Cerebellar vermis plays a causal role in visual motion discrimination

Zaira Cattaneoa,b,*, Chiara Renzib, Stefano Casalib,c, Juha Silvantom,d,e, Tomaso Vecchib,c, Costanza Papagnoa and Egidio D’Angelob,c

a Department of Psychology, University of Milano-Bicocca, Milano, Italy
b Brain Connectivity Center, National Neurological Institute C. Mondino, Pavia, Italy
c Department of Brain and Behavioral Sciences, University of Pavia, Pavia, Italy
d Department of Psychology, Faculty of Science and Technology, University of Westminster, UK
e Brain Research Unit, O.V. Lounasmaa Laboratory, School of Science, Aalto University, Espoo, Finland

Abstract

Cerebellar patients have been found to show deficits in visual motion discrimination, suggesting that the cerebellum may play a role in visual sensory processing beyond mediating motor control. Here we show that triple-pulse online transcranial magnetic stimulation (TMS) over cerebellar vermis but not over the cerebellar hemispheres significantly impaired motion discrimination. Critically, the interference caused by vermis TMS on motion discrimination did not depend on an indirect effect of TMS over nearby visual areas, as demonstrated by a control experiment in which TMS over V1 but not over cerebellar vermis significantly impaired orientation discrimination. These findings demonstrate the causal role of the cerebellar vermis in visual motion processing in neurologically normal participants.

Keywords:
Cerebellum
Visual
Motion detection
Orientation discrimination
TMS

1. Introduction

The cerebellum has been suggested to play a critical role in sensory processing, beyond being involved in motor control (Bower, 1997; D’Angelo & Casali, 2012; Manto et al., 2012). Specifically, psychophysical studies with cerebellar patients have demonstrated the functional significance of this region in motion perception independent from actual movements (Baumann & Mattingley, 2010; Jokisch, Troje, Koch, Schwarz, & Daum, 2005; Nawrot & Rizzo, 1995, 1998; Thier, Haarmeier, Treue, & Barash, 1999). Both in acute and chronic post-ictus phases, cerebellar patients with lesions in midline structures have deficits in motion discrimination, which was not the case when lesions were restricted to the cerebellar hemispheres (Nawrot & Rizzo, 1995, 1998). The performance deficits, which occurred even though cortical processing was intact, were neither due to aberrant eye movements nor an indirect consequence of motor deficits (Ivry & Diener, 1991;
Nawrot & Rizzo, 1995, 1998; Thier et al., 1999). However, lateral parts of the cerebellum have also been associated with deficits in motion discrimination (Jokisch et al., 2005); thus, the localization of cerebellar areas controlling motion discrimination remains unclear.

Neuroimaging evidence on the role of the cerebellum in motion discrimination is also somewhat conflicting. In positron emission tomography (PET) studies, Dupont, Orban, De Bruyn, Verbruggen, and Mortelmans (1994) found vermal activation in response to a moving dot pattern, and Barbur, Watson, Frackowiak, and Zeki (1993) reported such activity in a blindsight patient with a unilateral lesion in V1. However, using functional magnetic resonance imaging (fMRI) Baumann and Mattingley (2010) observed a complex pattern of activation in the cerebellar hemispheres, which was correlated with both auditory and visual motion signal strength.

Here we attempted to resolve this controversy by the use of brain stimulation in neurologically healthy volunteers in three different experiments. Brain stimulation can shed light on the causal role of the targeted brain regions in mediating specific perceptual functions, thus overcoming the correlational nature of neuroimaging and the poor focality of brain lesions. In the first two experiments online transcranial magnetic stimulation (TMS) was applied either over the cerebellar vermis (Experiment 1) or over the left and right cerebellar hemispheres (Experiment 2), while participants performed a visual motion discrimination task. In order to rule out effects due to nonspecific effects of TMS (e.g., tapping sensation, auditory distraction) or to spread of stimulation from the cerebellar vermis to the adjacent visual cortex, in Experiment 3 TMS was applied during a visual orientation discrimination task, which is known to causally involve the early visual cortex but not cerebellum (e.g., Neary, Anand, & Hotson, 2005 for previous TMS evidence using orientation discrimination).

