European Journal of Neuroscience, Vol. 21, pp. 3439–3446, 2005

Differential involvement of the cerebellum in biological and coherent motion perception

Daniel Jokisch,[1,2] Nikolaus F. Troje,[1,2,3] Benno Koch,[4] Michael Schwarz[4] and Irene Daum[1,2]
[1]Institute of Cognitive Neuroscience, Department of Neuropsychology, Ruhr-University Bochum, Universitätsstraße 150, D-44780 Bochum, Germany
[2]International Graduate School of Neuroscience, Ruhr-University, Bochum, Germany
[3]Department of Psychology, Queen's University, Kingston, Ontario, Canada
[4]Department of Neurology, Klinikum Dortmund, Dortmund, Germany

Keywords: biological motion, cerebellum, human, motion perception, threshold detection

Abstract

Perception of biological motion (BM) is a fundamental property of the human visual system. It is as yet unclear which role the cerebellum plays with respect to the perceptual analysis of BM represented as point-light displays. Imaging studies investigating BM perception revealed inconsistent results concerning cerebellar contribution. The present study aimed to explore the role of the cerebellum in the perception of BM by testing the performance of BM perception in patients suffering from circumscribed cerebellar lesions and comparing their performance with an age-matched control group. Perceptual performance was investigated in an experimental task testing the threshold to detect BM masked by scrambled motion and a control task testing the detection of motion direction of coherent motion masked by random noise. Results show clear evidence for a differential contribution of the cerebellum to the perceptual analysis of coherent motion compared with BM. Whereas the ability to detect BM masked by scrambled motion was unaffected in the patient group, their ability to discriminate the direction of coherent motion in random noise was substantially affected. We conclude that intact cerebellar function is not a prerequisite for a preserved ability to detect BM. Because the dorsal motion pathway as well as the ventral form pathway contribute to the visual perception of BM, the question of whether cerebellar dysfunction affecting the dorsal pathway is compensated for by the unaffected ventral pathway or whether perceptual analysis of BM is performed completely without cerebellar contribution remains to be determined.

Introduction

Motion patterns characteristic of living beings are termed biological motion (BM). Detection of such motion patterns is a fundamental property of the human visual system. Humans can efficiently detect another living being in the visual environment, and are able to retrieve many features from its kinematics. An experimental approach to uncouple information from BM from other non-dynamic sources of information is to represent the main joints of a person's body by bright dots against a dark background (Johansson, 1973). Employing this point-light display technique, observers can easily recognize a human walker, determine his or her gender (Kozlowski & Cutting, 1977; Barclay *et al*., 1978; Cutting, 1978; Mather & Murdoch, 1994; Troje, 2002), recognize various action patterns (Dittrich, 1993), identify individual persons (Cutting & Kozlowski, 1977) and even recognize themselves (Beardsworth & Buckner, 1981).

The highly adaptive value of an efficient perception of animate motion patterns is reflected by a specific neural machinery performing perceptual analysis of such visual information (Bonda *et al*., 1996; Grossman *et al*., 2000; Grezes *et al*., 2001; Grossman & Blake, 2001, 2002; Vaina *et al*., 2001; Servos *et al*., 2002). Neuroimaging studies report selective activation of the superior temporal sulcus (STS) to visual stimuli consisting of BM. In addition to area STS, activation specific for BM has also been shown in the cerebellum (Grossman *et al*., 2000; Vaina *et al*., 2001), area VP (Servos *et al*., 2002), the amygdala (Bonda *et al*., 1996), the occipital and fusiform face area (Grossman & Blake, 2002) and the premotor cortex (Saygin *et al*., 2004). Results from these studies and from computational modelling (Giese & Poggio, 2003) are consistent with neuropsychological findings in neurological patients suffering from focal cortical brain lesions (Vaina *et al*., 1990; Vaina, 1994; McLeod *et al*., 1996; Schenk & Zihl, 1997; Cowey & Vaina, 2000).

The cerebellum has traditionally been viewed as a brain structure subserving skilled motor behaviour. Although recent work has suggested a much broader functional role of the cerebellum with contributions to a wide range of cognitive and perceptual functions (for reviews see Daum *et al*., 2001; Justus & Ivry, 2001), the role of the cerebellum in BM perception is unclear. The neuroimaging literature on the role of the cerebellum with respect to the perception of BM is inconsistent, with some studies reporting cerebellar involvement (Grossman *et al*., 2000; Vaina *et al*., 2001), while others failed to detect cerebellar activity associated with BM perception (e.g. Grezes *et al*., 2001; Grossman & Blake, 2001; Servos *et al*., 2002). Moreover, there are some inconsistencies regarding the cerebellar substructure that may be involved in BM perception. Grossman *et al*. (2000) found cerebellar activity in the anterior portion near the midline, whereas Vaina *et al*. (2001) reported activity specific to BM in lateral parts of the cerebellum.

Correspondence: Dr D. Jokisch, at present address below.
E-mail: Daniel.Jokisch@fcdonders.ru.nl

Present address: F.C. Donders Centre for Cognitive Neuroimaging, P.O. Box 9101, NL-6500 HB Nijmegen, The Netherlands.

Received 26 November 2004, revised 7 March 2005, accepted 5 April 2005

doi:10.1111/j.1460-9568.2005.04145.x

The current study aims to elucidate the functional role of the cerebellum in perception of BM using a lesion approach, i.e. examining the perceptual performance of patients with selective cerebellar lesions. Within this context, a particular issue of interest was the differential cerebellar contribution to visual processing of BM relative to motion perception per se.

Materials and methods

Two experimental tasks were administered in order to explore the functional role of the cerebellum in the perception of BM and to compare its involvement in non-BM perception. A group of patients with selective ischaemic cerebellar lesions was examined in these tasks. The patients' perceptual performance was compared with the performance of an age-matched control group. Perceptual performance was assessed by determining the threshold for the detection of masked BM and masked non-BM. In the BM task, the presence or absence of a point-light walker that was masked by dots consisting of scrambled motion had to be detected. In the non-BM task, observers had to detect the motion direction of coherently moving dots that were masked by random noise dots.

