



## Research report

# Recovery of biological motion perception and network plasticity after cerebellar tumor removal



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## ABSTRACT

Visual perception of body motion is vital for everyday activities such as social interaction, motor learning or car driving. Tumors to the left lateral cerebellum impair visual perception of body motion. However, compensatory potential after cerebellar damage and underlying neural mechanisms remain unknown. In the present study, visual sensitivity to point-light body motion was psychophysically assessed in patient SL with dysplastic gangliocytoma (Lhermitte–Duclos disease) to the left cerebellum before and after neurosurgery, and in a group of healthy matched controls. Brain activity during processing of body motion was assessed by functional magnetic resonance imaging (fMRI). Alterations in underlying cerebro-cerebellar circuitry were studied by psychophysiological interaction (PPI) analysis. Visual sensitivity to body motion in patient SL before neurosurgery was substantially lower than in controls, with significant improvement after neurosurgery. Functional MRI in patient SL revealed a similar pattern of cerebellar activation during biological motion processing as in healthy participants, but located more medially, in the left cerebellar lobules III and IX. As in normalcy, PPI analysis showed cerebellar communication with a region in the superior temporal sulcus, but located more anteriorly. The findings demonstrate a potential for recovery of visual body motion processing after cerebellar damage, likely mediated by topographic shifts within the corresponding cerebro-cerebellar circuitry induced by cerebellar reorganization. The outcome is of importance for further understanding of cerebellar plasticity and neural circuits underpinning visual social cognition.

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## 1. Introduction

Visual processing of body motion is considered to be of substantial value for social competence and daily life

activities (Pavlova, 2012). Most studies have been aimed at investigation of cortical areas involved in visual processing of biological motion such as the superior temporal sulcus, STS (e.g., Grossman & Blake, 2002; Herrington, Nymberg, &

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Schultz, 2011; Pelphrey et al., 2003; Puce & Perrett, 2003), whereas possible contribution of brain structures outside the cerebral cortex remained unclear. In accord with evidence on cerebellar involvement not only in various motor but also cognitive functions (Schmahmann, 1996; Strick, Dum, & Fiez, 2009), recent lesion findings in neurosurgical patients with left lateral cerebellar tumors indicate substantial preoperative deficits in visual sensitivity to body motion (Sokolov, Gharabaghi, Tatagiba, & Pavlova, 2010). Furthermore, in healthy participants, left lateral cerebellar lobules Crus I and VIIb are activated during visual processing of body motion (Sokolov et al., 2012). Connectivity analyses indicate two-way effective communication and bidirectional fiber pathways between the cerebellum and right STS (Sokolov et al., 2012; Sokolov, Erb, Grodd, & Pavlova, 2014), a core area within the cortical network underlying visual processing of body motion (Grosbras, Beaton, & Eickhoff, 2012; Grossman & Blake, 2002; Pavlova, 2012; Pelphrey et al., 2003; Puce & Perrett, 2003). Both interaction of the STS with the cerebellum and the deficits encountered in patients with cerebellar lesions suggest that integrity of the cerebellum is of importance for proper functioning of the neural network underpinning visual perception of body motion.

Given the role of visual perception of body motion in social cognition, interaction and competence (Pavlova, 2012), evolution of this ability after cerebellar damage is of particular interest. Yet there is a lack of evidence as to possible changes in sensitivity to body motion after cerebellar tumor removal. In general, impaired motor coordination following damage to the cerebellum tends to swiftly recover (Flourens, 1824). In contrast, data on cognitive recovery are sparse, controversial, and indicate a general tendency for improvement over time (Schmahmann & Sherman, 1998; Silveri, Di Betta, Filippini, Leggio, & Molinari, 1998; Vokaer et al., 2002) though with persistence of some cognitive deficits (Vokaer et al., 2002). Alterations of cortical activity due to cerebellar damage have been assessed (Händel, Thier, & Haarmeier, 2009; Ziemus et al., 2007), but changes in cerebellar processing remain unclear.

On the basis of previous observations in motor and cognitive recovery (Flourens, 1824; Schmahmann & Sherman, 1998; Silveri et al., 1998; Vokaer et al., 2002), we hypothesized postoperative improvement in visual sensitivity to body motion. Furthermore, we intended to study underlying brain mechanisms, assuming topographic reorganization within the cerebellum, compensation by contralateral cerebellar regions or by the cerebral cortex.

## 2. Methods

### 2.1. Participants

We studied a patient SL (female, right-handed, aged 33.8 years at first admission) and six healthy matched controls (females, right-handed, aged  $35.6 \pm 3.7$  years) without history of psychiatric or neurological disorders. Patient SL was initially seen at the Department of Neurosurgery, University of Tübingen Medical School, prior to surgery of an extensive dysplastic gangliocytoma (World Health Organization – WHO grade I) to

the left cerebellum (Fig. 1B). Neurological examination, psychophysical evaluation of visual sensitivity to point-light biological motion and structural magnetic resonance imaging (MRI) recording were performed before neurosurgery, as well as 8 and 24 months postoperatively. Functional MRI (fMRI) was used to assess brain activity during body motion processing and to study possible alterations in the underlying brain network 52 months after neurosurgery. The study was approved by the local Ethics Committee of the University of Tübingen Medical School. Informed written consent was obtained from each subject.

Initial symptoms reported by patient SL were bilateral occipital headache and vertigo. Neurological examination at admission revealed ataxia of the left lower extremity, dysmetria and dysdiadochokinesia of the left upper extremity, as well as gait imbalance and pathological Romberg and Unterberger tests. Oculomotor deficits, papilledema and diminished visual acuity were excluded by neuro-ophthalmological testing. At the period of examination, the patient did not receive any medication. Preoperative neurological deficits disappeared shortly after neurosurgical lesion removal, with a normal neurological status at follow-up examinations. Several weeks after neurosurgery, SL was able to perform a number of everyday life activities (such as car driving) requiring not only highly coordinated motor but also visual perceptual skills. Cerebellar tumor was identified by structural MRI (Fig. 1B). Hydrocephalus and additional brain lesions were excluded. Radiological suspicion of dysplastic gangliocytoma (WHO grade I) was confirmed by pathological examination. Follow-up MRI scans illustrated the postoperative cerebellar tissue loss after 8 months (Fig. 1C), that remained stable after 24 months (Fig. 1D).

### 2.2. Psychophysical paradigm and task

The experimental paradigm is well established and had been used in a previous study on biological motion in patients with left cerebellar lesions, where it is described in more detail (Sokolov et al., 2010). In brief, participants had to detect a point-light walker embedded within an array of identical, moving dots. A canonical point-light walker was comprised of 11 bright dots placed on the head and major joints of an otherwise invisible human body. The point-light walker was seen facing right, moving with no net translation (Fig. 1A). The walker was masked by 44 additional moving dots, derived from spatially scrambled walker sets. The size, luminance, and phase relations of the dots remained unchanged. To preserve spatial characteristics (such as density), walker-absent stimuli were created using an additional set of 11 moving dots. Configurations were computer-generated by using modified Cutting's algorithm (Cutting, 1978). In a display, moving dots were distributed within about  $5.0^\circ$  in height by  $7.0^\circ$  in width, with the target subtending a visual angle of  $4.0^\circ$  in height and  $2.8^\circ$  in width at the most extended point of a gait cycle. A gait cycle was accomplished in 40 frames with frame duration of 36 msec. Participants were tested individually, seated at a distance of 57 cm from the screen. They viewed moving dot displays either with or without an embedded walker. Three experimental runs (32 stimuli per run  $\times$  three runs = 96 trials in total) were

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