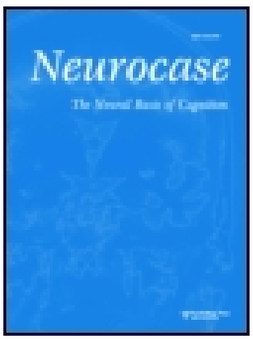

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Cerebellar damage affects the inference of human motion

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ABSTRACT

The present study aims at the cerebellum's role in prediction mechanisms triggered by action observation. Five cerebellar patients and six age-paired control subjects were asked to estimate the occluded end point position of the shoulder's trajectories in Sit-to-Stand (STS) or Back-to-Sit (BTS) conditions, following or not biological rules. Contrarily to the control group, the prediction accuracy of the end point position in cerebellar patients did not depend on biological rules. Interestingly, both groups presented similar results when estimating the vanishing position of the target. Taken together, these results suggest that cerebellar damage affectsthe capacity of predicting upcoming actions by observation.

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KEYWORDS
Motion inference; cerebellar damage; internal models of action; kinematic rules; motor resonance

Introduction

The cerebellum is classically referred to as a structure involved in the production and learning of actions (Glickstein et al., 2009; Ito, 2011), acting as a sensorimotor predictor (Hull, 2020). Current theories of motor control (Kawato et al., 1987; Miall et al., 1993; Shadmehr & Krakauer, 2008; Wolpert & Miall, 1996) propose that the cerebellum would contribute to the computation of inverse and/or forward internal models of actions through complex cortical-subcortical loops (Wolpert et al., 1998). Accordingly, the cerebellum, within these complex loops, would participate in the elaboration of the motor command necessary to respond at a desired sensory state (i.e., inverse internal model) as well as predict the sensory consequences of such motor command (i.e., forward internal model). Indeed, studies in cerebellar patients have evidenced that predictive aspects of motor control are altered whereas their reactive control is preserved (Morton & Bastian, 2006). Such findings reinforce the proposal that the cerebellum acts as a comparator between the prediction of the sensory consequences of one´s own movement and the peripheral sensory inflow (Kawato & Gomi, 1992). As a result, damage to the cerebellum, in addition to affecting motor production, could also affect the action-related perception.

A series of neuroimaging studies revealed that the cerebellum also participates in non-motor tasks, such as action observation (Calvo-Merino et al., 2006; Grèzes et al., 1999, 2004), perception of point-light human motion (Sokolov et al., 2010), motor imagery (Decety et al., 1994; Grèzes & Decety, 2001), temporal expectation (Avanzino et al., 2015; O'Reilly et al., 2008) and also in predicting others' actions (Cross et al., 2013), a fundamental aspect of skilled motor behavior (Blakemore & Sirigu, 2003; Kilner et al., 2004; Miall, 2003). During action observation, the observer would map the visual input onto its own motor representations through a fronto-parietal-Superior Temporal Sulcus circuit that might extend to the cerebellum (Gazzola & Keysers, 2009; Miall, 2003) through a motor resonance process (Rizzolatti et al., 1999). The interaction between these cortical areas and the cerebellum would allow us to recruit inverse and forward internal models during action observation (Gazzola & Keysers, 2009; Miall, 2003). One proposal is that such action-perception network (APN) would be reenacted when the observer predicts an upcoming action (Blakemore & Sirigu, 2003; Fontana et al., 2012; Jeannerod, 2001; Kilner et al., 2004; Miall, 2003). Furthermore, studies suggest that APN ensures the continuity of action perception in the absence of sensory input allowing the motion permanence (Cross et al., 2013; Saunier et al., 2013). For instance, a recent neuroimaging study indicated that the temporal prediction of the reappearance of a human being after disappearing behind an obstacle is accompanied by an increase in the cerebellum's hemodynamic response (Cross et al., 2013). However, putative effects of cerebellar lesions upon predicting upcoming movements remain elusive (Abdelgabar et al., 2019). In such a context, a neuropsychological approach appears as a valuable complement to neuroimaging results.

Employing a protocol in which the velocity profile of the motion was manipulated whereas the subject had to predict the final position of an occluded human motion (Pozzo et al.,

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2006; Saunier et al., 2015, 2008), it has been shown that the subjects were more accurate for movements complying with motor rules (Pozzo et al., 2006; Saunier et al., 2008). The authors proposed that such inference of human motion would rely on internal models of action (Pozzo et al., 2006; Saunier et al., 2008). Thus, the use of such a protocol to test motion inference after cerebellar damage could help establishing the role of the cerebellum in predicting upcoming actions.