Participants

Seven cerebellar patients and seven healthy control subjects participated in the investigation. The patients (ranging from 27 to 68 years, mean age 45.6 years) suffered from a cerebellar infarction of either the posterior inferior cerebellar artery (PICA), the anterior inferior cerebellar artery (AICA) or the superior cerebellar artery (SupCA) in the post-acute state. Inclusion criteria were absence of depression or other psychiatric or neurological disorders and absence of cortical lesions. Main cerebellar symptoms in the acute stage included ataxia, dysmetria, dysarthria and impairments of fine motor coordination.

Cerebellar lesions were documented by magnetic resonance imaging (MRI) scans, which were acquired using a standard three-dimensional (3D) T2 weighted sequence (1 mm × 1 mm × 5 mm voxel size). Figure 1 shows the MRI scans of the patients illustrating the sites of the cerebellar lesions. Table 1 summarizes patients' demographic data and lesion location. The examination was carried out between 16 and 47 months (mean 27.4 months) after the ischaemic event. At this time, patients suffered only from residual motor impairments.

Patients were extensively screened in neuropsychological functioning. Their present state IQ was assessed by the similarities and picture completion subtests of the short German version from the Wechsler Intelligence Scale (Dahl, 1972). According to these subtests, their mean IQ was 113.6 and therefore in the average to upper average range. Patients' ability to scan the visual field was tested with the visual scanning subtest of a widely used German attention test battery (Zimmermann & Fimm, 1993). In this subtest, patients' performance for search accuracy ranged between percentile score 14 and percentile score 58, indicating performance in the normal or close to the normal range (normal range: percentile score 15–85). Visual scanning was thus unimpaired in our sample of patients.

Healthy control subjects were recruited by advertisement to match the patients with respect to age (ranging from 24 to 67 years, mean

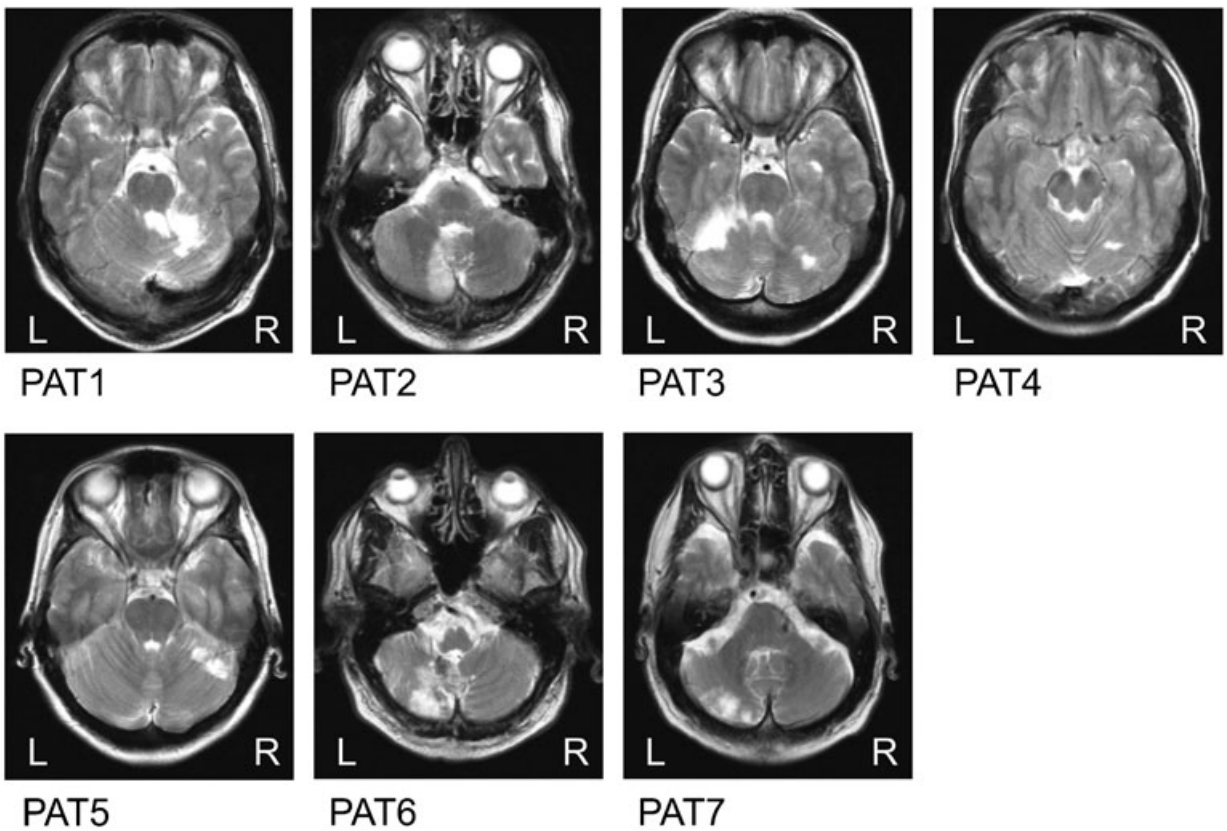

FIG. 1. Transaxial sections of the T2-weighted MRI scans documenting the cerebellar lesions. Note that for PAT4, Table 1 indicates a bilateral lesion, whereas Fig. 1 only illustrates a right lesion. The left lesion is located more ventrally and cannot be seen in the slice presented here.

TABLE 1. Patients' demographic data and lesion locations.

Patient	Age (years)	Sex	Time since lesion (months)	Side of lesion	Artery territory			Cerebellar area			Location
					AICA	SupCA	PICA	PL	AL	V	
PAT1	42	M	47	R	–	+	–	+	+	–	Medial (incl. deep nuclei)
PAT2*	47	M	23	L	–	–	+	+	+	+	Medial
PAT3*	27	F	20	L, R	–	+, +	–	+, +	+, +	–, –	Lateral
PAT4	28	F	28	L, R	–	+, +	–	+, +	–, +	–, –	Lateral
PAT5	41	F	29	R	+	+	–	+	+	–	Lateral
PAT6*	66	M	16	L	–	–	+	+	–	–	Medial
PAT7	68	M	29	L	–	–	+	+	–	–	Medial

*Patients being able to solve the control task. AICA: anterior inferior cerebellar artery, SupCA: superior cerebellar artery; PICA: posterior inferior cerebellar artery; AL: anterior lobe, PL: posterior lobe; V: vermis. The plus sign indicates the affected territory and the affected area. In the cases of bilateral lesions, each hemisphere is documented separately.

age 45.1 years) and sex. All participants had normal or corrected to normal vision. The examination was undertaken with the understanding and written consent of each participant. The study had been approved by the ethics committee of the Ruhr-University Bochum.