2. Experiments 1 and 2: motion discrimination

2.1. Methods

2.1.1. Participants

Twelve healthy volunteers (10 F, mean age: 21.58 ys, SD: 1.0, range: 20–23) took part in Experiment 1 and twelve healthy volunteers (11 F, mean age: 22.17 ys, SD: 2.2, range: 20–27), none of whom had participated in Experiment 1, took part in Experiment 2. All participants were right-handed (Oldfield, 1971) and had normal or corrected to normal vision. Prior to the experiment, each participant filled in a questionnaire (translated and adapted from Rossi, Hallett, Rossini, & Pascual-Leone, 2011) to evaluate compatibility with TMS. None of the volunteers had a history of neurological disorders or brain trauma or family history of epilepsy. Written informed consent was obtained from all participants before the experiment. The protocol was approved by the local ethical committee and participants were treated in accordance with the Declaration of Helsinki.

2.1.2. Visual stimuli

All stimuli were presented centrally on a 17” TFT-LCD computer monitor (screen resolution: 1440*900 pixels; refresh rate: 75 Hz). Stimuli consisted of 100 white dots (one pixel each), placed at random positions within an imaginary square that subtended 4.3° × 4.3° of visual angle. The coherent dots were moving either to the right or left within the virtual square on a black background at a speed of 1 pixel per frame (at 2.15 deg/sec); noise dots changed direction randomly at every screen refresh. Each trial began with a fixation cross appearing in the middle of the screen for an interval comprised between 2600 and 2800 msec, followed by a blank screen for 500 msec, after which the stimulus appeared. Stimulus duration was between 40 and 67 msec, depending on participant’s ability (see thresholding procedure below). After response, the next trial started. An example of experimental trial is shown in Fig. 1. Participants were required to report whether the visual stimulus moved to the left or right by left/right key presses using their right index and middle finger. Response speed was stressed in addition to accuracy.

2.1.3. Thresholding

Prior to the experiment, each participant underwent a thresholding procedure to determine the ratio of coherent moving dots and the number of frames (frame duration: 13 msec) necessary to obtain a stable performance around 75% accuracy. This was achieved by running a block in which motion coherence (i.e., the amount of dots moving coherently either to the left or right) ranged from 40% to 90% in steps of 10% with six levels. In this block, the motion stimulus contained five frames (with 20 trials per level with the method of

![Fig. 1](http://dx.doi.org/10.1016/j.cortex.2014.01.012)
constant stimuli, i.e., coherence levels were randomly interleaved). Hence, another block was run using the three consecutive levels of noise (20 trials each) that together resulted in a mean level of accuracy around 75% (if participant's accuracy was higher than 75% at the maximum levels of noise in the first block, the block was repeated with a lower number of displayed frames to make the discrimination more difficult). From this block, we selected the level of noise whose mean accuracy was closer to 75%. Overall, the percentage of noise dots used in the experiment ranged from 40% to 85%, the number of frames ranged from 3 (corresponding to a stimulus duration of 40 msec) to 5 (corresponding to a stimulus duration of 67 msec).

2.1.4. TMS
TMS was administered by means of a Magstim Rapid™ machine (Magstim Co Ltd, Whitland, UK) with a 70 mm butterfly coil. At the beginning of each session the resting motor threshold (rMT) was measured from the first dorsal interosseous (FDI) muscle of the right hand. This intensity was defined as the minimum stimulation intensity that produced motor evoked potentials (MEPs) \( \geq 50 \mu V \) peak to peak in at least 5 out of 10 trials (Rossini et al., 1994). For each participant, the intensity of TMS stimulation was equal to 100% of the rMT (mean intensity: 52.0%, SD: 6.5%).

