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Cortical networks for motion processing: Effects of focal brain lesions on perception of different motion types

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ABSTRACT

Neuropsychological studies in humans provide evidence for a variety of extrastriate cortical areas involved in visual motion perception. Multiple mechanisms underlying processing of different motion types have been proposed, however, support for cortical specialization has remained controversial so far. We therefore studied motion perception in 23 patients with focal lesions to various cortical areas and considered translational motion, heading from radial flow, as well as biological motion. Patients' detection thresholds were compared with age-specific data from a large healthy control sample ($n = 122$). Elevated thresholds and significant threshold asymmetries between both visual hemifields were defined as deficits. Contrary to prevalent opinion, we found a high prevalence of motion deficits in our sample. Impairment was restricted to a specific motion type in 10 patients, whereas only a single patient showed a deficit for multiple motion types. Functional areas were determined by lesion density plots and by comparison between patients with and without a specific deficit. Results emphasize a dissociation between basic motion processing and processing of complex motion. Anatomical analysis confirmed critical occipito-temporo-parietal areas for perception of translational motion. In contrast, heading perception from radial flow proved to be remarkably robust to most lesions. We exclusively identified the frontal eye fields as a critical structure. Biological motion perception relied on distinct pathways involving temporal, parietal, and frontal areas. Although precise functional roles of identified areas cannot be determined conclusively, results clearly indicate regional specialization for motion types of different complexity. We propose a network for motion processing involving widely distributed cortical areas.

1. Introduction

During the last decades motion processing mechanisms in the visual pathways have been subject to vivid research. There is no doubt that cortical areas beyond striate cortex are specialized in motion processing, however, knowledge about the variety of involved areas and mechanisms still appears fragmentary.

Understanding of visual motion analysis in the human brain relies essentially on neurophysiological studies in the macaque as most appropriate animal model. The first extrastriate area identified as being specifically sensitive to visual motion was area V5/MT (Albright, 1984; Newsome & Pare, 1988; Pasternak & Merigan, 1994; Zeki, 1974). Although research has consequently focused on this key structure, it has soon become evident that motion analysis is not completed in area V5/MT. Multiple interconnections with further cortical regions including parietal, temporal, and frontal areas as well as subcortical structures (Boussaoud, Ungerleider, & Desimone, 1990; Maunsell & Newsome, 1987) indicate that area V5/MT might rather be a gate to complex and specialized motion processing pathways. Comparative studies have shown that differences between human and monkey functional organization increase beyond early visual areas (Orban et al., 2003; Orban, Van Essen, & Vanduffel, 2004). Thus, involvement of higher cortical areas in motion processing requires caution when applying findings in monkeys to humans.

Indeed most importantly, insights from monkeys have prepared the ground for neuropsychological studies on motion perception in humans and guided the focus of research (for review see Zeki, 1991). The famous case report on patient LM complied well with neurophysiological findings in monkeys. LM showed a severe selective

Abbreviations: CT, computer tomography; FEF, frontal eye field; area V3, third visual area; area V5/MT, fifth visual area/middle temporal area; area MST, medial superior temporal area; IFS, inferior frontal sulcus; IPS, intraparietal sulcus; IT, inferotemporal cortex; MRI, magnetic resonance imaging; STS, superior temporal sulcus; RDK, random dot kinematogram; VF, visual field.

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deficit for motion perception which was attributed to her large, bilateral lesion affecting area V5/MT (Baker, Hess, & Zihl, 1991; Hess, Baker, & Zihl, 1989; Zihl, von Cramon, & Mai, 1983; Zihl, von Cramon, Mai, & Schmid, 1991). However, there was also evidence for preserved capacities to analyze motion which pointed to different processing subtypes which might not necessarily depend on area V5/MT (McLeod, Dittrich, Driver, Perrett, & Zihl, 1996; Rizzo, Nawrot, & Zihl, 1995; Shipp, de Jong, Zihl, Frackowiak, & Zeki, 1994). Further studies on motion perception in patients with brain lesions have supported the idea of multiple motion processing mechanisms with specific neural substrates (compare Battelli et al., 2001; Battelli, Cavanagh, & Thornton, 2003; Royden & Vaina, 2004; Vaina, Cowey, Jakab, & Kikinis, 2005; Vaina, Cowey, LeMay, Bienfang, & Kikinis, 2002; Vaina, LeMay, Bienfang, Choi, & Nakayama, 1990; Vaina & Soloviev, 2004a). In line with clinical data, neuroimaging studies in healthy humans have demonstrated an extensive network of motion areas throughout the human brain, including occipito-temporal, occipito-parietal, parietal and frontal regions (Bremmer et al., 2001; Culham, He, Dukelow, & Verstraten, 2001; Sunaert, Van Hecke, Marchal, & Orban, 1999). Although support for cortical specialization has remained controversial so far, the variety of areas responsive to motion suggests multiple processing mechanisms which are presumably associated with different types of motion information. In addition, the still prevalent view of a largely hierarchical organization of the visual pathways (Felleman & Van Essen, 1991) might not apply to motion processing. Functional dissociations found in patient studies have put into question that increasingly more complex motion types are encoded by successive processing stages which rely on input from earlier stages (Beardsley & Vaina, 2006; Vaina et al., 2005). A thorough distinction between motion types appears essential for a better understanding of processing mechanisms. Different extrastriate areas have been discussed to subserve the perception of pure translation in space, radial flow during motion through the environment, or biological motion, i.e. perception of a moving human figure.

In the present study, we investigated which neural substrates are associated with processing of different motion types, namely translational motion, expanding radial flow, and biological motion. Lesion studies in patients offer a unique possibility to improve our understanding of the relationship between brain activity and specific functions (for discussion see Berlucchi, 2004; Rorden & Karnath, 2004). Although the lesion method suffers from some limitations, i.e. insufficient control of lesion location or plasticity processes, there are important advantages which complement the functional imaging approach. On the basis of imaging results, areas involved in a specific processing mechanism can be identified, but it turns out difficult to evaluate the specific relevance of multiple co-activations (Logothetis, 2008). Deficits after lesions provide insights into critically required neural structures. Functional dissociations moreover allow to infer separability of processing pathways and organization principles. Our anatomical interest focused on extrastriate cortical regions, but was not restricted any further. We assumed possible large functional networks because many brain functions might be carried out in a distributed manner (see Farah, 1994). Processing of different motion types was expected to be accomplished by partially dissociable neural systems.

2. Methods

2.1. Participants

2.1.1. Patients

Over a period of 16 months, we considered all ischemic or haemorrhagic stroke patients admitted to the Neurologische Klinik Braunfels, a rehabilitation unit cooperating with the Justus-Liebig-Universität of Gießen. Individual screening sessions were scheduled if (i) medical records described focal cortical lesions visualized by magnetic resonance imaging (MRI) or by computer tomography (CT), (ii) clinical therapists confirmed sufficient cognitive, speech, and motor abilities, (iii) there was no history of psychiatric disorders, and (iv) patients were not taking medications known to interfere with visual functioning. Patients had to accomplish a battery of standard visual tests and those who fulfilled specified criteria were included in the study (see below). We obtained a group of 23 patients whose clinical characteristics are given in Table 1. Assessment by the Edinburgh inventory (Oldfield, 1971) showed right-handedness for all patients except for patient SS who was left-handed. Informed consent was given by all patients according to the Declaration of Helsinki (World Medical Association, 2004). Methods and procedures were approved by the ethics committee of the German Psychological Society.

Table 1
Clinical characteristics of the patient group.

Case	Age	Sex	Lesion type	Lesion location	Lesion-test-interval (weeks)	Visual field
AB	59	M	ICH	Right: O–T	31	
CH	60	F	ICH	Right: O–T	6	
EB	64	F	INF	Right: T	5	
GE	58	M	INF	Left: F	4	
GF	42	M	INF	Right: P	3	
KE	44	M	ICH	Left: F	9	
KK	74	M	INF	Right: O–P	5	
KN	27	F	SAH	Bilateral: F	5	
KS	50	M	INF	Left: F	4	
LL	40	M	INF	Right: P–F	6	
MAS	54	M	INF	Right: T	6	
MB	27	M	INF, ICH	Left: P, Striatum	9	
MDB	50	M	INF	Right: P–T	5	
MS	22	F	SAH, INF	Left: T	7	
PEK	42	F	SAH	Left: T–P	5	
PK	38	M	INF	Left: P	4	
RL	51	F	SAH	Right: T	5	
SB	38	M	ICH	Right: P	6	
SS	39	M	INF	Right: P–F	4	
UJ	45	M	INF	Left: P	4	
UW	53	F	SAH, INF	Right: F	104	
WK	56	M	INF	Right: O–P–T	5	
WR	52	M	INF	Right: P–T	4	

Abbreviations: M: male, F: female; INF: infarction; ICH: intracerebral haemorrhage; SAH: subarachnoidal haemorrhage; F: frontal; P: parietal; T: temporal; O: occipital. *Note*: Only patients KK, RL, SB, and SS showed visual field defects. They were restricted to specific quadrants as indicated by shading. The central radius of 20° was not affected in any patient.