Stimuli

Stimuli in all experimental tasks were presented using Matlab with the Psychophysics Toolbox extension (Brainard, 1997; Pelli, 1997). In all experimental trials, stimuli were presented for a duration of 200 ms in order to preclude an effect of fixation shifts.

Biological motion detection

Perception of BM was tested with stimuli of point-light walkers in frontal view masked by noise dots representative of scrambled motion. Stimuli were presented as white dots on a black screen (Fig. 2). The mask dots had the same local motion trajectories as the dots defining the point-light displays, but the spatial relation among the dots was removed by randomizing their initial starting position.

Three male individuals served as walking models for the construction of the point-light displays. Motion data of the models were acquired by recording their walking patterns in 3D space using a motion capture system equipped with nine CCD cameras (Oxford Metrics, Vicon 512). Models were instructed to walk at a comfortable speed through the capture volume which was 7 m long. A set of 41 retroreflective markers was attached to their bodies. The motion capture system tracks the 3D trajectories of the markers with spatial accuracy in the range of 1 mm and a temporal resolution of 120 Hz. From the original 41 markers the trajectories of 15 'virtual' markers positioned at major joints of the body were computed. Commercially available software (Bodybuilder, Oxford Metrics) for biomechanical modelling was used to perform the respective computations. Translational motion was subtracted such that the walkers appeared to walk on a treadmill.

Degree of difficulty of the detection task was manipulated by varying the number of mask dots from 0 to 60 dots in steps of five dots. Accordingly, 13 different degrees of difficulty were obtained. In half of the trials the walker was present, and in the other half the walker was absent and replaced by the same number of scrambled dots. Mask dots and random dots were displayed in an area subtending a $7.4 \times 7.4°$ visual angle. Within the display area, the position of the point-light displays as well as the positions of the mask dots were chosen randomly. The walkers subtended $5.5 \times 1.5°$ of visual angle at the viewing distance of 57 cm. Point-light displays of walkers were computed in real time on a frame-by-frame basis and synchronized with the 60-Hz refresh rate of the 15-inch monitor to ensure smooth, regular motion.

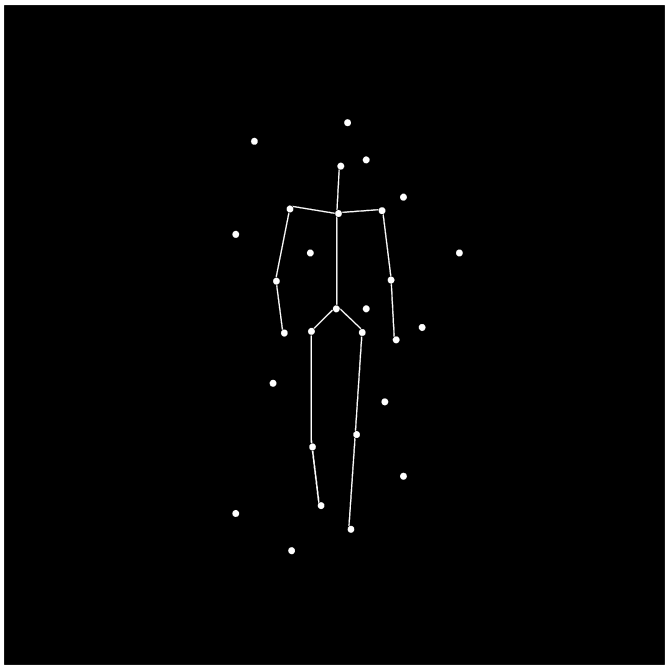

FIG. 2. Depiction of the stimuli used in the BM task: dots connected by lines represent the point-light walker. Remaining dots represent scrambled motion. Lines are only drawn in the figure depiction for the sake of clarity.

Coherent motion detection

Perception of non-BM was tested with displays of coherent motion in random noise, which were matched with respect to size to the BM stimuli ($7.4 \times 7.4°$). Displays consisted of 200 white dots with a size of $0.05 \times 0.05°$ presented on a black screen. A specified percentage of the dots moved coherently at a speed of 6°/s either to the right or to the left hand side (Fig. 3). Signal dots had a limited lifetime of five frames. After the end of lifetime the dots disappeared and reappeared on the screen at a location opposite to the direction of movement. The mask dots were positioned randomly within the display area and had a limited lifetime of two frames in which they were displayed stationary. After disappearing they reappeared at a new location. The percentage of

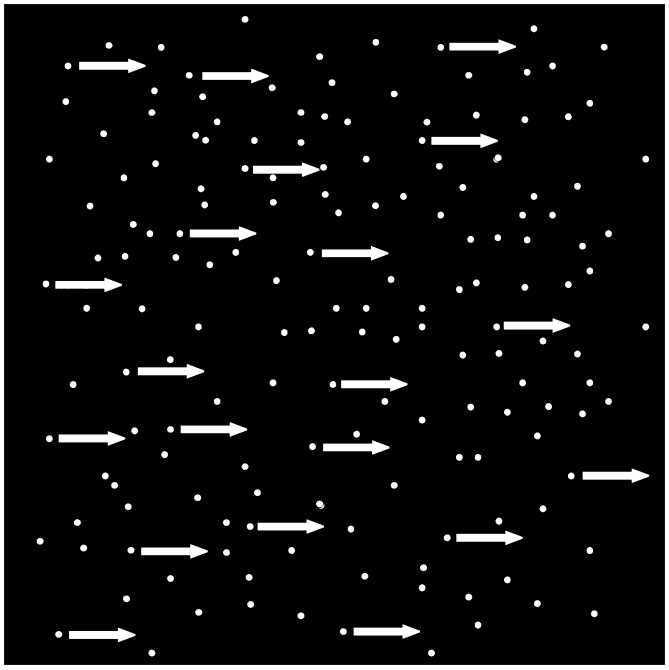

FIG. 3. Depiction of the stimuli used in the control task (coherent motion in random noise): the direction of coherently moving dots is illustrated by arrows. Remaining dots represent random noise.

signal dots was varied between 65% and 5% in steps of 5%, resulting in 13 different degrees of difficulty.