In Experiment 1, there were two stimulation sites: V1 and cerebellar vermis. The cerebellum was stimulated as in previous studies at a point 1 cm inferior to the inion on a line joining the inion to the nasion (Lee et al., 2007; Schutter, De Weijer, Meuwese, Morgan, & Van Honk, 2008; Théoret, Haque, & Pascual-Leone, 2001) with the handle pointing superiorly. V1 was localized as the point lying 1.5 cm superior to the inion on the midline joining the inion to the nasion (Campana, Cowey, & Walsh, 2006; Heinen, Jolij, & Lamme, 2005; Pascual-Leone & Walsh, 2001). The accuracy of this localization method was further verified by using a neuronavigation system (Nexstim, Helsinki, Finland) in three subjects (that did not take part in the experiment) for whom structural MRIs were available. On the structural MRIs of these three individuals, the scalp coordinate 1 cm below the inion indeed corresponded to the cerebellar vermis, and the scalp coordinate 1.5 cm above the inion corresponded indeed to V1 (see Fig. 2). A No TMS “baseline” condition and two sham TMS conditions were also included. In sham TMS conditions, TMS was delivered either over V1 or vermis but the coil was flipped 90 degrees leftward or rightward (counterbalanced within participants), in line with previous studies (Fried, Elkin-Frankston, Rushmore, Hilgetag, & Valero-Cabre, 2011; Lisanby, Gutman, Luber, Schroeder, & Sackeim, 2001). In the main experiment, on each trial, we applied triple-pulse TMS (20 Hz; i.e., pulse gap of 50 msec) at the onset of the motion stimulus. Before starting the experiment, triple-pulse TMS (20 Hz) at 100% of the rMT was delivered over V1 ten times (ISI: 3000 msec) to assess for phosphenes perception with eyes open: none of the participants reported phosphenes perception.

In Experiment 2, the stimulation parameters were adapted from Experiment 1. TMS was applied to the right cerebellar hemisphere (right Cb) or the left cerebellar hemisphere (left Cb); in addition, a No TMS “baseline” condition was conducted. In line with previous studies, the cerebellar hemispheres were localized at a point 1 cm inferior to the inion on a line joining the inion to the nasion and 3 cm laterally either to the left or to the right (Arasanz, Staines, & Schweizer, 2012; Théoret et al., 2001), with the handle pointing superiorly. As for Experiment 1, the precision of this localization method

![Fig. 2](image-url) - Localization of V1 and vermis using neuronavigation. The accuracy of localization method based on scalp coordinates confirmed by neuronavigation. In the upper panel, the pointer is indicating the scalp position 1.5 cm above the inion, the putative location of V1/V2. The lower panel shows the point localized 1 cm below the inion, the putative cerebellar vermis. For both V1/V2 and vermis, there is a good correspondence between the scalp coordinate and the anatomy.

Please cite this article in press as: Cattaneo, Z., et al., Cerebellar vermis plays a causal role in visual motion discrimination, Cortex (2014), http://dx.doi.org/10.1016/j.cortex.2014.01.012
was confirmed by using neuronavigation on the structural MRI of three individuals (that did not take part in the experiment). A sham TMS condition was also included: in half of the sham trials (one block) the coil was held over the right Cb, in the other half over the left Cb. For sham stimulation, the coil was flipped 90 degrees leftward or rightward (counterbalanced within participants). Mean intensity of stimulation was 51.9% (SD: 5.8%), corresponding to the participants’ mean 100% rMT. None of the participants reported phosphenes perception during the experiment.

2.1.5. Procedure
Participants sat comfortably at a distance of 57 cm from the screen with their head resting on an adjustable chin-rest. At the beginning of the first session, we localized each participant’s V1 ad vermis (Experiment 1) and cerebellar hemispheres (Experiment 2). In both experiments, initially, participants were given practice blocks with the motion discrimination task. They were then thresholded to determine a level of accuracy around 75% (see above). Once the visual motion parameters were decided, each participant underwent two blocks of stimulation for each TMS condition, for a total of ten blocks in Experiment 1 and eight blocks in Experiment 2. The first half of the experiment contained one block of each of the TMS conditions; the order of these was randomly assigned. The order of the second half of the experiment was the reverse of the first half. Each block consisted of 60 trials (30 with leftward and 30 with rightward motion) presented in random order. The software E-prime 2.0 (Psychology Software Tools, Pittsburgh, PA) was used for stimuli presentation, data collection and TMS triggering. Each experiment took approximately 2 h.

2.2. Results
Mean accuracy and mean reaction times (RT) for correct responses were computed for each TMS condition for each participant. Trials in which individual response latencies were beyond 3 standard deviations with respect to each participant’s mean performance in each experimental block were excluded from the analyses. Data were submitted to repeated-measures ANOVAs (significant threshold set at $p = .05$, Bonferroni-Holm correction applied to post-hoc comparisons) with TMS condition as within-subjects variable.