The aim of this study was to verify the effect of cerebellar damage during the inference of whole-body motion (i.e., sit-to-stand or back-to-sit) depicted in a computer screen by a point light dot corresponding to shoulder displacement. Since sit-to-stand (STS) and back-to-sit (BTS) actions imply postural adjustment, we hypothesized that a medial cerebellar damage, known to affect the postural control, would affect the ability to infer a natural upcoming body axis movement. Such a result would indicate that the cerebellum takes part of a complex predictive neural network involved in biological motion inference. Here, the terms biological or non-biological motion refer to display kinematics respecting or not motor rules (i.e., kinematic invariants), respectively. However, the persistence of biological effect after a cerebellar damage could indicate that the inference process relies on other subcortical and cortical networks.

Materials and methods

Participants

Seven patients were recruited at the Instituto de Neurologia Deolindo Couto – Universidade Federal do Rio de Janeiro, a public hospital facility. Access was granted to the patients' complete neurological diagnosis but not to their exam database. After the experiment, data from two patients were discarded because of instruction misunderstanding at the psychophysical task. Accordingly, five patients (51.4 ± 4.5 years) and six healthy control participants (48.5 ± 4.5 years) matched for age ($W = 9.5$, $p = 0.36$) were considered for the experiment. All participants had normal or corrected to normal vision and were unaware of the purpose of the experiment. The patients were screened for exclusion criteria (severe hand dysmetria, hands' tremor and nystagmus, history of trauma, orthopedic surgery, or other neurological disorder affecting arm´s function and balance in orthostatic position) prior to taking part in the experiment. The main symptomatology of all patients was an important postural instability (gait ataxia) as a consequence of spinocerebellar degeneration or of a medial cerebellar damage after a posterior inferior cerebellar artery stroke (see Table 1).

Although the cause of cerebellum disorder differs between the patients (neurodegenerative disease or infarct), it is essential to emphasize their homogeneity, namely, the presence of a severe postural control disorder as attested by posturographic evaluation.

All subjects gave written informed consent to the experimental procedures and to participate in the study, in accordance with the local Ethical Council guidelines and the Declaration of Helsinki. The project was approved by the local Ethics Committee.

Neuropsychological assessment

All patients were submitted for clinical neurological assessment. The Mini Mental State Examination (MMSE; Brucki et al., 2003) and the Functional Independence Measure (FIM) (Riberto et al., 2001) were applied to test, respectively, the cognitive functionality, and independence level of the patients. The MMSE had a score compatible to the patients´ education level (range 26–30, total 30) and did not depart between groups ($W = 21$, $p = 0.136$). The FIM reached values close to the maximum independence level (range 90–106, total 126). Light impairments were noticed in FIM motor items, such as taking a bath, gait and stair climbing, explaining the differences between group scores ($W = 30$, $p = 0.0039$). The scores of MMSE, FIM, additional personal features, and clinical signs of cerebellar patients and control subjects are described in Table 1.

Posturography

Five patients were selected on the basis of their performance during equilibrium evaluation. All of them were diagnosed with gait ataxia, based on the BERG Balance Scale (BBS; Miyamoto et al., 2004), which evaluates qualitatively deficits in postural control. The BBS score indicated a severe to moderate imbalance (range 32–47, total score 56). The most impaired BBS items were Stand up position, Standing in one leg, Sit to stand, and Back to sit. The BBS score of cerebellar patients (Table 1) confirmed an alteration of postural control in cerebellar patients when compared to the control group ($W = 30$, $p = 0.0038$).