Procedure

The experiment was carried out in the Klinikum Dortmund and in the Institute of Cognitive Neuroscience of the Ruhr-University Bochum. All participants were seated in front of a 15″ monitor at a distance of 57 cm with response buttons under their right hands. Stimuli in both tasks were presented at the centre of the screen for 200 ms in order to preclude eye movements. Successive trials were separated by intertrial intervals of 2000 ms during which a black screen was presented. Before each trial a fixation cross was presented for 2000 ms.

Both experiments comprised three blocks of 52 trials each (13 degrees of severity × 4 repetitions), resulting in 156 single trials per experimental task. Trials within each block were presented in random order. The two experimental tasks were presented in counterbalanced order. Participants were asked to maintain central eye fixation and to respond as accurately as possible by pressing one of the response buttons. Instructions stressed accuracy rather than speed of responding. Observers did not receive feedback on their responses. Before starting the experimental trials participants were shown demonstration trials in order to familiarize them with the display and the setup.

Data analysis

Experimental data were analysed in two consecutive steps separately for each experiment. First, the detection threshold for each subject was determined for both experimental paradigms. The likelihood to respond correctly by chance was 50% in both experimental tasks. The threshold was defined as the signal-to-noise ratio (SNR) needed to perform correctly at 75% of the trials per degree of severity. To achieve this, a sigmoidal curve (Boltzmann function) was fitted to the experimental data with the upper asymptote fixed at 100% performance and the lower asymptote fixed at chance level corresponding to 50% performance. Figure 4 illustrates this procedure for a single subject.

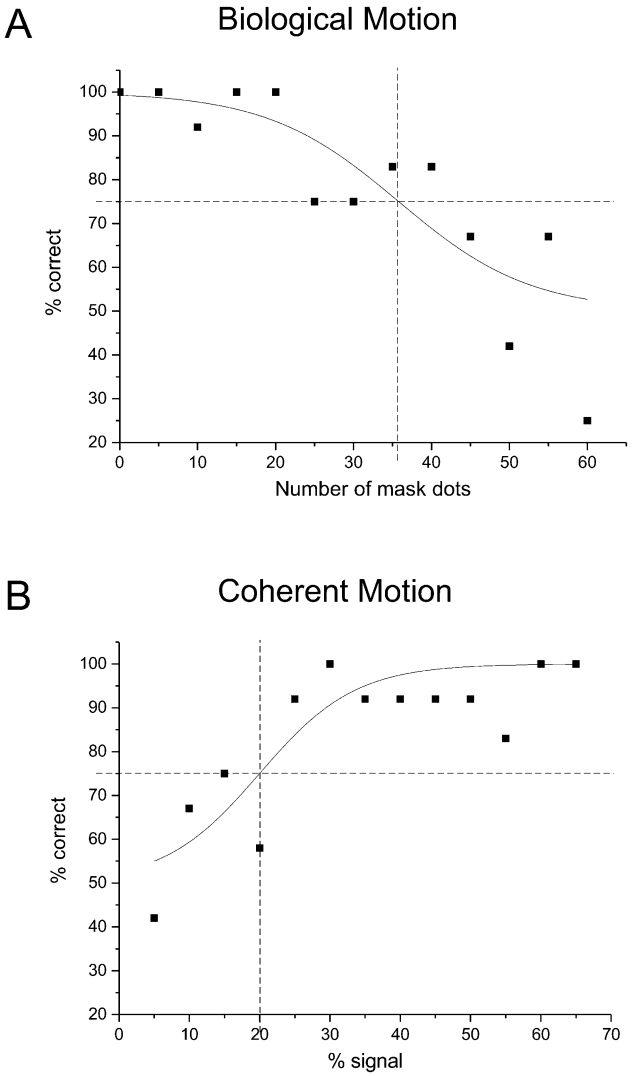

FIG. 4. Illustration of the procedure to determine the detection threshold for a single subject in BM detection (A) and coherent motion detection (B).

Subsequently, a group comparison of the thresholds of the subjects of the experimental and control groups was performed by a *t*-test for independent measures separately for both experiments. In addition, the reaction times were recorded. A group comparison of the median reaction time per subject was performed by a *t*-test for independent measures separately for both experiments.

Results

Biological motion detection

On average, the control group reached the threshold criterion in the BM paradigm at a noise level of 23.3 masking dots. Cerebellar patients showed a similar performance, reaching the criterion at a noise level of 22.2 masking dots (Fig. 5). A group comparison between the experimental and control groups revealed no significant

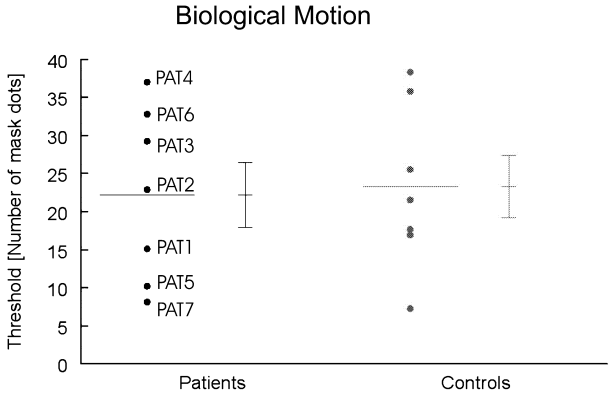

FIG. 5. BM task: perceptual threshold as the number of mask dots for the detection of a point-light walker masked by scrambled motion. Error bars indicate SEM.

differences of the threshold to detect BM in the current paradigm ($t_{12} = 0.183$, $P = 0.858$).