2.2.1. The effect of cerebellar vermis and V1 stimulation
In a first group of 12 subjects (Experiment 1), TMS was delivered to the posterior cerebellum. A total of .93% of the trials were excluded as outliers (see above for exclusion criteria). Planned pairwise comparisons indicated that the two sham conditions were not significantly different from each other neither in terms of accuracy, $t(11) = 1.24, p = .24$, nor in terms of response latencies, $t(11) = .19, p = .85$. In the following analyses, the mean of the two sham conditions was therefore considered as a unique control sham TMS condition.

Fig. 3a shows mean participants’ accuracy in each of the different TMS conditions. A repeated-measures ANOVA with TMS as within-subjects variable (No TMS, Sham, V1, and vermis) on mean accuracy scores revealed a significant effect of TMS, $F(3,33) = 9.38, p < .001, \eta^2_p = .46$. Pairwise comparisons (Bonferroni–Holm correction applied) indicated that TMS over V1 significantly reduced accuracy compared to both the Sham TMS condition, $t(11) = 4.64, p = .005$, and the baseline No TMS condition, $t(11) = 4.18, p = .008$. Similarly, TMS over vermis significantly reduced accuracy compared to both the Sham TMS condition, $t(11) = 2.99, p = .036$, and the No TMS condition, $t(11) = 2.72, p = .04$. The Sham TMS condition and the No TMS conditions did not significantly differ from each other, $t(11) = 1.04, p = .32$.

Fig. 3b shows mean RT for correct responses in the no TMS, Sham, V1 and vermis stimulation conditions. The ANOVA with TMS condition (No TMS, Sham TMS, V1, vermis) as within-subjects variable showed no significant effect of TMS, $F(3,33) = .580, p = .81, \eta^2_p = .01$.

Overall, these results indicate a significant role of the cerebellar vermis in controlling motion detection.

2.2.2. The effect of cerebellar hemispheres stimulation
In a second group of 12 subjects (Experiment 2), TMS was delivered to the lateral cerebellum. A total of 1.4% of the trials were overall excluded as outliers. Fig. 4a depicts mean participants’ accuracy in each of the different TMS conditions. A repeated-measures ANOVA with TMS as within-subjects variable (No TMS, Sham, left Cb, right Cb) on mean accuracy
scores showed no significant effect of TMS, F(3,33) = .95, p = .95, \( \eta^2_p = .01 \).

Fig. 4b depicts mean RT for correct responses in the different experimental conditions. The ANOVA with TMS condition (No TMS, Sham TMS, left Cb, right Cb) as within-subjects variable revealed no significant effect of TMS, F(3,33) = 1.07, p = .38, \( \eta^2_p = .09 \).

Overall, results of Experiment 2 indicate that the cerebellar hemispheres are not causally involved in processing visual motion detection.

3. Experiment 3

Findings of Experiment 1 point to a causal role of the cerebellar vermis in mediating visual motion discrimination. However, the detrimental effects of vermis TMS on performance observed in Experiment 1 may have depended on nonspecific effects of vermis TMS (e.g., tapping sensation, auditory distraction). Moreover, one may argue that the effects we reported in Experiment 1 depended on spread of stimulation effects from the cerebellar vermis to the adjacent visual cortex. In order to rule out these possibilities, in Experiment 3 TMS was applied during a visual orientation discrimination task which is known to causally involve the early visual cortex but not cerebellum (e.g., Neary et al., 2005).

In this experiment, participants had to decide about the orientation of a series of Gabor patches (typically used to assess orientation sensitivity in V1, see Ringach, 2004), while TMS was applied as in Experiment 1.

3.1. Method

3.1.1. Participants

Sixteen right-handed (Oldfield, 1971) students of the University of Pavia (4 males, mean age: 22.9 years, SD: 3.5, range: 20–35 years) took part in the experiment. All had normal or corrected to normal vision. Written informed consent was obtained from all participants before the experiment (same inclusion criteria were used as in Experiments 1 and 2). The protocol was approved by the local ethical committee and participants were treated in accordance with the Declaration of Helsinki.