2.1.2. Healthy comparison subjects

For evaluation of patients' motion perception, we were able to access comparison data from 122 healthy subjects ranging in age from 18 to 82 years. Part of the data has been published in Billino, Bremmer, and Gegenfurtner (2008). Healthy subjects were required to have normal or corrected-to-normal vision and to be free from ocular diseases. Any history of neurological or psychiatric disorders was screened out.

2.2. Battery of standard visual tests

Since we were interested in primary deficits for motion perception, we controlled for other visual deficits that could contribute to an indirect impairment. The following battery of standard visual tests facilitated recruitment of patients with extensively normal visual functioning.

2.2.1. Anamnesis of neurovisual deficits

An overview of visual changes experienced by patients was gained by the standardized questionnaire 'Anamnesis of cerebral visual disorders' developed by Kerkhoff, Schaub, and Zihl (1990). It explores patients' subjective complaints, covering for example image fusion, light sensitivity, spatial perception, visuo-motor coordination, reading, face perception. The questionnaire provided a basis for the further screening battery and prevented missing specific deficits.

2.2.2. Visual acuity

Visual acuity was measured binocularly by using a Landolt C chart constructed for near space. We considered visual acuity as normal or corrected-to-normal given a minimum visus of 0.8.

2.2.3. Contrast sensitivity

Contrast sensitivity was evaluated with the Pelli–Robson chart (Pelli, Robson, & Wilkins, 1988). Patients had to achieve a minimum score of 1.65 which corresponds to a contrast sensitivity of 44.7% and is considered to limit the normal range.

2.2.4. Perimetry

Static perimetry was carried out with an Oculus Centerfield perimeter using a threshold strategy. Visual fields up to a radius of 35° were examined separately for each eye. Intact vision for the central radius of at least 20° was required.

2.2.5. Neglect

We administered a line bisection task and a random shape cancellation task (Weintraub, 2000). In the line bisection task, patients were asked to determine the point which divided a given line into two equally long halves. The task was repeated three times. We applied the diagnostic criterion described by Schenkenberg, Bradford, and Ajax (1980). When the marked point deviated more than 14% of line length from the true midpoint, performance was rated as pathologically biased and patients were excluded from the study. In the cancellation task, patients were allowed up to 3 min for completion. A maximum of 2 omissions in either the right or the left half of the sheet was accepted as normal (see Weintraub, 2000).

2.2.6. Stereopsis

To evaluate stereoacuity we administered the Titmus Fly Stereotest (Titmus Optical Co., Petersburg, VA). We chose a very coarse screening criterion because considerable variation in stereoacuity has been described for normal populations (see Garnham & Sloper, 2006; Zaroff, Knutelska, & Frumkes, 2003). Patients had to show depth perception for at least the largest tested disparity of about 3000″ of arc. For the majority of patients in the final group (18 out of 23 patients), we observed stereoacuity below a disparity of 100″ of arc which well corresponds to normal limits.

2.2.7. Color perception

A set of 15 Ishihara plates was used to examine color perception (Ishihara, 1962). Correct identification of at least 10 plates was specified as normal functioning.

2.3. Stimuli

2.3.1. Apparatus

Stimuli were generated by a Dell Latitude 600 at a frame rate of 35 Hz and displayed on a 21 in. Iiyama Vision Master Pro 513 CRT monitor driven by a NVIDIA Quadro NVS 285 graphics card. The monitor resolution was set to 1154 × 864 pixels. White and black pixels had a luminance of 97.5 cd/m^2 and 0.3 cd/m^2, respectively, resulting in a maximum Michelson contrast of 99%. A gamma correction ensured linearity of gray levels.

2.3.2. Random dot kinematograms (RDKs)

RDKs illustrated in Fig. 1 were used to generate translational motion, radial flow, and biological motion. They were composed of white dots with a diameter of 0.1° on a black background.

The translational motion stimulus was presented within a circular aperture with a diameter of 9.4° containing 60 dots. Dots had a limited lifetime of four frames. Dots moving out of the aperture reappeared at a new random position within the aperture. A certain percentage of dots moved in the same horizontal direction, either to the right or to the left, at a speed of 6.6°/s, resulting in coherent motion. The other dots moved in random direction. Signal intensity was defined by percentage of coherently moving dots.

The radial flow stimulus consisted of 100 dots expanding within a rectangular aperture (37.5° × 28.5°) simulating forward motion on a straight path. Dots had a limited lifetime of four frames and dots moving out of the aperture reappeared at random position within the aperture. A certain percentage of dots expanded coherently whereas the remaining dots moved in random direction. The focus of expansion was shifted horizontally 5.6° either to the right or to the left of the center of the field. Speed of expansion increased linearly from the focus of expansion to a maximum speed of 18.6°/s in the periphery. Signal intensity was defined by percentage of coherently expanding dots.

The biological motion stimulus was a point-light walker consisting of eleven dots. It was defined by the point-light walker algorithm described by Cutting (1978). The duration of a stride cycle was set to 1 s which falls in the range for normal human walking as reported by Inman, Ralston, and Todd (1981). The walker subtended a visual angle of 5.3° in height and 2.0° in width, was shown in a sagittal view, and moved in place as if on a treadmill with either left- or right-ward gait. It appeared in a circular aperture with a diameter of 9.4° and was camouflaged by noise dots that moved randomly. Noise dots had a limited lifetime of four frames and reappeared at random position when moving out of the aperture. Signal intensity was defined by percentage of walker dots relative to the total number of dots.

2.4. Procedure

Patients were seated in a darkened room at a distance of 60 cm in front of the monitor. Viewing was binocular and patients' head was stabilized by a chinrest. Patients were instructed to fixate a red dot with a diameter of 0.7° at the center of the screen and to refrain from eye movements. Fixation was visually controlled by the examiner who was positioned behind the setup. Whereas very small eye movements might have been invisible to the examiner, it was straightforward to detect critical deviations from fixation, in particular saccades. If fixation was not maintained, trials were immediately rejected and repeated. Patients were reminded to refixate. Loss of fixation occurred very rarely which was presumably due to the screening of ability and compliance to fixate during perimetry. The background screen was set to minimum luminance. The fixation dot was provided 500 ms before stimulus onset and persisted during stimulus presentation. All stimuli were presented in spatial 2-alternative-forced-choice-paradigms and were displayed for 400 ms.

In the translational motion task, two apertures appeared simultaneously right and left of the fixation dot. Their centers were shifted horizontally to an eccentricity of 7.5°. One aperture contained coherent motion whereas in the other one all dots moved randomly. Patients had to indicate on which side they had perceived coherent motion. In the heading from radial flow task, patients had to detect the direction of heading, i.e. they had to indicate whether the focus of expansion was shifted to the right or to the left of the fixation dot. In the biological motion task, again two apertures appeared simultaneously right and left of the fixation dot with their centers shifted horizontally to an eccentricity of 7.5°. In one aperture, a canonical point-light walker occurred camouflaged by noise dots. In the other one, a scrambled walker and the same amount of noise dots were presented. The scrambled walker consisted also of 11 dots whose motion matched the motion of the dots in the canonical walker. However, dots' spatial position was randomized within the aperture so that the canonical structure was lost. Patients had to indicate at which side they had seen the canonical walker.

Parallel measurement of performance in different motion tasks was intended to differentiate between perceptual capacities for specific motion types. Since the limited resilience of a patient sample had to be considered, we refrained from extensively varying individual parameters of the specific tasks, but rather chose task

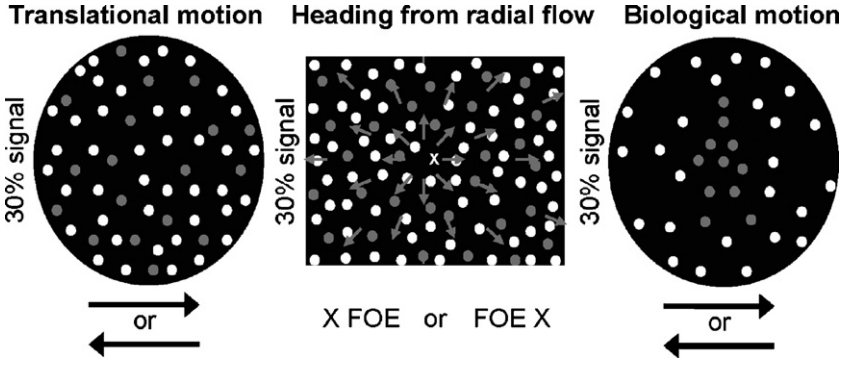

Fig. 1. Static representation of motion random dot kinematograms (RDKs). Signal dots are shown in grey and noise dots in white for clarification. In the actual stimuli, all dots were white. Translational motion was defined as horizontal coherent motion of the signal dots either to the right or to the left. In the radial flow stimulus, signal dots expanded with the focus of expansion (FOE) either right or left of the fixation dot. The latter is replaced here by X to demarcate the fixation sign from the dots. Small grey arrows indicate the motion direction of the signal dots but were not present in the actual stimulus. The biological motion stimulus consisted of a canonical point-light walker embedded in noise dots. It moved as if on a treadmill, facing either to the right or to the left.

configurations typically used in visual motion research. Eccentricity of stimulus presentation allowed for evaluation of visual hemifield differences in performance which we regarded as particularly relevant after unilateral brain damage.

