

VISUAL SENSITIVITY TO BODY MOTION AND SOCIAL COGNITION

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Abstract

Bodily movements help to improve our social communication by means of non-verbal information about social properties. Observers can discriminate between deceptive and true intentions conveyed by body dynamics, and true information is precisely detected despite deceptive endeavours. Brain imaging data point to existence of distributed networks that subserve revealing of social attributes through body dynamics. The right superior temporal sulcus is repeatedly reported to be a substantial part of these networks. Our MEG findings support this view shedding light on the temporal cortical dynamics. In particular, revealing of cortical regions in which the visual sensitivity to body motion parallels MEG cortical activity help to uncover temporal and spatial dynamics of the networks involved in social cognition. By using clinical models of abnormal development, namely, patients with early periventricular lesions and autistic spectrum disorders, we show that structural and functional brain connectivity is of immense importance for proper functioning of the networks underlying visual social perception.

Human body motion provides a rich source of visual information about others, which is of tremendous significance not only for a variety of daily-life activities, but in particular, for social interaction and non-verbal communication. In the present work, we assessed temporal and spatial changes in MEG cortical response in adolescents with periventricular leukomalacia, PVL, during detection of camouflaged body motion. PVL, the dominant form of brain injury in individuals who were born premature, is characterized by gliosis in the white matter and tissue loss with secondary ventricular dilatation, thereby affecting the pathways interconnecting subcortical structures with cortical regions and cortico-cortical connectivity (Scranes et al., 2007). Our previous work in this population shows that: (i) Visual processing of body motion is compromised in adolescents with PVL (Pavlova et al., 2005, 2006c, 2007a); (ii) These deficiencies are specifically related to the volumetric extent and topography of PVL (Pavlova et al., 2006c); (iii) Motor experience in human locomotion does not appear to be a necessary prerequisite for the visual analysis of body motion. (Pavlova et al., 2003). Here, by using demanding task to detect biological motion embedded in a simultaneous noise, we address the issue of how periventricular lesions modulate a proper functioning of the large-scaled neural network engaged in visual processing of body motion.

Method

Participants

Patients were eight adolescents (4 female and 4 male, age range 13-16 years) born premature between 27 and 33 weeks of gestation. All of them suffered PVL revealed on an MRI scan.

The average volume of parieto-occipital PVL of the left hemisphere was 15.123 ± 10.483 ml, and the volume of the right hemispheric parieto-occipital PVL was 16.54 ± 8.682 ml. The average volume of PVL in the left temporal region was 1.517 ± 1.005 ml, and in the right temporal region it was 1.976 ± 1.413 . The average volume of PVL in the left frontal region was 7.586 ± 3.762 ml, and in the right frontal region it was 7.748 ± 3.06 . The inter-hemispheric differences in PVL volumetric extent over the parieto-occipital, temporal and frontal regions were not significant (t-test). Ten term-born (4 female and 6 male) adolescents were recruited from the local community. They had MRI scans without any identifiable signs of brain abnormalities, and served as controls. All participants had normal or corrected vision. Verbal IQ (Intelligence Quotient) greater than 85 (HAWIK-III, Hamburg-Wechsler-Intelligence-Test-für-Kinder, based on the WISC III, adapted to the German population) was an inclusion criterion. For patients, verbal IQ scores were in the range of 101-124 (average, 108 ± 7.66), and for controls in the range of 97-127 (average, 117.87 ± 14.88). The groups did not differ in verbal IQ scores (t-test, n.s.). The inclusion criteria for all groups of children were also limited by the very nature of MEG recording. Children wearing teeth braces could not participate.

