: Mean percentage of errors and misses and mean reaction times (RT) for errors, hits, as well as the three trials preceding and following an error for patients (n=13) and controls (n=24) for the sustained attention to response task (SART).

Patient (lesion):	**% errors**	**error RT**	**% misses**	**Hits RT**	**Errors: 3 Prior RT**	**Errors: 3 Post RT**
29 (L)	48	567	39.5	696	691	878
61 (L)	36	561	3.5	475	525	497
117 (B)	56	555	4.5	673	784	827
182 (L)	36	319	0.5	415	418	462
309 (L)	92	294	5	301	291	280
378 (R)	68	324	0.5	307	302	339
523 (R)	12	469	1	603	543	665
564 (L)	24	473	7	556	505	638
670 (B)	28	391	2.5	590	435	632
678 (L)	52	408	1	457	458	558
867 (B)	12	424	1	564	509	720
909 (B)	20	514	0.5	582	503	664
953 (L)	12	424	0	465	400	536
Patient mean (SD), n=13	**38 (24)**	**440 (94)**	**5.1 (11)**	**514 (125)**	**490 (136)**	**592 (173)**
Control mean (SD), n=24	**31 (17)**	**445 (103)**	**3 (5.6)**	**481 (78)**	**449 (67)**	**523 (119)**

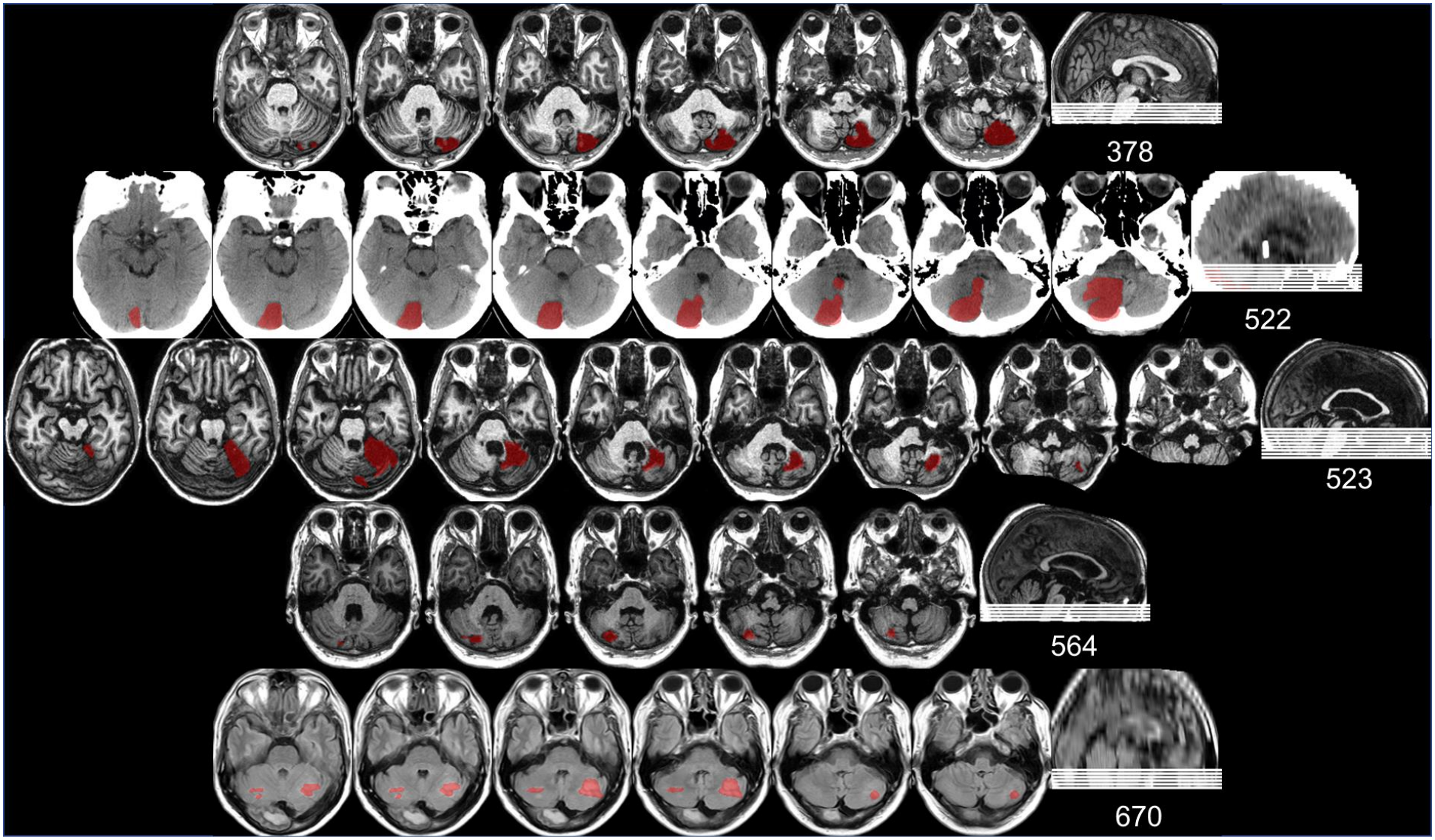
Supplementary Table 5. Talairach coordinates associated with regions of maximum lesion overlap for cerebellar patients who completed the covert spatial attention (n=11) and non-spatial attention (n=13) tasks.