In all motion tasks, responses were entered without temporal constraints directly on the keyboard after stimulus presentation. No feedback was given. Patients started each new trial by pressing the space bar. Before obtaining threshold data, for each task 12 practice trials at different signal intensities were provided, including 4 trials at a 100% signal-to-noise ratio. This opportunity to practice proved to be sufficient for the patients to get used to the specific tasks and to handle the keyboard. We used the method of constant stimuli to measure perception thresholds. Signal intensity in each task was varied by five different noise levels which were chosen to allow for fitting psychometric functions. Each noise level was presented in 32 trials, resulting in a total of 160 trials. The number of correct responses per noise level was recorded.

2.5. Psychophysical data analysis

Patients' detection thresholds for each motion type were determined separately for the contra- and ipsi-lesional visual hemifields. Using the psignifit toolbox in Matlab (Wichmann & Hill, 2001a,b), a Weibull function was fitted to the percentage of correct responses and thresholds were taken for a performance level of 75%. Using a bootstrap procedure, 95% confidence intervals of thresholds were calculated. Threshold asymmetries between both visual hemifields were analyzed by Monte Carlo simulations of the distribution of threshold differences and were evaluated at a significance level of $\alpha = 0.05$.

2.6. Calculation of age-specific predictions

Patients' thresholds were evaluated with reference to thresholds derived from data of healthy comparison subjects. Since several studies have provided evidence for motion sensitivity decline with increasing age (Gilmore, Wenk, Naylor, & Stuve, 1992; Snowden & Kavanagh, 2006; Tran, Silverman, Zimmerman, & Feldon, 1998; Trick & Silverman, 1991; Warren, Blackwell, & Morris, 1989; Wojciechowski, Trick, & Steinman, 1995), we considered age-specific thresholds as a reliable comparison reference. Using regression analysis, individual thresholds were predicted on the basis of age and for each prediction the upper 95% limit was determined. Prediction equations and standard errors of the estimate for the different motion types were the following: translational motion $Y' = 0.26X + 11.00$, $s_e = 8.95$; heading from radial flow $Y' = -0.01X + 9.65$, $s_e = 4.86$; biological motion $Y' = 0.12X + 8.80$, $s_e = 6.48$.

2.7. Lesion analysis

Anatomical analysis of the exact lesion location was based on MRT (including diffusion-weighted, T1, and T2 weighted MRT) scans for 14 patients and on CT (spiral CT) scans for 9 patients. Median time between lesion and imaging used for the present study was 3 days (range between 1 and 64 days) for MRT scans and 9 days (range between 1 and 63 days) for CT scans. The MRIcro software was used for detailed lesion analysis (Rorden & Brett, 2000). Patients' lesions were drawn manually onto transversal slices of the publicly available Montreal Neurological Institute (MNI) brain, a T1-weighted template MRT scan, which is oriented to match the Talairach space (Collins, Neelin, Peters, & Evans, 1994; Talairach & Tournoux, 1988). Slices chosen for mapping corresponded to Talairach z-coordinates 60, 50, 40, 32, 24, 16, 8, 0, −8, −16, and −24 mm. In order to ease comparison, lesions of the left hemisphere were flipped so that all lesions were mapped onto the right hemisphere of the template.

Lesions of patients who showed a deficit in a specific motion task were overlaid to explore regions that might be functionally important. However, simple overlay plots can be misleading because they also highlight regions that are merely more susceptible to damage, e.g. due to their vasculature (see Rorden & Karnath, 2004). We therefore qualified simple overlay plots by a masking procedure. Patients with normal perceptual performance in a specific task were considered as control group and their superimposed lesions provided a mask. By applying this mask to the simple overlay plot we obtained an illustration of damage unique to a given deficit. A more sophisticated statistical analysis of the association between lesion location and probability of a specific deficit was not applicable due to overall small and extremely differing group sizes, respectively.

3. Results and discussion

A summary of perceptual thresholds in the different motion tasks for each patient is given in Table 2. Thresholds exceeding the 95% limit of the age-specific prediction as well as significant threshold asymmetries between both visual hemifields were considered as deficient and are highlighted in bold.

3.1. Translational motion

3.1.1. Psychophysical data

We found three patients with deficient perception of translational motion (see Table 2). Their performance is illustrated in Fig. 2A. Thresholds of patients MAS and SB met the age-specific predictions, however, a significant performance difference between both visual hemifields was determined. Thresholds for the contralesional hemifield were higher than those for the ipsilesional hemifield. Patient WK showed an elevated threshold for the contralesional hemifield and the threshold for his ipsilesional hemifield

Table 2
Summary of patients' perceptual thresholds in different motion tasks.

Case	Translational motion			Heading from radial flow			Biological motion		
	Contralesional		Ipsilesional	Contralesional		Ipsilesional	Contralesional		Ipsilesional
AB	21.3	||	24.7	11.5	||	14.4	**31.1***	||	**25.3**
CH	21.4	||	27.9	8.6	||	6.7	23.4	||	17.9
EB	34.9	||	37.0	10.8	||	5.8	**33.5***	||	**25.8**
GE	15.0	||	20.5	4.1	||	5.8	**41.6***	||	**39.7***
GF	20.5	||	24.9	8.8	||	9.6	13.1	||	11.1
KE	37.0	||	27.3	9.1	||	11.3	14.0	||	14.8
KK	30.8	||	33.5	7.3	||	6.4	14.3	||	11.1
KN	10.2	||	9.6	5.8	||	5.0	**19.1**	||	**24.1***
KS	10.1	||	11.6	10.8	||	9.0	11.8	||	15.8
LL	17.6	||	24.9	4.5	||	6.5	**27.1***	||	**31.8***
MAS	**28.1**	>	**11.4**	10.0	||	9.5	**29.6***	||	**30.4***
MB	15.9	||	13.7	4.5	||	4.6	13.6	||	9.2
MDB	14.6	||	10.0	4.6	||	5.8	13.7	||	12.9
MS	28.1	||	26.8	12.9	||	14.7	**23.6***	||	**27.0***
PEK	25.2	||	28.6	14.3	||	12.2	13.0	||	10.4
PK	8.9	||	10.2	4.5	||	4.8	13.5	||	11.8
RL	18.7	||	16.4	14.1	||	10.7	17.2	||	19.7
SB	**30.0**	>	**14.7**	12.1	||	14.2	18.1	||	5.2
SS	16.1	||	12.6	7.4	||	5.8	16.6	||	13.5
UJ	27.3	||	26.8	16.3	||	9.4	**23.3**	||	**25.4***
UW	25.6	||	31.3	**20.5***	||	**7.0**	15.4	||	25.4
WK	**40.9***	||	**36.6**	9.4	||	6.4	22.2	||	20.7
WR	18.8	||	23.9	12.0	>	10.1	7.8	||	6.7

Note: Detection thresholds are given for the contralesional visual field and the ipsilesional visual field. Thresholds which exceed the upper 95% limit of the age-specific prediction are marked by an asterisk. Asymmetries between thresholds in both visual hemifields have been determined by Monte Carlo tests with a significance level of 0.05; || indicates equivalence between thresholds, > indicates asymmetry between thresholds. Elevated thresholds and asymmetries between thresholds are defined as deficits and are highlighted in bold.