3.1.2. Visual stimuli and thresholding

The stimuli were sinusoidal luminance-modulated gratings (with a diameter of 5° of visual angle; generated with MATLAB, The MathWorks, Natick, MA), presented foveally on a gray background from a viewing distance of 57 cm and oriented 1, 2, 3 or 4 degrees to the left or to the right from the vertical meridian. The spatial frequency of the gratings was 1.44 cycles/° and Michelson contrast of .1. Each trial started with a central fixation cross with a duration changing randomly between 500 and 600 msec, followed by the Gabor patch that was presented for 13 msec. The Gabor patch was immediately replaced by a stimulus mask consisting of a black circle (same diameter as the Gabor patch) that was presented for 100 msec. Participants’ response was followed by the presentation of a 2500 msec grey screen background. An example of experimental trial is shown in Fig. 5.

3.1.3. TMS

The stimulation parameters were the same as in Experiments 1 and 2. TMS was applied over the same cortical sites as in Experiment 1, for both real and sham TMS. Three pulses of TMS (20 Hz) were delivered at the 100% of the rMT (see Methods of Experiments 1 and 2 for rMT assessment; mean intensity: 54.0%, SD: 6.1%) over the primary visual area (V1) or over the cerebellar vermis at the offset of the Gabor patch. Before starting the experiment, triple-pulse TMS (20 Hz) at 100% of the rMT was delivered over V1 ten times (ISI: minimum 3000 msec) to assess for phosphene perception with eyes open: none of the participants reported phosphene perception.

3.1.4. Procedure

Participants sat comfortably at a distance of 57 cm from a 17” TFT-LCD computer monitor (screen resolution: 800*600 pixels; refresh rate: 75 Hz) with their heads resting on an adjustable chin-rest. Participants were asked to judge on each trial whether the grating was oriented rightward or leftward compared to the vertical meridian, by rightward/leftward key presses using their right middle and index finger. Response speed was stressed in addition to accuracy. Initially, participants were given practice blocks with the orientation discrimination task. Hence, each participant underwent eight blocks of stimulation (two for each TMS condition). The first half of the experiment (i.e., the first four blocks) contained one block of each of the four TMS conditions; the order of these was randomly assigned. The order of the second half of the
experiment was the reverse of the first half. Each block consisted of 40 trials (5 leftward and 5 rightward orientated Gabor for each of the four possible orientation degrees) presented in random order, for a total of 80 trials for each stimulation condition. The software E-prime 2.0 (Psychology Software Tools, Pittsburgh, PA) was used for stimuli presentation, data collection and TMS triggering. The whole experiment took approximately 90 min.

4. Results

Mean accuracy and mean RT for correct responses were computed for each TMS condition for each participant. Trials in which individual response latencies were beyond 3 standard deviations with respect to each participant’s mean performance in each experimental block were excluded from the analyses. A total of 1.4% of the trials were excluded as outliers (see above for exclusion criteria).

Planned pairwise comparisons indicated that the two sham conditions were not significantly different from each other neither in terms of accuracy, $t(15) = .32, p = .76,$ nor in terms of response latencies (mean RT $= 416$ msec and $402$ msec for sham V1 and for sham vermis, respectively), $t(15) = 1.74, p = .10.$ In the following analyses, the mean of the two sham conditions was therefore considered as a unique control sham TMS condition.

Fig. 6a shows mean participants’ accuracy in each of the different TMS conditions. A repeated-measures ANOVA with TMS as within-subjects variable (Sham, V1, and vermis) on mean accuracy scores revealed a significant effect of TMS, $F(2,30) = 5.95, p = .007, \eta_p^2 = .28.$

Pairwise comparisons (Bonferroni–Holm correction applied) indicated that TMS over V1 significantly reduced
accuracy compared to both the Sham TMS condition, t(15) = 2.90, p = .033, and the vermis condition, t(15) = 2.56, p = .044. Accuracy did not differ when TMS was applied over vermis compared to the Sham TMS condition, t(15) < 1, p = .93.

Fig. 6b shows mean RT for correct responses in the Sham, V1 and vermis stimulation conditions. The ANOVA with TMS condition (Sham, V1, vermis) as within-subjects variable showed no significant effect of TMS, F(2,30) = .25, p = .78, $\eta^2_p = .02$ on participants’ response latencies.

Overall, results of Experiment 3 indicate that the cerebellar vermis is not involved in orientation discrimination.