Detection task with camouflaged body motion

Participants were presented with computer-generated point-light configurations. One type of stimuli represented a canonical point-light walker embedded in an array of 33 distracters competing with motions of the walker's dots. The other type of stimuli was a 44-dot mask: additional 11 dots were added to the walker-absent displays so that their density matched that of the walker-present displays. A canonical point-light walker was comprised of 11 dots placed on the joints (ankles, shoulder, etc) of an invisible human body. It was seen moving and facing right, in a sagittal view, with no net translation. A gait cycle was accomplished in 40 frames with frame duration of 36 ms. The target subtended a visual angle of 9° in height and 6° in width at the most extended point of a gait cycle. A thirty-three-dot distracter consisted of three sets of spatially scrambled dots on the joints of a canonical walker. Within a set, the motion of each dot mimicked the motion of one of the dots defining the point-light target figure. The size, luminance, and phase relations of the dots also remained unchanged. In a display, moving dots were distributed within a region of about 12° in height by 18° in width. Each run contained 200 trials with an equal number of walker-present and walker-absent displays. The order of display presentations was randomized. In a yes-no paradigm, participants had to detect the presence of a point-light walker. No immediate feedback was given regarding performance. The stimulus appeared for 1 s on a blank screen with an inter-stimulus interval that varied randomly between 3.5-4.0 s. To eliminate the influence of motor activity on recorded MEG traces, participants were asked to respond right after the stimulus offset and avoid responding during the stimulus presentation. If a subject responded to the stimulus within the stimulus presentation, this trial was discarded.

MEG recording and data processing

A participant was seated in an electromagnetically shielded chamber (Vakuum-Schmelze, Hanau, Germany). The cortical responses were recorded with the whole-head MEG system (CTF Systems, Inc.; Vancouver, Canada) comprising 151 hardware first-order magnetic gradiometers distributed with an average distance of 3 cm between sensors. Sessions with head movements exceeding 0.5 cm were discarded. Epochs containing blinks or eye movements ($> \pm 100$ mV) were rejected. MEG recording session (during presentation of a set of 200 stimuli) lasted 10-12 min. Because, we were interested in relatively early brain

activation, root-mean-square (RMS) analysis was performed in temporal windows of 180-244 ms and 276-340 ms separately for occipital, parietal, temporal, and frontal regions, and for each hemisphere. For analysis of visual sensitivity to biological motion, the sensitivity index (d'), was computed for each participant.

Results

In PVL patients, the sensitivity index (d') to camouflaged body motion was in the range of 0.8-2.84 (mean 2.29 ± 0.67), and in controls in the range of 1.24-5.05 (mean 3.13 ± 1.22). Pair-wise comparison reveals significant difference in visual sensitivity between the PVL patients and controls. No differences were found between PVL patients and controls in cognitive decision criteria ($\ln \beta$). This indicates that it is a reduction in sensitivity that is responsible for poorer performance of PVL patients. Although participants made on average only few false alarms, no difference was found between PVL patients and controls in the false alarm rate (calculated as a ratio of the number of false alarms to the total number of stimuli, for which this type of error might occur). This indicates that poorer performance of PVL patients cannot be explained by possible changes in personality characteristics (e.g., by anxiety). In patients, difficulties in body motion detection were also reflected in response time that was longer for walker-absent than for walker-present displays (mean 566 ± 289 ms, for walker, and 645 ± 261 ms, for mask; t-test, one-tailed, $p < 0.017$). This difference was absent in controls (mean 563 ± 270 ms, for walker, and 591 ± 208 , for mask; n.s.).

In the first time window of 180-244 ms, a three-way ANOVA with factors Stimulus (Walker-present/Mask only), Group (PVL patients/Controls) and Cortical Region (Occipital right, Parietal right, Temporal right, Frontal right, Occipital left, Parietal left, Temporal left, Frontal left) was performed on individual RMS values. This analysis revealed a highly significant effect of region ($F(7,112) = 8.028$, $p < 0.0001$). Neither main effects of stimulus and group, nor interactions between factors were significant. Post-hoc pair-wise comparisons showed that in response to the walker-present displays, the cortical activation over the right temporal cortex was weaker in PVL patients as compared with healthy controls (mean 89.2 fT, SE ± 8.85 in PVL patients, and 123 fT, SE ± 18.56 in controls, $p < 0.05$). By contrast, the activation did not differ between PVL patients and controls in response to the control mask-only display (mean 85.1 fT, SE ± 10.52 in PVL patients, and 98.1 fT, SE ± 10.72 in controls). This alteration in activation, therefore, was specific for body motion processing. In controls, the RMS activation over the right temporal cortex was stronger to walker-present than to mask displays ($p < 0.027$), but did not differ in PVL patients. At latencies of 276-340 ms, a three-way ANOVA with factors Stimulus (Walker-present/Mask only), Group (PVL patients/Controls) and Cortical Region (Occipital right, Occipital left, Parietal right, Parietal left, Temporal right, Temporal left, Frontal right, Frontal left) was performed on individual RMS values. This analysis revealed a highly significant effect of region ($F(7,112) = 8.482$, $p < 0.0001$). The main effect of stimulus also was significant ($F(1, 16) = 5.194$, $p < 0.036$). Neither main effect of group, nor interactions between factors were significant. RMS activation over the right frontal cortex in response to walker was weaker in PVL patients than in healthy controls (mean 49.1 fT, SE ± 9.17 ; and mean 79.6 fT, SE ± 13.3 ; $p < 0.031$). By contrast, the activation did not differ between PVL patients and controls in response to the control walker-absent displays. Most striking, in time window of 180-244 ms, in healthy adolescents the visual sensitivity to biological motion, d' , was negatively linked to right temporal RMS activation ($r = -0.778$, $p < 0.01$; Fig. 1A). In healthy adolescents, the sensitivity to body motion dropped with increasing cortical activation over the right temporal cortex. In PVL patients, there was no correlation between the sensitivity to body motion and RMS activation over this region ($r = 0.305$, n.s.; Fig. 1B).