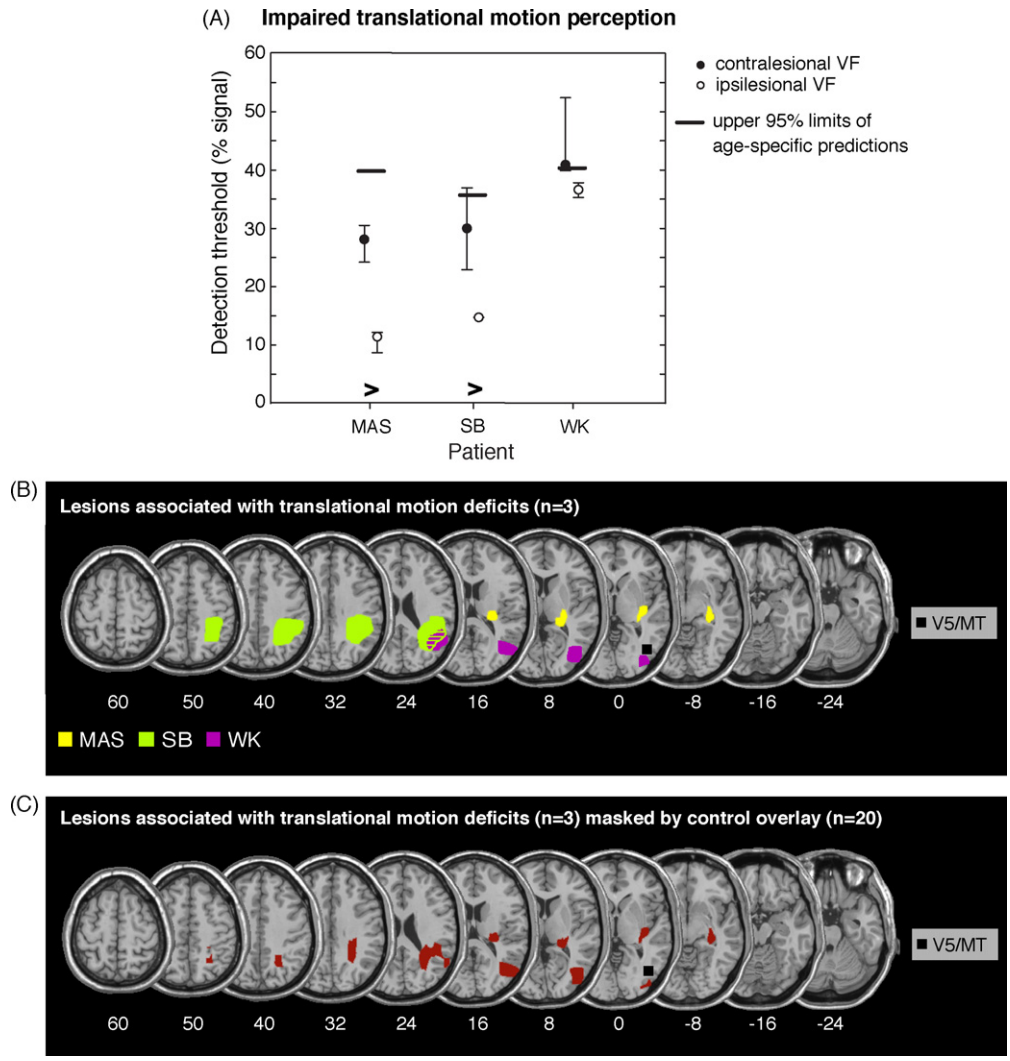

Fig. 2. (A) Deficits for translational motion perception. On the *x*-axis, patients who showed elevated thresholds or asymmetries between thresholds in both visual hemifields are presented. The *y*-axis indicates detection threshold in % signal for a performance level of 75%. Solid circles symbolize thresholds in the contralesional visual field (VF); open circles symbolize thresholds in the ipsilesional visual field (VF). Error bars depict 95% confidence intervals of thresholds. For each patient, the bold black horizontal line indicates the upper 95% limit of the age-specific prediction. Significant asymmetries between thresholds in both visual hemifields are marked by a greater-than sign. (B) Lesion plot of the patients who showed a deficit for translational motion perception ($n = 3$). Lesions of individual patients are depicted by different colors. (C) Overlay lesion plot of patients with translational motion deficit ($n = 3$) masked by superimposed lesions of control patients with normal translational motion perception ($n = 20$). Talairach *z*-coordinates (Talairach & Tournoux, 1988) of each transverse section are given. Supposed critical functional locations are marked: V5/MT, fifth visual area/middle temporal area (Watson et al., 1993).

scarcely lay below the critical age-specific prediction. No significant asymmetry between both hemifields was observed.

3.1.2. Lesions

Lesions of patients MAS, SB, and WK are depicted in Fig. 2B. All three patients suffered from right-sided lesions. Patient MAS showed a very small lesion in the medial temporal lobe, but moreover the thalamus was affected marginally. In patient SB, we determined a high parietal lesion bordering ventrally the superior temporal lobe. Patient WK's lesion was located primarily in the occipital lobe, but covered the occipito-temporo-parietal junction. Lesions of patients SB and WK overlapped in the temporo-parietal area.

Lesions of the 20 patients without deficit for translational motion perception were superimposed and applied as a mask to lesions of patients MAS, SB, and WK. Fig. 2C gives the masked overlay plot. The majority of control patients had lesions at locations different from those of lesions associated with translational motion deficits. The masking procedure specified that the complete lesion of patient MAS and lesions in the occipito-temporo-parietal area were unique to impaired translational motion perception.

3.1.3. Discussion

We found a deficit for translational motion perception in 3 out of 23 patients. Frequency of occurrence corresponds well to findings in previous patient studies concerned with translational motion detection in RDKs (compare Braun, Petersen, Schonle, & Fahle, 1998; Schenk & Zihl, 1997a). Overall, deficits for translational motion perception can be considered as rare after cortical lesions. Previous neuropsychological studies have confirmed area V5/MT as critical functional region for translational motion analysis (Dumoulin et al., 2000; Plant, Laxer, Barbaro, Schiffman, & Nakayama, 1993; Schenk & Zihl, 1997a; Sunaert et al., 1999; Vaina, Cowey, Eskew, LeMay, & Kemper, 2001; Zihl et al., 1983). Since this structure represents a small visual area, it might be expected that circumscribed vascular events are rather unlikely to affect the critical region. Moreover, area V5/MT at the occipito-temporo-parietal junction potentially receives blood supply by branches of the middle as well as of the

posterior cerebral artery. Loss of blood supply by a single artery might be compensated to some degree. Thus, functionality of area V5/MT might be relatively robust to vascular events.

Analysis of lesion sites in patients with deficits for translational motion perception yielded ambiguous results. Location of human area V5/MT has been determined by several imaging studies (Dumoulin et al., 2000; Sunaert et al., 1999; Watson et al., 1993) and reported coordinates converge to the region marked in Fig. 2B and C. Patient WK's lesion borders on the critical location and thus his deficit appears in line with predictions. In contrast, patient SB's lesion site that we identified as unique to deficient translational motion perception is located more dorsally in the parietal lobe. Although the parietal lobes comprise areas highly specialized for attentional processes (see Posner & Dehaene, 1994), we consider it as unlikely that his perceptual deficit for translational motion can be attributed to disturbed attentional processes. Patient SB showed no signs of impaired attention during the screening procedure nor was his perception of any other motion type deficient. We suppose that the deficit points to interindividual differences in the position of area V5/MT in stereotaxic space. Watson et al. (1993) have reported interindividual variation of specific coordinates by almost 30 mm. Assuming a pronounced deviation from group data, patient SB's lesion might still affect his individual functional V5/MT region. For patient MAS, the association between lesion site and observed deficit appears to be quite different. Areas in the medial temporal lobe are generally not considered as motion responsive (Culham et al., 2001; Sunaert et al., 1999). His lesion presumably affected the pulvinar of thalamus. Damage to the posterior thalamus is known to contribute to contralateral inattention (compare Kooistra & Heilman, 1989; Mesulam, 1999). Recent studies have shown that in particular damage to the pulvinar could contribute to an impaired visual representation for complex visual information (Cotton & Smith, 2007) or attentional constraints (Kastner & Pinsk, 2004; Shipp, 2004; Van Essen, 2005). Thus, we conjecture that patient MAS' deficit might be due to a subtle attentional deficit. Asymmetries in his visual performance probably did not become evident in the screening procedure because stimuli were of lower complexity accordingly. Indeed, interpretation of his deficit has to remain strongly tentative because a detailed assessment of attentional capacities was not covered by our screening procedure.

In consideration of the retinotopic organization of area V5/MT (see Born & Bradley, 2005; Huk, Dougherty, & Heeger, 2002), patients with lesions in this area can be expected to show contralateral perceptual deficits. Several patient studies have confirmed a deficit for motion perception in the contralesional visual hemifield after unilateral posterior lesions (Braun et al., 1998; Plant et al., 1993; Vaina, Cowey, et al., 2001). However, there is also evidence for non-retinotopic deficits and there are speculations about differential organizational principles within substructures of area V5/MT (Barton, Sharpe, & Raymond, 1995; Schenk & Zihl, 1997a). Deficit profiles of our patients SB and WK, who presumably suffered from damage to area V5/MT, mirror these previous findings. Whereas patient SB's performance asymmetry between both hemifields supports a retinotopic organization, patient WK showed a non-retinotopic deficit. Though lesion analysis does not allow for further differentiation between damage to substructures of area V5/MT, data points to retinotopic as well as to non-retinotopic organizational principles in visual motion areas (compare d'Avossa et al., 2007; Gardner, Merriam, Movshon, & Heeger, 2008).