Coherent motion detection

In the control task comprising the detection of the direction of coherent motion in random noise, control subjects reached the criterion at an SNR of 34.40%. By contrast, only three out of seven cerebellar patients were able to solve the task even at the highest SNR of 65% presented in the experiment. In order to compare both groups statistically, for those patients who did not succeed in solving the task, a threshold value of 70% SNR was submitted to analysis. Note that this conservative procedure underestimates the magnitude of the impairment in direction detection of coherent motion in random noise in the patient group. Applying this procedure, the patient group needed on average an SNR of 61% to fulfil the criterion (Fig. 6). A group comparison revealed a significant difference between the experimental and control groups ($t_{12} = 2.757$, $P = 0.020$).

Relation between biological and coherent motion perception

In order to examine the relation between performance in BM detection and coherent motion detection, a correlation analysis was calculated. For the patient group, Pearson's correlation coefficient between performance in both tasks was −0.293 ($P = 0.524$). For the control group, Pearson's correlation coefficient was 0.116 ($P = 0.805$). The correlation between performance in BM and coherent motion detection failed to reach significance in both groups, although Pearson's correlation coefficient was slightly higher in the patient group. According to these results, BM perception and coherent motion perception seem to be independent processes.

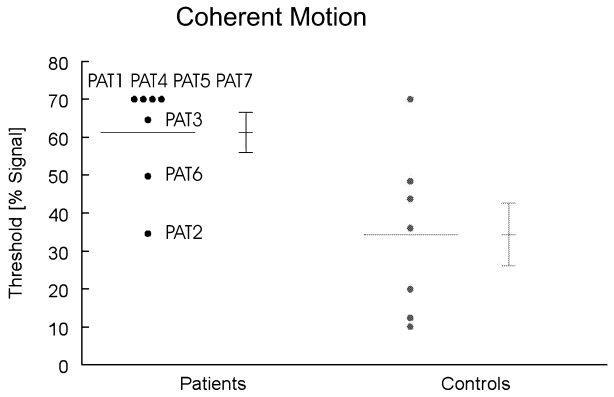

FIG. 6. Control task: perceptual threshold as the SNR for direction detection of coherent motion masked by random noise. Error bars indicate SEM.

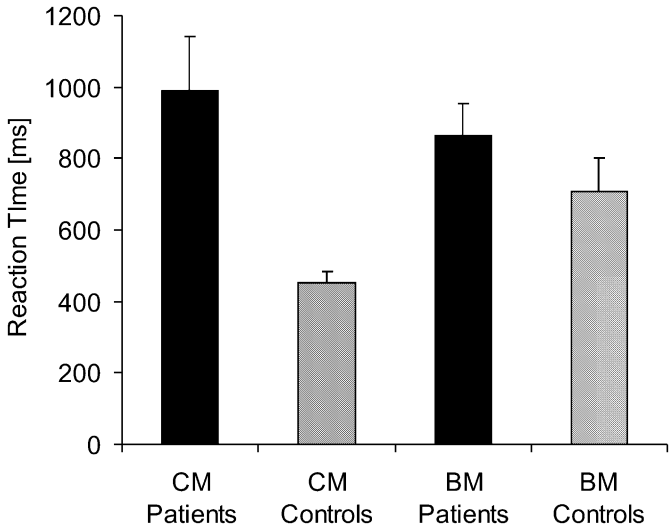

FIG. 7. Reaction times in the BM detection task (BM) and the coherent motion detection task (CM) for each group. Error bars indicate SEM.

Reaction times

In addition to accuracy, the reaction times were analysed (Fig. 7). For coherent motion detection, a group comparison revealed longer reaction times for patients (989 ms) than for controls (451 ms) ($t_{12} = 3.454$, $P = 0.005$). Separate analyses of correct and error trials yielded significant group differences for correct trials ($P = 0.005$) and a trend towards significant differences for incorrect trials ($P = 0.107$). A different pattern emerged for BM detection. Reaction times for patients (864 ms) and controls (707 ms) did not differ significantly ($t_{12} = 1.2$, $P = 0.253$). This is true for correct trials ($P = 0.175$) as well as for incorrect trials ($P = 0.267$).

Discussion

The objective of the current study was to elucidate the differential contribution of the cerebellum to the perceptual analysis of BM by examining perceptual performance of patients with selective cerebellar lesions. Previous imaging studies revealed inconsistent results with respect to cerebellar activation in BM perception (Bonda *et al*., 1996; Grossman *et al*., 2000; Grezes *et al*., 2001; Grossman & Blake, 2001, 2002; Vaina *et al*., 2001; Servos *et al*., 2002). It is difficult to estimate on the basis of neuroimaging studies alone which brain regions are critically involved in specific aspects of cognitive function, because multiple co-activations are usually observed when applying this method. As a consequence, neuropsychological studies of patients with selective lesions to different regions play an important role in the evaluation of the distinct nature of information processing in each brain region.

The present results show clear evidence for a differential contribution of the cerebellum to the perceptual analysis of coherent motion perception on the one hand and BM on the other hand. Whereas the

perception of coherent motion in random noise was substantially affected in our patients with selective cerebellar lesions, the ability to perceive BM camouflaged by scrambled motion was unaffected. In addition, we did not observe significant correlations between the perceptual threshold for BM detection and coherent motion detection in each group. Moreover, patients' higher threshold for coherent motion detection corresponds to longer reaction times. When comparing overall performance in the present BM detection task with performance in other studies exploring BM detection using scrambled motion as a mask (Bertenthal & Pinto, 1994), the detection threshold obtained in the present work is substantially higher. This difference is probably due to the very short presentation time in the present study (200 ms) compared with 1000 ms in the study by Bertenthal & Pinto (1994).