Quantitative data confirmed these qualitative observations about postural deficit of our patient's sample. Indeed, we analyzed the body sway of all participants (Matlab 6.5; Mathworks) by means of the measurement of the Center of Pressure (CoP) displacement using a force platform (AccuSway, AMTI, USA; frequency of 50 Hz; low-pass filter of 5 Hz). Two data acquisition blocks, each of them consisting of four successive 1-min-lasting periods alternating closed eyes (CE) with open eyes (OE), were used to collect postural data. The two blocks were split by a 5-min rest, during which the subjects were allowed to sit and relax. The four acquisition steps within each block were separated from each other by 12–15 seconds, just enough for the examiner to ask the subjects to either close or open the eyes and activate data recording. The first acquisition was randomly chosen between the CE and OE situations. This first recording was systematically discarded. All the recorded posturographic parameters are listed in Table 2 and show a statistical significance between cerebellar and control groups, with higher cerebellar values for the following stabilometric parameters: Area (mm2), frequency (Hz) in AP (anterior-posterior) axis for OE as well as Standard deviation (SD) (mm) in AP and ML (medio-lateral) axes (Figure 1). Moreover, cerebellar patients presented an increase in mean power frequency (Frequency, Hz) in the anterior-posterior axis for OE condition (0.277 ± 0.071 Hz) compared to controls (0.144 ± 0.031 Hz) ($W = 2$, $p = 0.017$). These data suggest that the anteroposterior axis is more severely affected by cerebellar damage than the medial-lateral one. Such results confirm previous reports suggesting that healthy volunteers rely more than cerebellar

Table 1. Personal features and clinical signs of cerebellar patients and control subjects.

Subjects	Age (years; range)	Gender (M/F)	Education (years; range)	Hand laterality (R/L)	Time of diagnosis (years; range)	Diagnosis	NeuroImage	Main Neurological signs	Other signs	BBS (total = 56) (range)	MMSE (total = 30) (range)	FIM (total = 126) (range)
Cerebellar patients	45–57	1 F/ 4 M	8–15	R	1–12					32–47	26–30	90–106
FF	57	M	>15	R	1	Infarct in Medial PICA territory	*MRI* Gliosis in posterior cerebellum, Left lateral posterior Medulla Obl and Left thalamus	Disorders of equilibrium in SUP, STS and BTS, Gait Ataxia	Nystagmus, light Hypermetria Left Arm, dysdiadochokinesia	32	26	98
AB	54	M	>15	R	12	SCA3 (Machado-Joseph Disease)	*MRI* Wider sulci and thiner cerebellar gyri	Disorders of equilibrium in SUP, STS and BTS, Gait Ataxia	Nystagmus, light dysmetria and intention tremor L and R Arm,	34	30	90
HF	51	M	11	R	4	Infarct in Left Para-vermis cerebellum region after aneurysmal subarachnoid hemorrhage	*CT* Hipodensity in Left cerebelar paravermic region	Disorders of equilibrium in SUP, STS and BTS, Gait Ataxia	Nystagmus, dysmetria L and R arm and leg, dysdiadochokinesia	47	30	106
MSP	45	F	8	R	6	SCA7	*MRI* Wider cerebellar sulci	Disorders of equilibrium in SUP, STS and BTS, Gait Ataxia	Nystagmus, light dysmetria L arm and leg	38	28	102
WJFF	50	M	10	R	7	SCA3 (Machado-Joseph Disease)	*MRI* Wider cerebellar sulci and IV ventricule	Disorders of equilibrium in SUP, STS and BTS, Gait Ataxia	Nystagmus, light Hypermetria L arm dysdiadochokinesia	34	30	96
Control subjects	42–53	M	>15	R	-	-	-	-	-	56	30	126

Right (R) and left (L) arm and leg; Female (F) and Male (M); Ischemic (I) and Hemorrhagic (H) stroke, Stand up position (SUP), Stand to sit (STS), Back to sit BTS, BERG balance scale (BBS), Mini-Mental State Examination (MMSE), Functional Independence Measure (FIM), Posterior Inferior Cerebellar Artery (PICA) Spinocerebellar Ataxia (SCA)

Table 2. Posturographic parameters in cerebellar and control subjects.

	CoP					
	Open Eyes			Closed Eyes		
	Control	Cerebellar	p value	Control	Cerebellar	p value
Area (mm2)	332.31 ± 152.21	1261.17 ± 358.73	**0.004**	391.81 ± 189.09	2276.84 ± 1737.63	**0.004**
SD (mm)						
AP	5.660 ± 1.447	11.159 ± 2.061	**0.004**	6.060 ± 1.632	14.724 ± 4.708	**0.004**
ML	4.858 ± 1.522	9.502 ± 1.470	**0.004**	5.303 ± 1.777	11.851 ± 4.758	**0.004**
Frequency (Hz)						
AP	0.144 ± 0.031	0.277 ± 0.071	**0.017**	0.224 ± 0.055	0.301 ± 0.067	0.125
ML	0.212 ± 0.038	0.271 ± 0.109	0.429	0.256 ± 0.051	0.333 ± 0.104	0.177

Posturographic parameter values in open eyes and closed eyes conditions for control and cerebellar group. Data presented as mean ± standard deviation. SD, standard deviation; AP, anterior–posterior direction; ML, medial–lateral direction.