5. Discussion

Online TMS applied over the cerebellar vermis but not over the cerebellar hemispheres significantly impaired participants’ performance in a visual motion discrimination task (Experiments 1 and 2). The detrimental effects of vermis TMS on motion discrimination were similar to those observed when V1 was stimulated. In Experiment 3, TMS over vermis did not affect participants’ accuracy in an orientation discrimination task, whereas TMS over V1 did induce an impairment. The results of Experiment 3 thus demonstrate that the effect of vermis TMS on motion discrimination cannot be explained in terms of nonspecific effects of TMS (such as discomfort), as no effects were observed on the detection of another type of visual stimuli (gratings). Furthermore, they also rule out the possibility that the effect of vermis TMS in Experiment 1 was due to the spread of the TMS effect to V1, rather than due to a role of vermis in motion processing. Our data shed light on previous neuroimaging and neurophysiological evidence that have reported cerebellar activations during visual motion processing but have been inconsistent regarding whether only midline cerebellar structures or also cerebellar hemispheres were involved (Barbur et al., 1993; Baumann & Mattingley, 2010; Dupont et al., 1994; Ivy & Diener, 1991; Jokisch et al., 2005; Nawrot & Rizzo, 1995, 1998; Thier et al., 1999).

Previous patient and nonhuman primate evidence have highlighted the role of midline cerebellar structures in mediating motion discrimination (Ignashchenkova et al., 2009; Nawrot & Rizzo, 1995, 1998): our data extend this evidence by showing that direct stimulation of the cerebellar vermis region affects an ongoing visual process in healthy humans.

TMS also affected motion discrimination when delivered over V1 at the onset of the visual stimulus, according to previous evidence (Laycock, Crewther, Fitzgerald, & Crewther, 2007; Nawrot & Rizzo, 1995, 1998; Silvanto, Cowey, Lavié, & Walsh, 2005). These findings are in line with the tenet that motion processing is mediated by a feedforward/feedback loop between striate and extrastriate cortex ( Bullier, Hupé, James, & Girard, 2001; Galletti & Fattori, 2003). In our study, TMS applied over vermis at the onset of the motion stimulus also disrupted motion discrimination. How does the cerebellum take part to the network subtending motion discrimination? Using MEG, Handel et al. (Händel, Thier, & Haarmeier, 2009) recorded cortical responses in a group of patients with cerebellar lesions while viewing motion stimuli of varied coherence. Interestingly, the impairment in global motion discrimination shown by the patients was paralleled by qualitative differences in responses recorded from parieto-temporal cortex, in the form of a reduced responsiveness to coherent visual motion and a loss of bilateral representations of motion coherence. These data suggest that visual motion processing in cerebral cortex critically depends on an intact cerebellum (Händel et al., 2009). In line with this, a number of recent fMRI studies have reported functional connectivity between the cerebellar vermis and the visual network (Kellerman et al., 2012; O’Reilly, Beckmann, Tomassini, Ramnani, & Johansen-Berg, 2010; Sang et al., 2012). For instance, Kellermann et al. (2012) showed modulation of cerebellar inputs over a large portion of the dorsal visual stream, including V1, V5 and the posterior parietal cortex (PPC), in an attention-demanding task requiring participants to attend to slight changes in the velocity of vertical bars. Psychophysiological analyses revealed enhanced connectivity between cerebellum and V5 during the task, whereas connections between V5 and PPC were suppressed. Kellermann et al. (2012) argued that their findings suggest a modulatory role of cerebellum in predictive coding (Kellermann et al., 2012; Schlierf, Ivry, & Diedrichsen, 2012). In this context, the cerebellar activation would reflect top-down predictions about motion characteristics, whereas the PPC suppression would reflect diminished sensitivity to bottom up information.