The finding of an impairment in the detection of movement direction in the control paradigm confirms previous reports (Ivry & Diener, 1991; Nawrot & Rizzo, 1995, 1998). Results from a study comparing perceptual judgements of the velocity of moving stimuli and the position of static stimuli in cerebellar patients (Ivry & Diener, 1991) showed selective impairments for the discrimination of moving stimuli. Further support for the notion of cerebellar involvement in motion perception was given by Nawrot & Rizzo (1995, 1998), who showed that midline cerebellar lesions can cause visual motion perception deficits in tasks such as detecting the direction of dot movements in a masking paradigm. These deficits occur during the acute stage as well as in the chronic stage of lesions. The primary interest of the current study was to explore whether there is a differential contribution of the cerebellum to BM perception as compared with motion perception per se.

The general framework, i.e. presentation time and general procedure, was identical in both experimental tasks and cannot explain the performance deficits of the patients in the control task. This is particularly true for the role of eye movements. In order to control the influence of eye movements, a very short presentation time of 200 ms was chosen. Within this short time period it is almost impossible to initiate eye movements. If ocular motor problems or defective fixation had played a major role in these tasks, performance in both tasks would have been affected to a similar extent.

The cerebellum plays a critical role in motor control, with the lateral regions mediating movement planning and programming, and the medial regions contributing to the execution of movements (Dichgans & Diener, 1984). Accordingly, the most prominent symptoms after cerebellar dysfunction are impairments in motor control. For this reason, accuracy rather than reaction time was stressed in the instructions of the experiments. Because significant reaction time differences between patients and controls were observed only in the coherent motion detection protocol, these differences cannot be attributed to motor impairments.

To understand the current results of intact perception of BM in patients with selective cerebellar lesions, consideration of the cortical network involved in the perception of BM might provide deeper insight. Both the dorsal motion pathway and the ventral form pathway contribute to the perceptual analysis of BM (for a review see Giese & Poggio, 2003). Findings from imaging studies are complemented by computational simulations modelling key experimental findings with respect to BM perception (Giese & Poggio, 2003) and neuropsychological studies examining patients suffering from selective cortical lesions (MacLeod, 1988; Vaina *et al*., 1990; Vaina, 1994; Schenk & Zihl, 1997; Cowey & Vaina, 2000). These case studies provide evidence for a dissociation between mechanisms involved in the perception of BM on the one hand and mechanisms involved in inanimate visual motion tasks or static object recognition tasks on the other hand. Patients LM (MacLeod, 1988) and AF (Vaina *et al*., 1990) who have bilateral lesions involving the posterior visual pathway showed severe deficits in visual motion perception but can nevertheless recognize human action patterns presented as point-light displays. Patients with bilateral ventral lesions involving the posterior temporal lobes such as patient EW (Vaina, 1994), who suffered from prosopagnosia and object agnosia, could identify BM in point-light animations as well. By contrast is patient AL (Cowey & Vaina, 2000) who is hemianopic and suffers from visual perceptual impairments in her intact hemifield as a consequence of an additional lesion in the ventral extrastriate cortex. AL fails to recognize BM displays despite intact static form perception and motion detection. This pattern of impairments makes sense when assuming that the lesion in the intact hemifield includes the STS-complex, which receives input from both the ventral and the dorsal visual stream.

Given these case studies and computational simulations, it is reasonable to assume that detection of BM can be achieved by the ventral or dorsal visual stream alone if the STS-complex is still intact. From this point of view it might be possible that the cerebellum facilitates perceptual analysis of BM in the dorsal visual stream. Nevertheless, dysfunctional cerebellar processing would not necessarily lead to a significant impairment in BM perception, as dysfunctions of the dorsal visual stream could be compensated for by intact processing of the ventral visual stream. Alternatively, one might argue that the perceptual analysis of BM is completely performed by neocortical structures without any cerebellar contribution to BM perception at all. This view is also in accordance with our present findings. Moreover, there is empirical evidence that the cerebellum becomes not only active during execution of movement sequences but also during motor imagery in tasks such as imagination of complex movement sequences (Decety *et al*., 1990; Ryding *et al*., 1993; Decety, 1996; Luft *et al*., 1998; Hanakawa *et al*., 2003). Activity in response to point-light displays of BM as observed in some imaging studies (Grossman *et al*., 2000; Vaina *et al*., 2001) might be a result of such feedforward mechanisms.

The variety of neural connections of the cerebellum to cortical areas provides the neuroanatomical basis for cerebellar contributions to a variety of perceptual tasks. The cerebellum projects from lateral parts via the dentate nucleus and the thalamus to several neocortical structures, among them the prefrontal cortex, the superior temporal sulcus and the parietal cortex (Schmahmann & Pandya, 1997). These regions project back to the cerebellum via the pontine nuclei. The lateral cerebellum was shown to be engaged during the acquisition and discrimination of somatosensory information (Gao *et al*., 1996). It was suggested that the lateral cerebellum may be specifically active during motor, perceptual and cognitive performances because of the requirement to process sensory data. In the view of Bower (1997), the cerebellum is assumed to facilitate the efficiency with which other brain structures perform their own function, and therefore, it is considered useful but not imperative for many different kinds of brain functions. The view of a general contribution of the cerebellum to the acquisition of sensory data is inconsistent with the present findings, because the ability of cerebellar patients to perceive BM was spared. Therefore, cerebellar function with respect to sensory data acquisition must be more specific.

Keele & Ivry (1990) have put forward the idea that the cerebellum has the function of an internal clock that measures time intervals in the millisecond range. Such an exact timing of very short intervals subserves motor as well as non-motor functions. The demonstration of the role of the cerebellum in visual perceptual functions that require velocity perception (Ivry & Diener, 1991; Nawrot & Rizzo, 1995, 1998) was also interpreted to be in accordance with the timing hypothesis. Similarly, deficits in speech perception (Ackermann *et al*.,

1999) and classical conditioning (Daum *et al*., 1993; Topka *et al*., 1993; Woodruff-Pak *et al*., 1996) have also been discussed in relation to impaired timing in cerebellar patients.

Recently the timing hypothesis of cerebellar function has been modified by differentiating event timing from emergent timing (Ivry *et al*., 2002; Spencer *et al*., 2003). Event timing is defined as a form of representation in which the temporal goals are explicitly represented. In contrast, emergent timing reflects temporal consistencies that arise through the control of other parameters. Whereas the cerebellum is involved in tasks requiring explicit temporal representation (event timing), it seems to be less important in emergent timing, which requires other control parameters not associated with the cerebellum.