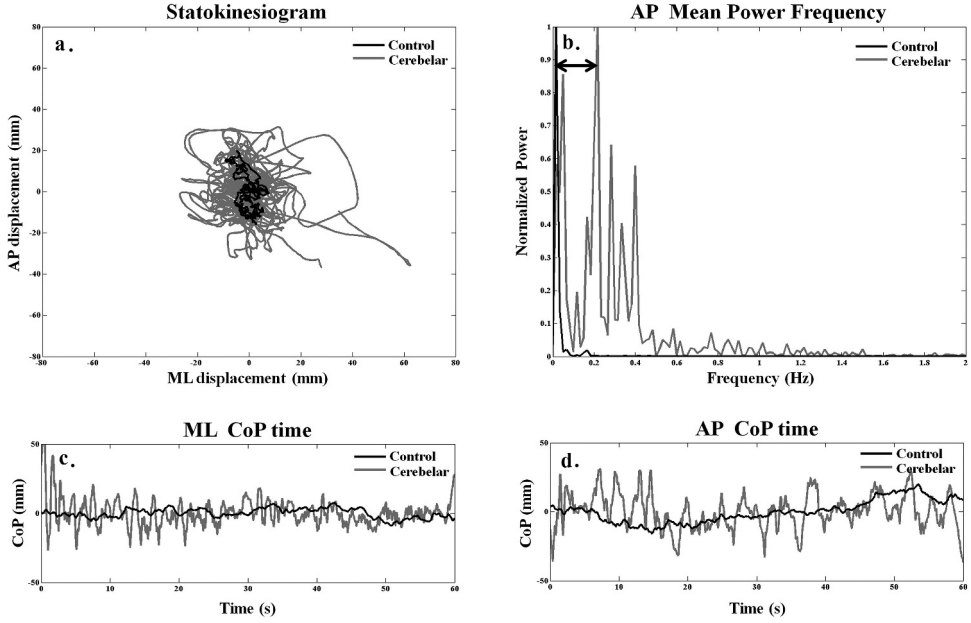

Figure 1. Representative data from one control (black line) and one cerebellar participant (gray line) during 1 minof body sway acquisition. Illustration of the displacement of the Center of Pressure (a), the mean power of frequency in anterior-posterior axis (b), CoP time series in lateral (c) and anterior-posterior directions (d).

patients on visual feedback to control posture (Ohashi, 1984). The postural data confirms the homogeneity of our cerebellar group concerning the deficit in postural control (see Table 2 for a complete overview).

Psychophysical experiments

Cerebellar patients and healthy controls were asked to estimate the final position or the vanishing position of a moving point-light dot (3 pixels in diameter) portraying the shoulder trajectory throughout Sit-to-Stand (STS) or Back-to-Sit movements. An optoelectronic device (Elite System, BTS Bioengineering, Italy) allowed the acquisition of the shoulder marker at a frequency of 100 Hz (Papaxanthis et al., 2003). The STS and BTS movements were displayed on a computer screen and the participant could only see the first 65% of the trajectories. Moreover, the shoulder kinematics followed well-known motor laws (called biological motions) or could violate these same motor laws (called non-biological motions). The lengths of the trajectories were 264 and 274 mm whereas the movement duration was 1.88 s for STS and BTS (Figure 2; see also Saunier et al., 2008 for more details of the experimental protocol).

Before starting the experiment, the participants were notified about the nature of the motion displayed, which corresponded to the shoulder trajectory of STS or BTS performed in the sagittal plane. The space bar was used in order to initiate the movement. After pressing the space bar, the point-light dot movement began within a random interval of 0.2 and 1 s. The order presentation of motions (STS biological – STS B, STS non-biological – STS N, BTS biological – BTS B and BTS non-biological – NB) was randomized within each experiment. For experiments 1 (end point estimation) and 2 (vanishing point estimation) performed 12 trials for each condition (STS B, STS NB, BTS B and BTS NB) for a total of 48 repetitions per experiment.