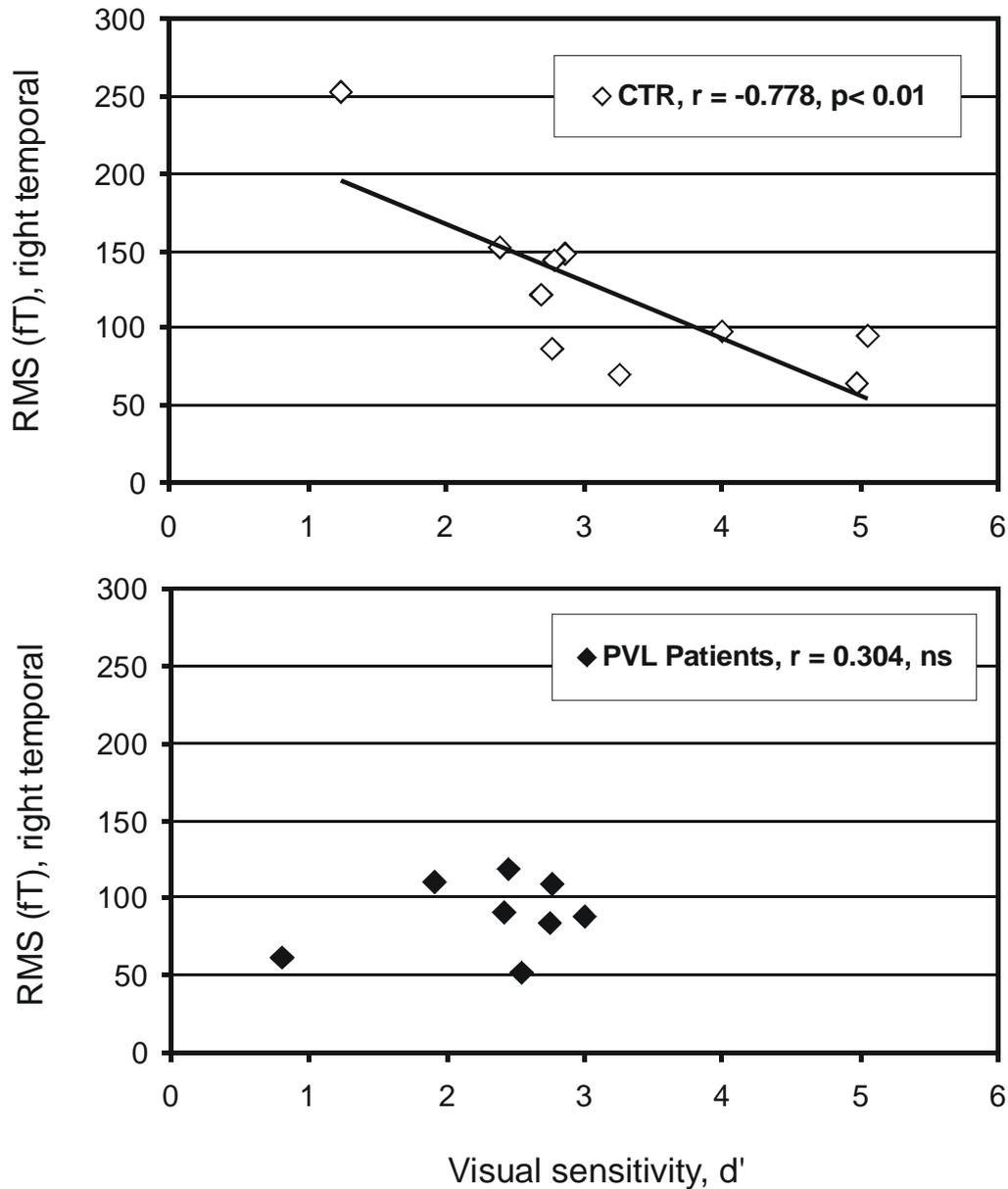


Fig. 1. The visual sensitivity (d') to camouflaged human locomotion plotted against the RMS response (in femtotesla, fT) in healthy adolescents (Pearson product-moment correlation, $r = -0.778$, $P < 0.01$; upper panel, open diamonds), and in PVL patients ($r = 0.304$, n.s.; filled diamonds, lower panel).

Discussion

By combining visual psychophysics with MEG recording, we identify two main effects of PVL on cortical activity. Specific for body motion processing alterations of neuromagnetic response occur over the right temporal cortex at a latency of 180-244 ms and over the right frontal cortex at a latency of 270-344 ms. The early (180-244 ms) evoked neuromagnetic RMS response to body motion over the right temporal region is weaker in PVL patients as compared to healthy controls, whereas right temporal response to the control walker-absent displays does not differ between PVL patients and controls. This indicates that cortical MEG response to body motion over the right temporal region is specifically modulated by early

periventricular lesions. This finding is of substantial value because the right temporal region is not only involved in the visual analysis of body motion (e.g., Pavlova et al., 2004, 2006), but is also known as a key node of the social brain (Allison et al., 2000; Tankerley et al., 2007). Most recent work indicates that healthy perceivers easily reveal socially relevant attributes through body motion (Grèzes et al., 2004; Heberlein et al., 2004). It was shown, for example, that detection of camouflaged human locomotion is modulated by the emotional content of the gait with the highest visual sensitivity to angry point-light walking (Chouchourelou et al., 2006). The right STS is supposed to play a crucial role for this ability (Pelphrey et al., 2004). For example, the increased fMRI response is observed to fearful body motions as compared to emotionally neutral motions in the right temporal pole and STS (Grèzes et al., 2007). These data suggest that PVL patients might be impaired not only on action observation, but also on some aspects of social cognition revealed through body motion. In accord with this assumption, we have recently found that PVL patients exhibit some difficulties in perception and understanding of others' actions, and the extent of periventricular lesions over the right temporal region serves a best predictor of this impairment (Pavlova et al., 2007b).