The timing hypothesis, especially in its modified form, can best explain our present findings. An exact timing is necessary in order to detect coherent motion in random noise. Considering motion as spatial displacement per time unit, the direct link between the accurate representation of small time units and motion perception becomes obvious. In the case of BM, the motion information only mediates the form of a human observer. A precise timing in the millisecond range seems to be unnecessary with respect to this perceptual demand. Nevertheless, the timing hypothesis may not explain all deficits seen in cerebellar patients. Thier *et al*. (1999) tried to identify the nature of visual impairments resulting from cerebellar dysfunction by a set of experiments. Their results support the presence of visual deficits in cerebellar disease, but in contrast to previous studies, they provide evidence against a common, simple denominator that can explain the deficits in both motion perception and position discrimination.

Previous studies (Ivry & Diener, 1991; Nawrot & Rizzo, 1998, 1995) reported that visual motion perception is linked to the medial rather than the lateral cerebellum. The impairment pattern found in our sample of cerebellar patients does not support this view. Patients in our study failed to show such a clear distinction between more medial and more lateral located lesions with respect to perceptual performance in the control task.

Two out of four patients who showed deficits in direction discrimination of coherently moving dots had lesions primarily affecting medial parts and two patients had lesions primarily affecting lateral parts. By contrast, the three patients who were able to solve the task had lesions in medial parts as well as in lateral parts. This result pattern does not allow us to conclude that the functional integrity of the medial cerebellum is necessary for normal motion perception.

Taken together, functional integrity of the cerebellum is not required for BM detection. Taking into consideration that both the dorsal visual stream as well as the ventral visual stream contribute to the perception of BM, it might be possible that processing in the dorsal motion pathway is affected by cerebellar dysfunction but processing in the ventral form pathway can compensate for this deficit. An impairment would thus not emerge on the behavioural level when the ventral form pathway is intact.

Acknowledgements

This research was funded by the International Graduate School of Neuroscience (IGSN) of the University of Bochum, Germany, and by the Volkswagen Foundation. We would like to thank Cord Westhoff for programming essential parts of the experiments and Katrin Heyder for providing assistance with screening patients in neuropsychological functioning.

Abbreviations

3D, three-dimensional; BM, biological motion; SNR, signal-to-noise ratio; STS, superior temporal sulcus.

References

Ackermann, H., Graber, S., Hertrich, I. & Daum, I. (1999) Cerebellar contributions to the perception of temporal cues within the speech and nonspeech domain. *Brain Lang.*, **67**, 228–241.

Barclay, C.D., Cutting, J.E. & Kozlowski, L.T. (1978) Temporal and spatial factors in gait perception that influence gender recognition. *Percept. Psychophys.*, **23**, 145–152.

Beardsworth, T. & Buckner, T. (1981) The ability to recognize oneself from a video recording of one's movements without seeing one's body. *Bull. Psychonomic Soc.*, **18**, 19–22.

Bertenthal, B.I. & Pinto, J. (1994) Global processing of biological motions. *Psychol. Sci.*, **5**, 221–225.

Bonda, E., Petrides, M., Ostry, D. & Evans, A. (1996) Specific involvement of human parietal systems and the amygdala in the perception of biological motion. *J. Neurosci.*, **16**, 3737–3744.

Bower, J.B. (1997) Control of sensory data acquisition. In Schmahmann, J.D. (Ed.), *The Cerebellum and Cognition*. Academic Press, San Diego, pp. 490–513.

Brainard, D.H. (1997) The Psychophysics Toolbox. *Spat. Vis.*, **10**, 433–436.

Cowey, A. & Vaina, L.M. (2000) Blindness to form from motion despite intact static form perception and motion detection. *Neuropsychologia*, **38**, 566–578.

Cutting, J.E. (1978) Generation of synthetic male and female walkers through manipulation of a biomechanical invariant. *Perception*, **7**, 393–405.

Cutting, J.E. & Kozlowski, L.T. (1977) Recognizing friends by their walk: gait perception without familiarity cues. *Bull. Psychonomic Soc.*, **9**, 353–356.

Dahl, G. (1972) *Reduzierter Wechsler Intelligenztest (Short Version of the Wechsler Intelligence Test).* Hain, Meisenheim.

Daum, I., Schugens, M.M., Ackermann, H., Lutzenberger, W., Dichgans, J. & Birbaumer, N. (1993) Classical conditioning after cerebellar lesions in humans. *Behav. Neurosci.*, **107**, 748–756.

Daum, I., Snitz, B.E. & Ackermann, H. (2001) Neuropsychological deficits in cerebellar syndromes. *Int. Rev. Psychiatry*, **13**, 268–275.

Decety, J. (1996) Do imagined and executed actions share the same neural substrate? *Brain Res. Cogn. Brain Res.*, **3**, 87–93.

Decety, J., Sjoholm, H., Ryding, E., Stenberg, G. & Ingvar, D.H. (1990) The cerebellum participates in mental activity: tomographic measurements of regional cerebral blood flow. *Brain Res.*, **535**, 313–317.

Dichgans, J.E. & Diener, H.C. (1984) Clinical evidence for functional compartmentalisation of the cerebellum. In Bloedel, J., Diechgans, J. & Precht, W. (Eds), *Cerebellar Functions*. Springer, Berlin, pp. 126–147.

Dittrich, W.H. (1993) Action categories and the perception of biological motion. *Perception*, **22**, 15–22.

Gao, J.H., Parsons, L.M., Bower, J.M., Xiong, J., Li, J. & Fox, P.T. (1996) Cerebellum implicated in sensory acquisition and discrimination rather than motor control. *Science*, **272**, 545–547.

Giese, M.A. & Poggio, T. (2003) Neural mechanisms for the recognition of biological movements. *Nat. Rev. Neurosci.*, **4**, 179–192.

Grezes, J., Fonlupt, P., Bertenthal, B.I., Delon-Martin, C., Segebarth, C. & Decety, J. (2001) Does perception of biological motion rely on specific brain regions? *Neuroimage*, **13**, 775–785.