End point estimation (EP)

The task of the participants consisted to reconstruct the 35% of the occluded trajectory in order to infer the final position of the shoulder's point-light dot since only first 65% of its trajectory was visible. For this, the participants used the mouse to displace the crosshair cursor on the inferred final position of the motion. To validate their responses, the subjects clicked on the left button of the mouse and the cartesian coordinates on x and y axes of the crosshair cursor on the screen were recorded.

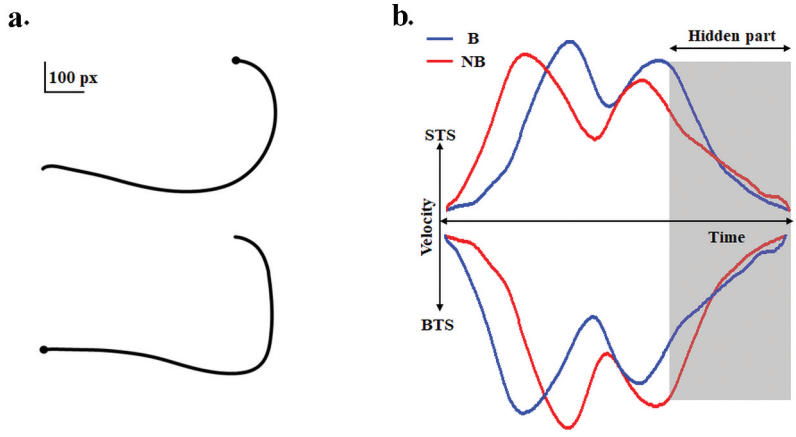

Figure 2. A. The upper part corresponds to the STS shoulder trajectory whereas the lower part corresponds to the BTS shoulder trajectory. The black dots at the end of the trajectories correspond to the final position of STS and BTS motions. B. Shoulder tangential velocity profiles for STS (upper part) and BTS (lower part) motions. The point-light dot could move according to biological rules (B-blue curves) or non-biological rules (NB-red curves). We used the asymmetry of velocity profiles between STS and BTS motion in order to create the non-biological motion. Accordingly, the STS N corresponds to STS trajectory with BTS velocity profile whereas the BTS N corresponds to BTS trajectory with STS velocity profile. The last 35% of the trajectory was occluded, as represented by the gray rectangle on the figure.

Vanishing point estimation (VP)

All participants took part in VP, which consisted in estimate the vanishing position of the point-light dot. Similarly, to EP, the cartesian coordinates of the crosshair cursor were recorded on the vanishing position. Previous studies have shown that EP and VP tasks rely on different mechanisms (Pozzo et al., 2006; Saunier et al., 2008). Whereas the EP task would recall the endogenous information (i.e., in this case, an implicit knowledge about kinematic law) specific to observed movement, the VP estimation is dependent on the moving target features (i.e., velocity at vanishing position and trajectory shape). The VP experiment permits to verify the capacity of cerebellar patients in using visual information to estimate the vanishing position of a moving target. Accordingly, if both groups behave in a similar way to estimate the VP whereas the EP estimation differs, we could deduce that the cerebellum is involved in the EP process.

Fixed point estimation (FP)

All volunteers had to accurately estimate the position of a fixation point that randomly appeared in the screen. This experiment consisted of 24 trials. To respond, they used the mouse, similarly to the procedure previously described in experiment 1. This control permitted us to test whether the possible difference in accuracy between control and cerebellar patients could be the consequence of a visuomotor deficit in the use of the mouse device.

Data analysis

For each trial, the accuracy in the estimation of the end point (EP) and vanishing point (VP) of the stimulus position was defined as the difference between the position estimated by the subject in the x and y axes and the true final position. Then, for each subject we calculated the mean radial error (RE). The RE was calculated as $\sqrt{(x^2 + y^2)}$. A non-parametric test, Wilcoxon t-test, was applied to compare the differences between biological and non-biological motion estimations for the cerebellar participants and the control volunteers (R software environment). The significance level of the statistical tests was $p < 0.05$. Trials where the estimations of EP or VP exceeded three standard deviations above or below the group mean for each condition were considered as outliers and were excluded of the statistical analysis.