Most intriguing, in healthy adolescents the RMS response over the right temporal cortex is negatively linked to the visual sensitivity to body motion (see Fig. 1). The strong link between the behavioral measures of performance and the cortical response over the right temporal cortex provides direct support for the particular significance of this cortical area for visual processing of body motion.

The other essential finding is that at later latencies of 276-340 ms, specific for biological motion processing alterations in the RMS response to human locomotion in PVL patients occurred over the right frontal cortex. Periventricular lesions affect proper functioning of the distributed network specialized for body motion processing, and the right frontal cortex appears to be a part of this network. The finding dovetails with earlier fMRI data (Saygin et al., 2004), which show specific for body motion increases in the BOLD signal over the frontal regions. Frontal regions are a part of the mirror neuron system, which has been considered as a core for understanding of actions and intentions of others (Iacoboni and Dapretto, 2006). Specific engagement of the frontal regions in visual processing of body motion found in our study might indicate, therefore, that these cortical regions are also of importance for social perception through body motion and understanding of others' actions. In accord with this, right frontal fMRI activation along with activation of the right temporal pole and STS is greater in response to fearful as compared with neutral whole-body motions (Grèzes et al., 2007). The reduced cortical MEG response to body motion over the right frontal cortex in PVL patients, therefore, might reflect possible deficits in social cognition. High-functioning autistic patients, for example, show no specific fMRI activity in the frontal regions, and brain activity over these regions is inversely related to the severity of impairments in social domain (Dapretto et al., 2006). A dysfunctional mirror neuron system may, therefore, underlie deficits in social perception (Williams et al., 2006).

Taken together, the present data shed light on the role of brain connectivity for visual processing of body motion. We show that disturbances in structural brain connectivity caused by periventricular brain damage lead not only to reduced visual sensitivity to camouflaged body motion, but also to alterations in neuromagnetic RMS response over the temporal and frontal cortices of the right hemisphere.

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