Finally, observed deficits for translational motion perception can be discussed with regard to functional plasticity. After small lesions to area V5/MT, monkeys recover from motion perception deficits within days to a few weeks (Newsome & Pare, 1988; Rudolph & Pasternak, 1999; Yamasaki & Wurtz, 1991). In human patients, corresponding recovery processes have rarely been investigated and seem to take place much more slowly. Braun et al. (1998) have longitudinally studied a patient who completely recovered from motion perception deficits within 20 months. Schenk and Zihl (1997a) have concluded from cross-sectional data that recovery does not occur within the first months after lesion. Since lesion-test-intervals for our impaired patients did not exceed six weeks, persistent deficits for translational motion perception can be considered as in line with earlier studies. It appears noteworthy that we observed another patient, namely patient AB, who suffered from a lesion presumably affecting area V5/MT (see Fig. 4B), but who showed no deficit for translational motion perception when his thresholds were determined 31 weeks after lesion. Thus, one might speculate about a rapid recovery process.

3.2. Heading from radial flow

3.2.1. Psychophysical data

A deficit for heading perception from radial flow was only determined in patient UW (see Table 2). She showed an elevated threshold for perception of heading to the contralesional hemifield, but not for perception of heading to the ipsilesional hemifield. Analysis of the threshold difference yielded a significant asymmetry between both visual hemifields. Fig. 3A shows patient UW's performance.

3.2.2. Lesion

Fig. 3B demonstrates the location of patient UW's lesion: it was extensive and covered wide parts of the right anterior frontal cortex. It spanned from superior areas to the frontal pole.

Lesions of 22 control patients who showed no deficit for heading perception from radial flow hardly overlapped with UW's lesion. Thus, the masked overlay plot shown in Fig. 3C retained a rather large frontal region that was unique to the deficit for heading perception from radial flow.

3.2.3. Discussion

Although our 23 patients demonstrated lesions at quite diverse cortical locations, heading perception from radial flow was only impaired in patient UW. Processing of this specific motion type proved to be remarkably robust to most lesions. Our data appears congruent with the lack of neuropsychological case reports on deficits for heading perception. Patient studies considering heading from radial flow rather have found preserved perceptual capacities despite impaired performance in other motion tasks indicating a dissociation from low level and other high level types of motion processing (Beardsley & Vaina, 2005; Royden & Vaina, 2004). We suppose that the robustness of heading perception observed in brain-lesioned patients is due to a network of distributed functional areas. Multiple processing pathways might allow for compensation of damage to circumscribed regions.

The anatomical basis of radial flow analysis and heading perception has remained elusive so far. In monkeys, selectivity for expanding radial flow emerges at the level of area MST which receives strong input from area V5/MT (Duffy & Wurtz, 1991a,b; Lappe, Bremmer, Pekel, Thiele, & Hoffmann, 1996; Saito et al., 1986). However, further cortical regions contribute to flow analysis. Representations of expanding radial flow have been described in the superior polysensory area (Siegel & Read, 1997), the ventral intraparietal cortex (Bremmer, Duhamel, Ben Hamed, & Graf, 2002; Konen & Kastner, 2008; Schaafsma & Duysens, 1996; Schaafsma, Duysens, & Gielen, 1997), and the frontal eye fields (Xiao, Barborica, & Ferrera, 2006). The finding of heterogeneous neural substrates is mirrored in human imaging studies. There is consensus on flow analysis in areas V5/MT and MST (Greenlee, 2000; Morrone et al., 2000; Ptito, Kupers, Faubert, & Gjedde, 2001; Smith, Wall, Williams, & Singh, 2006; Wunderlich et al., 2002), but further activation by radial flow analysis has been found for various distributed

structures. Results support the view that heading perception engages a wide network of neural regions (de Jong, Shipp, Skidmore, Frackowiak, & Zeki, 1994; Field, Wilkie, & Wann, 2007; Orban, Sunaert, Todd, Van Hecke, & Marchal, 1999; Peuskens, Sunaert, Dupont, Van Hecke, & Orban, 2001; Vaina & Soloviev, 2004b; Wall & Smith, 2008).

Lesion analysis in our only patient who showed impaired heading perception pointed to crucial functional significance of frontal regions. This finding appears unexpected since predominantly temporal and parietal areas as marked in Fig. 3B and C have been described to be involved in heading perception (compare Vaina & Soloviev, 2004b). We conjecture that patient UW's lesion includes the right FEF. The FEF is primarily considered to be involved in saccade generation and allocation of spatial attention towards the contralateral visual hemifield (Barborica & Ferrera, 2004; Kustov & Robinson, 1996; Moore & Fallah, 2001; Rizzolatti, Matelli, & Pavesi, 1983; Robinson & Fuchs, 1969; Schafer & Moore, 2007). However, there is a growing body of evidence that the FEF contributes also to many aspects of visual processing (see e.g. Campana, Cowey, Casco, Oudsen, & Walsh, 2007; Silvanto, Lavie, & Walsh, 2006). In particular, Xiao et al. (2006) have reported a preference for radial motion in motion-sensitive neurons in the FEF. We suggest that patient UW's deficit for heading perception from radial flow provides further support for functional significance of the FEF in radial flow analysis. Unilateral damage seems to impair perception of heading towards the contralateral visual hemifield. The observation of a contralateral retinotopic organization in the FEF agrees with recent findings by Hagler and Sereno (2006) who explored spatial maps in frontal and prefrontal cortex. Focusing on attentional and working memory processes, they provided evidence of a strong contralateral preference in the FEF.

Although rough assessment of attentional capacities in the screening procedure does not allow us to exclude spatial inattention as explanation for patient UW's deficit completely, we consider this possibility as rather unlikely. Patient UW showed no deficits, particularly performance asymmetries, in both other motion tasks whose stimulus configuration put relatively higher demands on allocation of spatial attention (compare Table 2).