Results

End point estimation

Our main result is that the level of cerebellar accuracy did not change regardless of the type of motion (biological or non-biological) as compared to the control group, which was more accurate in inferring the final position of the biological motion (Figure 3). Indeed, the mean cerebellar RE for biological motion was 76.16 ± 19.25 pixels against 76.40 ± 23.63 pixels (V = 9, p = 0.81) for non-biological motion. For the control group, we observed that the participants were more accurate to infer the final position of biological motion (RE = 62.44 ± 32.56 pixels; RE = 75.78 ± 32.93 pixels for biological and non-biological motion, respectively; V = 0, p = 0.031). In short, contrarily to age-paired control subjects, the biological velocity profile of the motion did not allow an increase in the end point accuracy in cerebellar patients (Figure 4(a)).

Vanishing point estimation

No differences were observed between biological and non-biological estimation in order to estimate the VP for the cerebellar group (RE = 35.39 ± 10.46 pixels; RE = 32.5 ± 14.93 pixels for biological and non-biological motion, respectively; V = 9, p = 0.81) and the control group (RE = 33.66 ± 15.46 pixels; RE = 34.47 ± 9.01 pixels for biological and non-biological motion, respectively; V = 9, p = 0.84) respectively (Figure 4(b)). Figure 5 presents the vanishing position estimations from a typical cerebellar patient and a typical control participant.

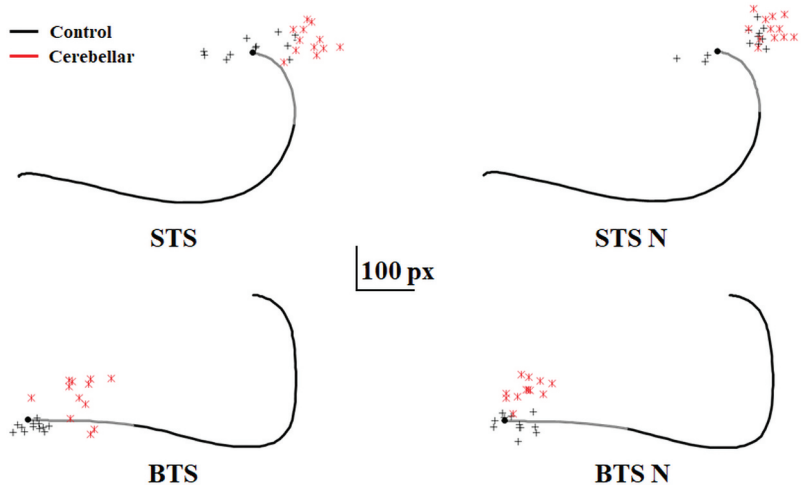

Figure 3. End point estimation of a typical control and a typical cerebellar participant. Black corresponds to the visible part of the trajectory whereas gray corresponds to the hidden part of the trajectory. The black dot corresponds to the final position for STS biological (STS), STS non-biological (STS N), BTS biological (BTS) and BTS non-biological (BTS N) motions. Crosses correspond to the end point position estimated by a control (black) and cerebellar (red) participants.