Grossman, E.D. & Blake, R. (2001) Brain activity evoked by inverted and imagined biological motion. *Vision Res.*, **41**, 1475–1482.

Grossman, E.D. & Blake, R. (2002) Brain areas active during visual perception of biological motion. *Neuron*, **35**, 1167–1175.

Grossman, E.D., Donnelly, M., Price, R., Pickens, D., Morgan, V., Neighbor, G. & Blake, R. (2000) Brain areas involved in perception of biological motion. *J. Cogn. Neurosci.*, **12**, 711–720.

Hanakawa, T., Immisch, I., Toma, K., Dimyan, M.A., Van Gelderen, P. & Hallett, M. (2003) Functional properties of brain areas associated with motor execution and imagery. *J. Neurophysiol.*, **89**, 989–1002.

Ivry, R.B. & Diener, H.C. (1991) Impaired velocity perception in patients with lesions of the cerebellum. *J. Cogn. Neurosci.*, **3**, 355–366.

Ivry, R.B., Spencer, R.M., Zelaznik, H.N. & Diedrichsen, J. (2002) The cerebellum and event timing. *Ann. NY Acad. Sci.*, **978**, 302–317.

Johansson, G. (1973) Visual perception of biological motion and a model for its analysis. *Percept. Psychophys.*, **14**, 201–211.

Justus, T.C. & Ivry, R.B. (2001) The cognitive neuropsychology of the cerebellum. *Int. Rev. Psychiatry*, **13**, 276–282.

Keele, S.W. & Ivry, R. (1990) Does the cerebellum provide a common computation for diverse tasks? A timing hypothesis. *Ann. NY Acad. Sci.*, **608**, 179–207; discussion 207–111.

Kozlowski, L.T. & Cutting, J.E. (1977) Recognizing the sex of a walker from a dynamic point-light display. *Percept. Psychophys.*, **21**, 575–580.

Luft, A.R., Skalej, M., Stefanou, A., Klose, U. & Voigt, K. (1998) Comparing motion- and imagery-related activation in the human cerebellum: a functional MRI study. *Hum. Brain Mapp.*, **6**, 105–113.

MacLeod, C.M. (1988) Forgotten but not gone: savings for pictures and words in long-term memory. *J. Exp. Psychol. Learning, Memory, Cognition*, **14**, 195–212.

Mather, G. & Murdoch, L. (1994) Gender discrimination in biological motion displays based on dynamic cues. *Proc. R. Soc. Lond. B*, **258**, 273–279.

McLeod, P., Dittrich, W., Driver, J., Perrett, D. & Ziehl, J. (1996) Preserved and impaired detection of structure from motion by a 'motion-blind' patient. *Visual Cognition*, **3**, 363–391.

Nawrot, M. & Rizzo, M. (1995) Motion perception deficits from midline cerebellar lesions in human. *Vision Res.*, **35**, 723–731.

Nawrot, M. & Rizzo, M. (1998) Chronic motion perception deficits from midline cerebellar lesions in human. *Vision Res.*, **38**, 2219–2224.

Pelli, D.G. (1997) The VideoToolbox software for visual psychophysics: transforming numbers into movies. *Spat. Vis.*, **10**, 437–442.

Ryding, E., Decety, J., Sjoholm, H., Stenberg, G. & Ingvar, D.H. (1993) Motor imagery activates the cerebellum regionally. A SPECT rCBF study with 99mTc-HMPAO. *Brain Res. Cogn. Brain Res.*, **1**, 94–99.

Saygin, A.P., Wilson, S.M., Hagler, D.J. Jr, Bates, E. & Sereno, M.I. (2004) Point-light biological motion perception activates human premotor cortex. *J. Neurosci.*, **24**, 6181–6188.

Schenk, T. & Zihl, J. (1997) Visual motion perception after brain damage. II. Deficits in form-from-motion perception. *Neuropsychologia*, **35**, 1299–1310.

Schmahmann, J.D. & Pandya, D.N. (1997) The cerebrocerebellar system. In Schmahmann, J.D. (Ed.), *The Cerebellum and Cognition*. Academic Press, San Diego, pp. 31–60.

Servos, P., Osu, R., Santi, A. & Kawato, M. (2002) The neural substrates of biological motion perception: an fMRI study. *Cereb. Cortex*, **12**, 772–782.

Spencer, R.M., Zelaznik, H.N., Diedrichsen, J. & Ivry, R.B. (2003) Disrupted timing of discontinuous but not continuous movements by cerebellar lesions. *Science*, **300**, 1437–1439.

Thier, P., Haarmeier, T., Treue, S. & Barash, S. (1999) Absence of a common functional denominator of visual disturbances in cerebellar disease. *Brain*, **122**, 2133–2146.

Topka, H., Valls-Sole, J., Massaquoi, S.G. & Hallett, M. (1993) Deficit in classical conditioning in patients with cerebellar degeneration. *Brain*, **116**, 961–969.

Troje, N.F. (2002) Decomposing biological motion: a framework for analysis and synthesis of human gait patterns. *J. Vis.*, **2**, 371–387.

Vaina, L.M. (1994) Functional segregation of color and motion processing in the human visual cortex: clinical evidence. *Cereb. Cortex*, **4**, 555–572.

Vaina, L.M., Lemay, M., Bienfang, D.C., Choi, A.Y. & Nakayama, K. (1990) Intact 'biological motion' and 'structure from motion' perception in a patient with impaired motion mechanisms: a case study. *Vis. Neurosci.*, **5**, 353–369.

Vaina, L.M., Solomon, J., Chowdhury, S., Sinha, P. & Belliveau, J.W. (2001) Functional neuroanatomy of biological motion perception in humans. *Proc. Natl Acad. Sci. USA*, **11**, 11.

Woodruff-Pak, D.S., Papka, M. & Ivry, R.B. (1996) Cerebellar involvment in eyeblink classical conditioning in humans. *Neuropsychology*, **10**, 443–458.

Zimmermann, P. & Fimm, B. (1993) *Testbatterie zur Aufmerksamkeitsprüfung*. PSYTEST, Würselen.