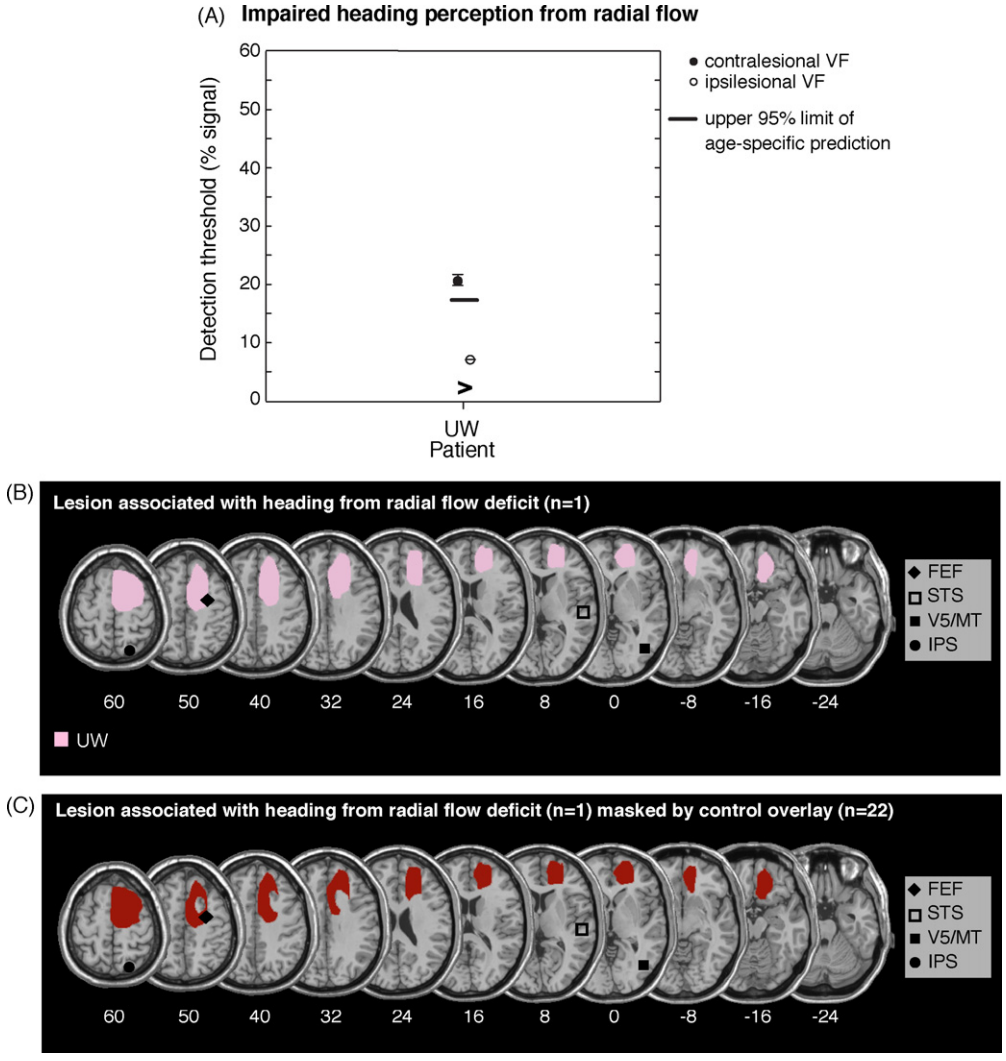

Fig. 3. (A) Deficits for heading perception from radial flow. On the *x*-axis, the patient who showed a threshold elevation and an asymmetry between thresholds in both visual hemifields is presented. The *y*-axis indicates detection threshold in % signal for a performance level of 75%. The solid circle symbolizes the threshold in the contralesional visual field (VF); the open circle symbolizes the threshold in the ipsilesional visual field (VF). Error bars depict 95% confidence intervals of thresholds. The bold black horizontal line indicates the upper 95% limit of the age-specific prediction. The significant asymmetry between thresholds in both visual hemifields is marked by a greater-than sign. (B) Lesion plot of the patient who showed a deficit for heading perception from radial flow ($n = 1$). The lesion is depicted by light pink. (C) Overlay lesion plot of the patient with heading from radial flow deficit ($n = 1$) masked by superimposed lesions of control patients with normal heading perception from radial flow ($n = 20$). Talairach *z*-coordinates (Talairach & Tournoux, 1988) of each transverse section are given. Supposed critical functional locations are marked: FEF, frontal eye field (Orban et al., 1999); STS, superior temporal sulcus (Howard et al., 1996); V5/MT, fifth visual area/middle temporal area (Watson et al., 1993); IPS, intraparietal sulcus (Peuskens et al., 2001).

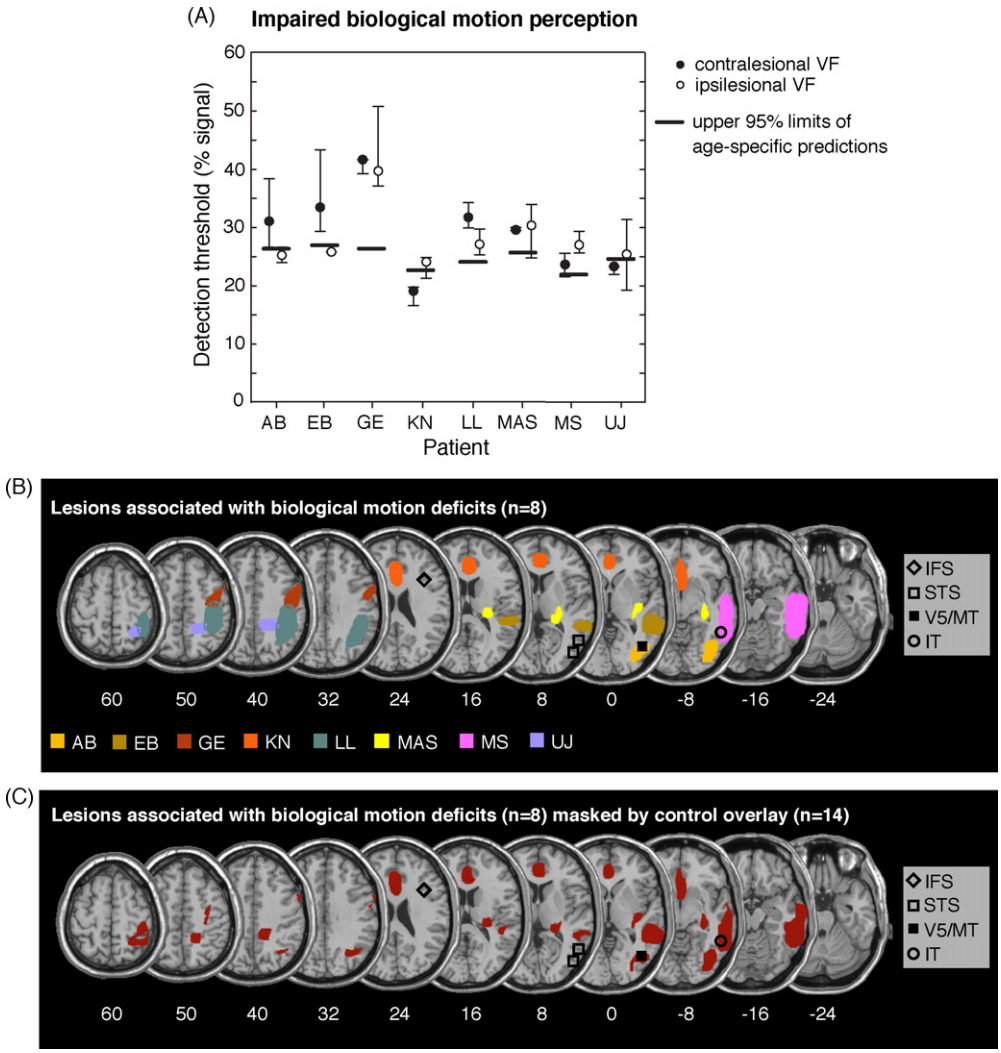

Fig. 4. (A) Deficits for biological motion perception. On the *x*-axis, patients who showed elevated thresholds are presented. The *y*-axis indicates detection threshold in % signal for a performance level of 75%. Solid circles symbolize thresholds in the contralesional visual field (VF); open circles symbolize thresholds in the ipsilesional visual field (VF). Error bars depict 95% confidence intervals of thresholds. For each patient, the bold black horizontal line indicates the upper 95% limit of the age-specific prediction. No patient showed an asymmetry between thresholds in both visual hemifields. (B) Lesion plot of the patients who showed a deficit for biological motion perception ($n = 8$). Lesions of individual patients are depicted by different colors. (C) Overlay lesion plot of patients with biological motion deficit ($n = 8$) masked by superimposed lesions of control patients with normal biological motion perception ($n = 15$). Talairach *z*-coordinates (Talairach & Tournoux, 1988) of each transverse section are given. Supposed critical functional locations are marked: IFS, inferior frontal sulcus (Saygin et al., 2004); IT, inferotemporal cortex (Saygin et al., 2004); STS, superior temporal sulcus (Grossman et al., 2000; Saygin et al., 2004); V5/MT, fifth visual area/middle temporal area (Watson et al., 1993).

With regard to compensation and plasticity processes, patient UW's deficit suggests that the FEF makes a necessary contribution to heading perception. Since her lesion occurred approximately 2 years before testing, compensation by alternative processing pathways and functional plasticity have to be considered as limited.

3.3. Biological motion

3.3.1. Psychophysical data

Deficits for biological motion perception were found in eight patients as shown in Fig. 4A (compare also Table 2). Four patients, i.e. patients GE, LL, MAS, and MS, showed elevated threshold in both visual hemifields. Threshold elevation was restricted to the contralesional hemifield in patients AB and EB, whereas it was observed only for the ipsilesional hemifield in patients KN and UJ. However, normal thresholds of the latter four patients lay only scarcely below the upper limit of age-specific predictions. Elevated thresholds were not associated with performance asymmetries between both visual hemifields.

3.3.2. Lesions

Location of lesions associated with biological motion deficits is illustrated in Fig. 4B. Note that all lesions are mapped onto the right hemisphere for the sake of comparability. Right-sided lesions were determined in patients AB, EB, LL, and MAS, whereas patients GE, MS, and UJ showed left-sided lesions. Patient KN's lesion was classified as bilateral because it affected frontal areas surrounding the anterior corpus callosum. Size of six lesions could be considered as being rather small, while the two patients LL and MS suffered from more extensive damage. Critical lesion sites barely overlapped and involved quite diverse cortical areas in the temporal, parietal, and frontal lobes.

The diversity of functional lesion locations is supported by the masked overlay plot given in Fig. 4C. Lesions of the 14 control patients who showed normal biological motion perception provided a mask which was applied to the superimposed lesions of patients with deficient biological motion perception. Lesion sites unique to biological motion deficits were determined in the superior parietal lobe, in the lateral temporal lobe, near the

occipito-temporo-parietal junction as well as in the medial frontal lobe.