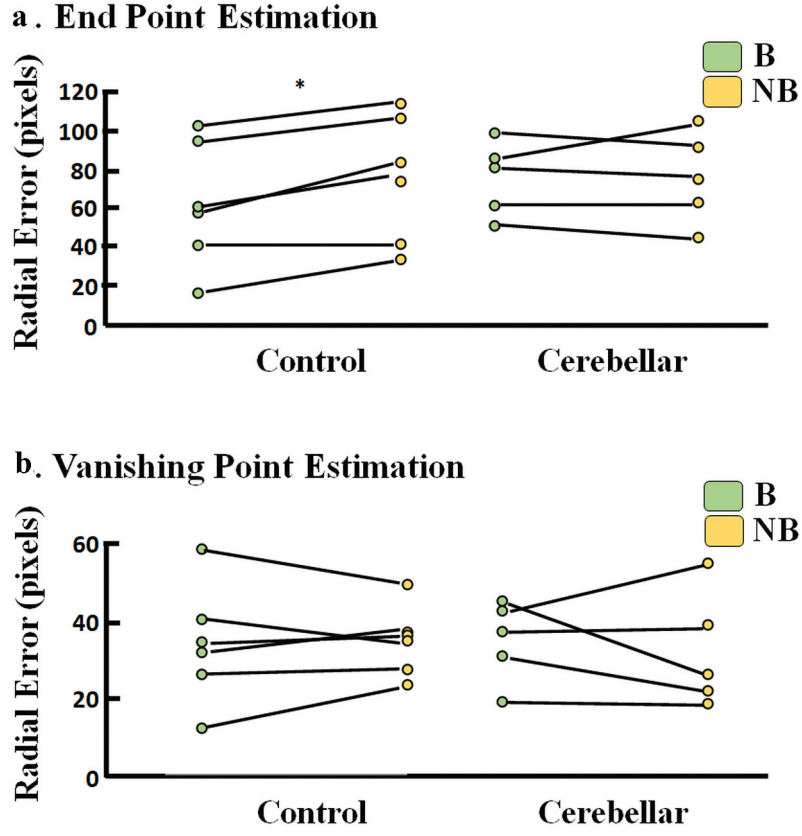

Figure 4. Psychophysical results of the control and cerebellar groups. The circles represent the mean radial errors of the subjects for the end point estimation (a) and for the vanishing point estimation (b). The solid black line links the response of the same participants for the biological and non-biological condition. Star indicates statistical significance ($p < 0.05$).

Fixed point estimation

All subjects were very precise in their spatial estimation of a fixed point randomly appearing in the screen. A Mann–Whitney U test did not reveal any significant differences between groups ($W = 7$, $p = 0.177$). The RE was 1.11 ± 0.42 pixels and 1.67 ± 0.65 pixels for control and cerebellar groups, respectively.

Discussion

The present study addresses the effect of cerebellum damage in the retrieval of internal models of action triggered during the inference process of an occluded whole-body trajectory (i.e., sit-to-stand or back-to-sit) depicted from an impoverished visual display. Consistent with previous studies that have established

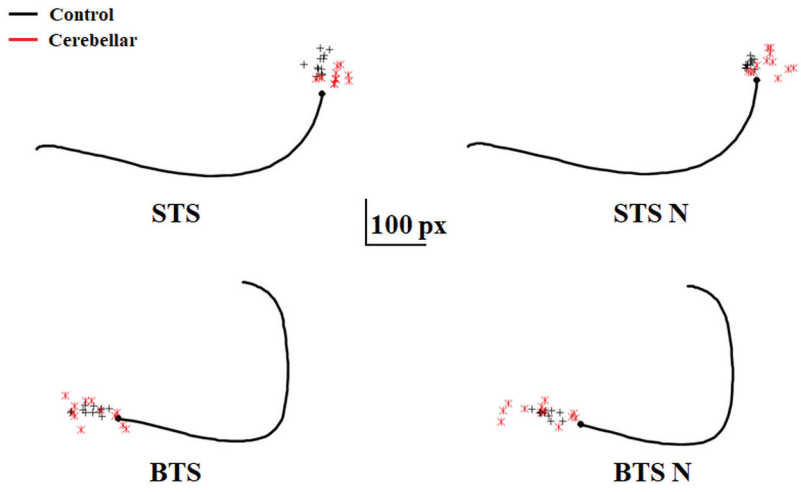

Figure 5. Vanishing point estimation of a typical control and a typical cerebellar participant. Black corresponds to the visible part of the trajectory. Dots correspond to the vanishing position for STS biological (STS), STS non-biological (STS N), BTS biological (BTS) and BTS non-biological (BTS N) motions. Crosses correspond to the estimated vanishing position given by the control (black) and cerebellar (red) participants.

the medial cerebellum region as a key region for upright stance control (Grimaldi & Manto, 2012; Morton & Bastian, 2007), the cerebellar group presented an alteration of postural control parameters as compared to age paired control subjects. Crucially, cerebellar patients were insensitive to subtle kinematic differences (biological vs. non-biological velocity profiles) of a moving point-light depicting a human body's axial movement. Interestingly, in contrast to the results found for the end point estimation, cerebellar damage did not affect the ability to estimate the vanishing position of a moving target. Corroborating with our previous study (Saunier et al., 2008), the feature of velocity profile (biological or non-biological) did not modulate the RE magnitude of VP for both cerebellar and control groups. Such results confirmed that both groups estimated similarly the vanishing position of a moving target. This control condition permits us to exclude the possibility that the impaired capacity to employ kinematic laws to estimate the final position of a whole-body trajectory was not due to any deficiency in visuomotor coding in our cerebellar patients.