Covert spatial attention (n=11):	**Talairach coordinates:**		
	X	**Y**	**Z**
Left cerebellum, posterior lobe, uvula (lobule IX)	-18	-82	-25
Left cerebellum, posterior lobe, tuber (lobule VII)	-29	-78	-28
Left cerebellum, posterior lobe, inferior semi-lunar lobule (Crus II)	-20	-77	-36
Non-spatial attention (AB + SART; n=13)			
Left cerebellum, posterior lobe, tuber (lobule VII)	-28	-78	-28
Left cerebellum, posterior lobe, pyramis (lobule VIII)	-27	-77	-33
Left cerebellum, posterior lobe, inferior semi-lunar lobule (Crus II)	-23	-73	-49

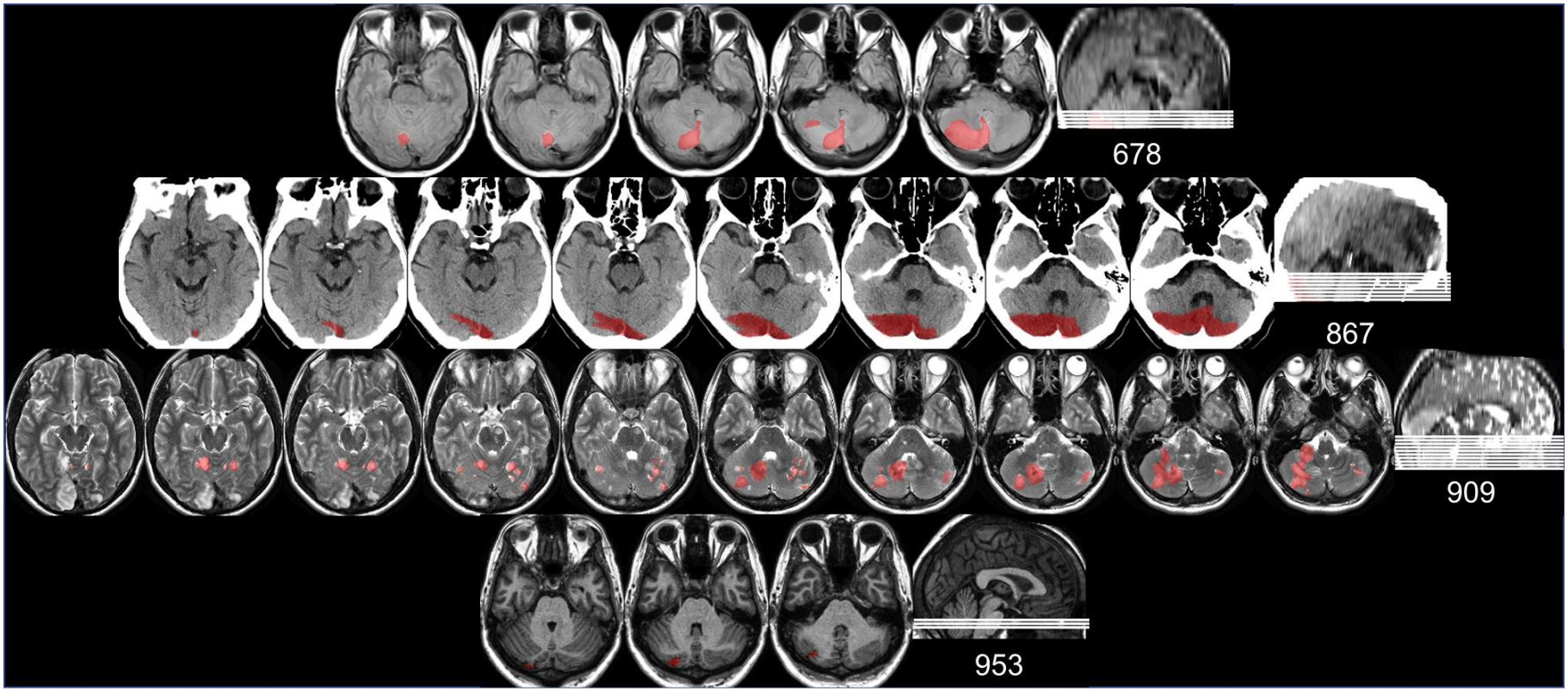
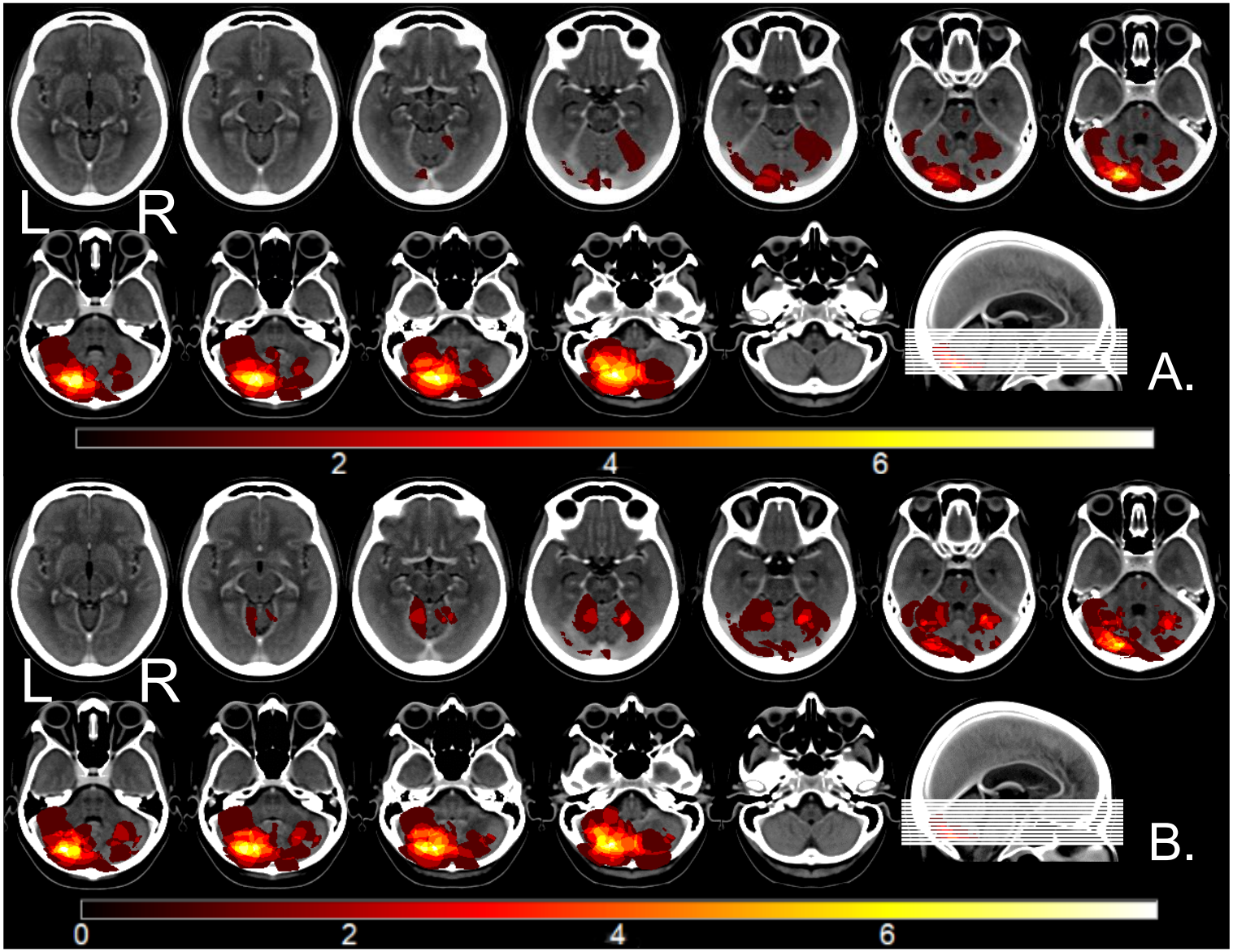
Supplementary Figure 1 Part 1. Individual lesion maps for each of the patients (patient number located to the right). See Table 2 in the main manuscript for clinical details for each patient. Images are presented in neurological convention.



Supplementary Figure 1 Part 2. Individual lesion maps for each of the patients (patient number located to the right). See Table 2 in the main manuscript for clinical details for each patient.



Supplementary Figure 1 Part 3. Individual lesion maps for each of the patients (patient number located to the right). See Table 2 in the main manuscript for clinical details for each patient.



Supplementary Figure 2. Group Lesion maps for A) COVAT patients only (n=11); and B) attentional blink patients only (n=13).