In line with this, we may speculate that the impairment in motion detection following TMS over the vermis depended on TMS interfering with those sensory predictions mechanisms handled by this cerebellar region that are needed for oculo-motor control. The role of cerebellar vermis in oculo-motor control is well established. Indeed, TMS over the posterior cerebellum delivered in close relationship with movement interferes with smooth pursuit (Ohtsuka & Enoki, 1998), visually guided saccades (Hashimoto & Ohtsuka, 1995) and with the synkinesis of eye and head coordination (Nagel & Zangemeister, 2003). Moreover, theta-burst stimulation (TBS) over the posterior cerebellum has long-term effects on saccadic adaptation (jenkinson & Miall, 2010), internal representations of motor information related to eye movements (Colnaghi et al., 2011), and classical eye-blink conditioning (Hofland et al., 2012). Following from this, one may argue though that the detrimental effects of vermis TMS we reported depended on vermis TMS directly interfering with eye movements control during motion discrimination (e.g., Colnaghi et al., 2011; Ohtsuka & Enoki, 1998). However, it is important to consider that the average latency of a saccade has been reported to be around 200 msec (Carpenter, 1988), beyond the duration of our motion stimuli (that lasted between 40 and 67 msec). Although microsaccades may have indeed been affected by cerebellar TMS, the main function of microsaccades is to restoring faded vision during fixation, for both foveal and peripheral targets (see Martinez-Conde, Otero-Millan, & Macknik, 2013, for a review). Indeed, fading only occurs with longer lasting stimuli compared to ours, and microsaccades usually occur at a frequency of around 1 Hz (Martinez-Conde et al., 2013), whereas our motion stimuli last less than 1 sec. In light of the above, it is unlikely that the effects we reported merely reflected TMS effects on eye movements control. Moreover, neuroimaging, primate neurophysiology and TMS evidence converges in indicating that the cerebellar hemispheres are also involved in oculo-motor control (e.g., Haarmeier & Kammer, 2010; Liem, Frens,
Smits, & van der Geest, 2013; Nitschke, Arp, Stavrou, Erdmann, & Heide, 2005; Ohki et al., 2008; Panouille et al., 2012. In light of this evidence, if the decrease in performance we observed depended on vermis TMS affecting eye movements, similar detrimental effects should have been observed following TMS over the cerebellar hemispheres, whereas this was not the case.

Several findings point to a role of the cerebellar hemispheres in visual discrimination (Claeys et al., 2003; Orban, Dupont, Vogels, Bormans, & Mortelmans, 1997) and in sensory prediction mechanisms related to arm and eyes’ motion (Cerminara, Apps, & Marple-Horvat, 2009; Liu et al., 2000). Also, neuroimaging data suggest that the lateral cerebellum may be more driven by the requirements for (tactile) sensory processing than by motor coordination per se (Gao et al., 1996; Liu et al., 2000). In this perspective, the lack of effect of TMS over the cerebellar hemispheres is not completely clear. Our data suggest that the vermis plays a more prominent role in visual sensory processing (beyond sensory prediction mechanisms or visual attention) compared to the cerebellar hemispheres, although further investigation is needed to clarify this issue.

It might be argued that vermis TMS impaired motion perception not because this region contributes to motion discrimination, but because online vermis TMS modulated activity in striate cortex due to spread of activation (see also Renzi, Vecchi, D’Angelo, Silvanto, & Cattaneo, 2014). However, this explanation is inconsistent with the results of Experiment 3, in which stimulation of the cerebellar vermis did not impair orientation discrimination whereas TMS over V1 did. Although the impact of vermis TMS may spread to anatomically connected regions (this likely depending on intensity and frequency of stimulation), such a spread of activation was not sufficient to modulate behavior in Experiment 3, supporting the view that the results in Experiment 1 do reflect the role of vermis in motion encoding. Moreover, the TMS effects over vermis found in Experiment 1 are unlikely to result from an inaccurate localization procedure, since its accuracy was confirmed by neuronavigation in individuals for whom structural MRI was available. Finally, any discomfort induced by TMS is unlikely to have driven these effects, since cerebellar vermis and cerebellar hemispheres stimulation produced similar level of discomfort and because the effects of discomfort should have also been present in Experiment 3.

In conclusion, the effect of TMS on visual motion reported here points to a causal role of the cerebellar vermis in visual motion discrimination. In a broader perspective, the involvement of the cerebellar vermis in visual motion discrimination could be for motor sake and be related to the complex arrangements that the cerebellum imposes to various brain structures in order to plan, regulate and coordinate learned movements (Bower, 1997; Manto et al., 2012).


REFERENCES


Please cite this article in press as: Cattaneo, Z., et al., Cerebellar vermis plays a causal role in visual motion discrimination, Cortex (2014), http://dx.doi.org/10.1016/j.cortex.2014.01.012