3.3.3. Discussion

Our data supports the notion that deficits for biological motion perception represent a relevant problem after cortical lesions. Almost 35% of studied patients suffered from elevated perceptional thresholds for this motion type. Previous patient studies have only provided few case reports showing deficient biological motion perception (Battelli et al., 2003; Cowey & Vaina, 2000; Schenk & Zihl, 1997b; Vaina & Gross, 2004). Higher frequency of deficit occurrence might be explained by demand characteristics of the biological motion task in the present study. We used simultaneous presentations of canonical and scrambled point-light walkers camouflaged by dynamic noise. Previous studies either did not require direct comparison between canonical and scrambled structures or did not manipulate noise. Thus, our task might have been more sensitive to perceptual deficits (compare also Saygin, 2007). Pronounced vulnerability appears consistent with the complexity of biological motion analysis. It requires not only correct perception of form and motion signals, but also integration of derived information. Moreover, there is recent evidence that biological motion perception is subject to top-down influences and is modulated by attention (Battelli et al., 2003; Pavlova, Birbaumer, & Sokolov, 2006). Diverse functional constraints might be expected to interfere with biological motion processing.

Though subject to vivid research during the last years, specific neural substrates of biological motion analysis are still under debate and particularly contributions of structures beyond early extrastriate areas appear not well understood. Imaging studies have shown activation of area V5/MT during biological motion analysis, but have emphasized involvement of a widespread network of brain areas (e.g. Michels, Lappe, & Vaina, 2005). Particular functional significance has been attributed to the temporal and frontal areas marked in Fig. 4B and C. Since biological motion perception requires analysis of form and motion, it has been proposed that it involves both ventral and dorsal systems and in particular their confluence in posterior STS (Grossman, Battelli, & Pascual-Leone, 2005; Grossman & Blake, 2002; Grossman et al., 2000; Saygin, 2007; Saygin, Wilson, Hagler, Bates, & Sereno, 2004; Vaina, Solomon, Chowdhury, Sinha, & Belliveau, 2001). Activity in the ventrolateral inferotemporal (IT) cortex is supposed to reflect necessary form analysis (Saygin et al., 2004). Finally, observed frontal activity might indicate contributions of action observation networks (Saygin, 2007; compare Fabbri-Destro & Rizzolatti, 2008; Rizzolatti & Craighero, 2004). Kourtzi, Krekelberg, and Van Wezel (2008) have emphasized that linking form and motion require complex interactions between early and higher visual areas. Functional dissociations between biological motion perception and perception of other motion types are suggested by some lesion studies. Biological motion perception has been found selectively preserved as well as selectively impaired in patients (Battelli et al., 2003; Cowey & Vaina, 2000; McLeod et al., 1996; Schenk & Zihl, 1997b; Vaina et al., 2002; Vaina & Gross, 2004; Vaina et al., 1990).

Critical lesion sites in our patient sample confirm some of the reported findings and add additional details. With regard to the STS, we have not determined exactly matching lesions. However, patient EB's lesion was localized in a directly bordering region which might mirror interindividual variations of functional anatomy. Observed lesion sites in the temporal lobe appear in line with imaging results. In particular, patient MS's lesion affected the region in the ventrolateral IT cortex that has been found activated by biological motion (Saygin et al., 2004). In contrast, patient AB's lesion covered posterior parts of the temporal lobe. His perceptual deficit for biological motion points to significance of further temporal regions. Frontal contributions to biological motion processing are not supported by our data. Patient GE's lesion was located in the left superior frontal lobe, but masking out lesions of control patients without perceptual deficit for biological motion only left minor critical spots. Thus, damage appears to be not consistently associated with a deficit and eludes further functional interpretation. Patient KN's lesion persists after the masking procedure, but we conjecture that her deficit might rather be due to disruption of interhemispheric transfer via the anterior corpus callosum than to lesioned frontal tissue. Multiple cortical areas are involved in biological motion perception and at least for some areas there is evidence of lateralization (compare Saygin et al., 2004). Effective processing might therefore require exchange between both hemispheres. Moreover, we have found critical parietal lesions. Indeed, there have been few patient and imaging studies suggesting functional contributions of parietal areas (Battelli et al., 2003; Bonda, Petrides, Ostry, & Evans, 1996; Claeys, Lindsey, De, & Orban, 2003; Schenk & Zihl, 1997b; Vaina, Solomon, et al., 2001). Parietal lesion sites in our patients LL and UJ are roughly in line with anatomical data from previous case studies. We conclude that results provide further evidence for biological motion processing in the parietal lobes. However, functional details explaining absent activation in most imaging studies (Grossman & Blake, 2002; Grossman et al., 2000; Michels et al., 2005; Saygin, 2007; Saygin et al., 2004) remain to be clarified. As discussed above (see Section 3.1), patient MAS's deficits should be considered in the context of his thalamus lesion and a potential attentional deficit. Finally, our patient data does not provide evidence for a hemispheric dominance for biological motion perception. The number of patients was insufficient to allow for studying such differences in detail. However, we did not observe a striking difference between occurrence of deficits for biological motion perception after left-sided and right-sided lesions.

Deficits for biological motion perception were determined as non-retinotopic in all patients. Previous patient studies have not considered performance differences between both visual hemifields (Battelli et al., 2003; Schenk & Zihl, 1997b; Vaina et al., 2002; Vaina & Gross, 2004; Vaina et al., 1990). Biological motion processing involves higher visual areas where receptive fields of neurons become increasingly large (Gattass et al., 2005; Serences & Yantis, 2007). Thus, bilateral deficits might reflect non-retinotopic organization of functional areas. Indeed, Saygin and Sereno (2008) have reported retinotopic organization of higher visual areas, but retinotopy was primary driven by attention not by stimulus.

To conclude, there is little knowledge on recovery from perceptual deficits for biological motion. There are few case reports on patients showing impaired biological motion perception and lesion-test-intervals have generally been only few months (e.g. Schenk & Zihl, 1997b). Intervals for our patients with deficits also ranged between 4 and 31 weeks. Deficits obviously persist over a period of some weeks, but long-term outcome remains to be explored. It might be speculated that multiplicity of involved functional areas bears potential for compensation and reorganization.

4. Conclusion

Studying perception of different motion types in patients with focal cortical lesions, we aimed to provide further insights into functional dissociations and specific processing pathways. The focus was put on widely distributed extrastriate lesions. We determined perceptual thresholds for translational motion, heading from radial flow, and biological motion in the same patients. Furthermore, we were able to evaluate patients' performance in consideration of age-specific predictions.

Although individual lesions affected quite diverse cortical areas, we determined a high incidence of constraints on motion pro-

cessing. Almost 50% of our patients showed deficient perception for a specific motion type. Moreover, clearly dissociated deficits indicate several different pathways for processing specific motion information and regional specialization. An extensive network of motion areas has as well been suggested by neuroimaging studies (Culham et al., 2001; Sunaert et al., 1999). Various cortical areas beyond the classical motion area V5/MT contribute to analysis of motion information and damage to these areas can obviously cause significant perceptual deficits. Since lesions in our patient sample inevitably also involved white matter, observed deficits might be associated with either direct damage of cortical areas or disabling due to disconnection. On the basis of our methods, we cannot differentiate sufficiently between these possible underlying mechanisms. However, selective deficits for different motion types in our patient sample definitely support specialized processing networks that are dissociated from each other. It seems noteworthy that Konen and Kastner (2008) have recently reported motion-selective responses in various cortical areas regardless of motion type, i. e. planar, circular, and radial motion. We suggest that results might not be in conflict, but rather complement one another. Beyond imaging results, patient studies add important information on functional necessity of specific brain regions (compare Berlucchi, 2004; Rorden & Karnath, 2004).

Our results confirm that translational motion perception is accomplished at early extrastriate processing stages. However, adequate translational motion analysis is not required for perception of more complex motion types. Despite perceptual deficits for translational motion, patients WK and SB showed normal perception of heading from radial flow and biological motion. This result corresponds to earlier case reports (Beardsley & Vaina, 2005; McLeod et al., 1996; Royden & Vaina, 2004; Vaina et al., 2002, 1990).

Preserved heading perception from radial flow in the presence of damage to area V5/MT points to the complexity of optic flow analysis. Indeed, area MST which has been identified as selectively responsive to optic flow receives strong input from area V5/MT (Duffy & Wurtz, 1991a,b; Lappe et al., 1996). Heading perception though involves a wide range of other cortical areas which might receive alternative input (Bremmer et al., 2001, 2002; compare Vaina & Soloviev, 2004b). Given the distribution of functional areas, the existence of multiple pathways for processing heading information seems plausible. We suggest that pathways bypassing area V5/MT might be sufficient to determine heading from radial flow. Alternative processing pathways could ensure robust heading perception, which bears high ecological relevance, even in the presence of constraints on the visual system. The observation that only patient UW demonstrated a deficit for heading perception might be considered as being in line with this speculation.