Inference process after cerebellar damage

Cerebellar patients were insensitive to subtle kinematic differences in order to reconstruct a human motion trajectory when its final portion was occluded, contrasting with the preserved capacity to estimate the vanishing position in a similar way than control participants. This confirms previous studies (Pozzo et al., 2006; Saunier et al., 2008) demonstrating that the inference process relies on motoric knowledge whereas the estimation of vanishing point appears dependent of the stimulus characteristics at its vanishing position (i.e., the velocity or the trajectory shape). In other words, departing from age-paired healthy controls, cerebellar patients were less accurate to estimate the final position of a motion complying with kinematic laws as compared to non-biological motion. Thus, the novelty of the present report is the behavioral evidence of the cerebellum involvement during the reconstruction of a human trajectory based onto subtle whole-body kinematic differences and from the displacement of an impoverished visual stimulus (a unique point-light dot).

Our report also complements a recent action perception study that found the involvement of cerebellum in perceiving/discriminating kinematics features of hand action whereas spinocerebellar patients presented a perceptual deficit for the same experimental condition (Abdelgabar et al., 2019). Accordingly, the cerebellum's integrity appears as a *sine qua non* condition to extract or discriminate motor invariants during motion perception. Taken together, these results are also in line with a fMRI study that demonstrated how the passive observation of a point light motion complying with kinematic laws was sufficient to increase the hemodynamic response of the cerebellum (Dayan et al., 2007).

The potential role of the cerebellum in estimating an upcoming movement is supported by a theoretical framework proposing the involvement of cortico-subcortical pathways linking the fronto-parietal complex and the cerebellum during action anticipation, through the recall of internal models of action (Balser et al., 2014; Gazzola & Keysers, 2009; Miall, 2003). In a previous similar study (Saunier et al., 2008), we proposed that the cortical core of an action perception network (APN) (i.e., the fronto-parietal complex) would host inverse and forward internal models of action. The interaction between them might directly tune the inference process of visual motion. An inverse model would map the visual input to its corresponding motor counterpart (i.e., STS or BTS motor planning). Then, from this inverse internal model, the elaboration of an efference copy would permit to elaborate the sensory consequences of STS or BTS motion. This forward model would be used to reconstruct the occluded trajectory.

The introduction of a non-biological velocity profile might thus generate a discrepancy between the kinematics extracted through the visual input and the kinematics probably stored in the parietal cortex (Kalaska et al., 1990), explaining the greater magnitude of radial error for non-biological motion in healthy subjects. Hence, we propose that the cerebellum is embedded within this complex network endowed with the implicit capacity of discriminating biological from non-biological kinematics. This

raises the interesting hypothesis of cerebellum role in encoding the motoric features of the visual input. A recent neuroimaging study (Balser et al., 2014) found a strong involvement of the cerebellum during an anticipation task consisting in estimate the direction of tennis or volleyball services. The cerebellum would host a forward internal model of the observed actions that would permit to resolve this anticipation task.

The anatomical connections between the cerebellum and cortical areas belonging to the Action Perception Network (APN) support such a view (Coffman et al., 2011). For instance, it was recently demonstrated that the vermis received afferent projections from the motor cortex in non-human primates whereas the cerebellum indirectly projects efferent signals to the parietal lobule (Coffman et al., 2011), which is regularly described as a key structure in the elaboration of action prediction (Blakemore & Sirigu, 2003; Fontana et al., 2012). Moreover, a functional connectivity study by Buckner et al. (2011) provided evidence that cerebellar somato-motor representations of the body were mostly present within the medial region of the cerebellum, mainly the affected region within our patient sample. We hypothesize that a medial cerebellum damage could affect the motor representation of whole-body motion as well as its associated processes such as action prediction.

The present study has limitations that should be worth noting. First, our sample of cerebellar patients is reduced. Second, in the absence of anatomical images of the patients' damage at the time of the experiment, we cannot rule out the possibility that their damage extended beyond the medial cerebellar region.

Conclusion

Motor rules, subtended within complex internal model loops based onto cerebro-cerebellar pathways carrying out specific action representations, also seem to be retrieved to predict the final position of an occluded biological motion. Herein we support the direct involvement of the cerebellum within such complex cortical-subcortical networks. This pathway might play a crucial role in anticipating the consequence of whole-body movement and its potential effects upon postural control during action observation. Thus, classically, described as a motor control structure, the cerebellum appears also to be involved in predicting upcoming actions by observation.

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