Deficit profiles furthermore provide evidence for the notion that biological motion analysis does not require intact processing of translational motion in area V5/MT. This finding agrees with case reports on patients with normal biological motion perception despite impaired basic motion analysis (McLeod et al., 1996; Vaina et al., 2002, 1990). On the other hand, perceptual deficits for biological motion in our patient sample have occurred despite intact translational motion perception. This dissociation has also been described by some earlier case reports (Battelli et al., 2003; Cowey & Vaina, 2000; Schenk & Zihl, 1997b; Vaina & Gross, 2004). Our data contributes to the ongoing debate on which signals drive biological motion perception (compare Garcia & Grossman, 2008; Lange, Georg, & Lappe, 2006). Granted, our focus on motion perception limits the scope of conclusions on further relevant signals. However, results suggest that biological motion analysis might be more vulnerable to a disruption of form processing or integrating capacities than to impaired basic motion perception. Motion analysis at the level of area V5/MT might neither be sufficient nor necessary for biological motion perception.

Since the first report on famous 'motion-blind' patient LM (Zihl et al., 1983), numerous neuropsychological and neuroimaging studies have improved our understanding of motion processing in the human brain. There is evidence that a variety of cortical areas is involved in motion analysis and that processing of different motion types might be dissociated. However, the functional contributions of structures beyond the early extrastriate area V5/MT to motion perception have remained controversial. Although deficits for motion perception are presumably present in many patients suffering from cortical lesions, they might not be considered sufficiently in clinical practice so far (compare Kerkhoff, 2000).

We propose a network for motion processing involving a multiplicity of critical areas. Since neural segregation appears determined rather by type of motion information than by complexity, a hierarchical organization of motion processing should be put into question. AreaV5/MT might represent a highly specialized visual processing stage, but not a key area for processing of complex motion types. Analysis of specific motion types might rely on specialized pathways connecting widely distributed cortical areas. Indeed, these pathways are ultimately not only defined by their involved cortical substrates identified in the present study, but also by the complexity of forward and feedback connections between these substrates. Functional characteristics of white matter connections in visual motion processing remain to be explored.

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References

Albright, T. D. (1984). Direction and orientation selectivity of neurons in visual area MT of the macaque. *Journal of Neurophysiology*, *52*, 1106–1130.

Baker, C. L., Hess, R. F., & Zihl, J. (1991). Residual motion perception in a "motion-blind" patient, assessed with limited-lifetime random dot stimuli. *Journal of Neuroscience*, *11*, 454–461.

Barborica, A., & Ferrera, V. P. (2004). Modification of saccades evoked by stimulation of frontal eye field during invisible target tracking. *Journal of Neuroscience*, *24*, 3260–3267.

Barton, J. J., Sharpe, J. A., & Raymond, J. E. (1995). Retinotopic and directional defects in motion discrimination in humans with cerebral lesions. *Annals of Neurology*, *37*, 665–675.

Battelli, L., Cavanagh, P., Intriligator, J., Tramo, M. J., Henaff, M. A., Michel, F., et al. (2001). Unilateral right parietal damage leads to bilateral deficit for high-level motion. *Neuron*, *32*, 985–995.

Battelli, L., Cavanagh, P., & Thornton, I. M. (2003). Perception of biological motion in parietal patients. *Neuropsychologia*, *41*, 1808–1816.

Beardsley, S. A., & Vaina, L. M. (2005). How can a patient blind to radial motion discriminate shifts in the center-of-motion? *Journal of Computational Neuroscience*, *18*, 55–66.

Beardsley, S. A., & Vaina, L. M. (2006). Global motion mechanisms compensate local motion deficits in a patient with a bilateral occipital lobe lesion. *Experimental Brain Research*, *173*, 724–732.

Berlucchi, G. (2004). Some effects of cortical and callosal damage on conscious and unconscious processing of visual information and other sensory inputs. *Progress in Brain Research*, *144*, 79–93.

Billino, J., Bremmer, F., & Gegenfurtner, K. R. (2008). Differential aging of motion processing mechanisms: Evidence against general perceptual decline. *Vision Research*, *48*, 1254–1261.

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

Born, R. T., & Bradley, D. C. (2005). Structure and function of visual area MT. *Annual Review of Neuroscience*, *28*, 157–189.

Boussaoud, D., Ungerleider, L. G., & Desimone, R. (1990). Pathways for motion analysis: Cortical connections of the medial superior temporal and fundus of the superior temporal visual areas in the macaque. *Journal of Comparative Neurology*, *296*, 462–495.

Braun, D., Petersen, D., Schonle, P., & Fahle, M. (1998). Deficits and recovery of first- and second-order motion perception in patients with unilateral cortical lesions. *European Journal of Neuroscience*, *10*, 2117–2128.

Bremmer, F., Duhamel, J. R., Ben Hamed, S., & Graf, W. (2002). Heading encoding in the macaque ventral intraparietal area (VIP). *European Journal of Neuroscience*, *16*, 1554–1568.

Bremmer, F., Schlack, A., Shah, N. J., Zafiris, O., Kubischik, M., Hoffmann, K., et al. (2001). Polymodal motion processing in posterior parietal and premotor cortex: A human fMRI study strongly implies equivalencies between humans and monkeys. *Neuron*, *29*, 287–296.

Campana, G., Cowey, A., Casco, C., Oudsen, I., & Walsh, V. (2007). Left frontal eye field remembers "where" but not "what". *Neuropsychologia*, *45*, 2340–2345.

Claeys, K. G., Lindsey, D. T., De, S. E., & Orban, G. A. (2003). A higher order motion region in human inferior parietal lobule: Evidence from fMRI. *Neuron*, *40*, 631–642.

Collins, D. L., Neelin, P., Peters, T. M., & Evans, A. C. (1994). Automatic 3D intersubject registration of MR volumetric data in standardized Talairach space. *Journal of Computer Assisted Tomography*, *18*, 192–205.

Cotton, P. L., & Smith, A. T. (2007). Contralateral visual hemifield representations in the human pulvinar nucleus. *Journal of Neurophysiology*, *98*, 1600–1609.

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

Culham, J., He, S., Dukelow, S., & Verstraten, F. A. (2001). Visual motion and the human brain: What has neuroimaging told us? *Acta Psychologica*, *107*, 69–94.

Cutting, J. E. (1978). A program to generate synthetic walkers as dynamic point-light displays. *Behavior Research Methods and Instruments*, *10*, 91–94.

d'Avossa, G., Tosetti, M., Crespi, S., Biagi, L., Burr, D. C., & Morrone, M. C. (2007). Spatiotopic selectivity of BOLD responses to visual motion in human area MT. *Nature Neuroscience*, *10*, 249–255.

de Jong, B. M., Shipp, S., Skidmore, B., Frackowiak, R. S., & Zeki, S. (1994). The cerebral activity related to the visual perception of forward motion in depth. *Brain*, *117*, 1039–1054.

Duffy, C. J., & Wurtz, R. H. (1991a). Sensitivity of MST neurons to optic flow stimuli. I. A continuum of response selectivity to large-field stimuli. *Journal of Neurophysiology*, *65*, 1329–1345.

Duffy, C. J., & Wurtz, R. H. (1991b). Sensitivity of MST neurons to optic flow stimuli. II. Mechanisms of response selectivity revealed by small-field stimuli. *Journal of Neurophysiology*, *65*, 1346–1359.

Dumoulin, S. O., Bittar, R. G., Kabani, N. J., Baker, C. L., Le, G. G., Bruce, P. G., et al. (2000). A new anatomical landmark for reliable identification of human area V5/MT: A quantitative analysis of sulcal patterning. *Cerebral Cortex*, *10*, 454–463.

Fabbri-Destro, M., & Rizzolatti, G. (2008). Mirror neurons and mirror systems in monkeys and humans. *Physiology*, *23*, 171–179.

Farah, M. J. (1994). Neuropsychological inference with an interactive brain: A critique of the "locality" assumption. *Behavioral and Brain Sciences*, *17*, 43–61.

Felleman, D. J., & Van Essen, D. C. (1991). Distributed hierarchical processing in the primate cerebral cortex. *Cerebral Cortex*, *1*, 1–47.

Field, D. T., Wilkie, R. M., & Wann, J. P. (2007). Neural systems in the visual control of steering. *Journal of Neuroscience*, *27*, 8002–8010.

Garcia, J. O., & Grossman, E. D. (2008). Necessary but not sufficient: Motion perception is required for perceiving biological motion. *Vision Research*, *48*, 1144–1149.

Gardner, J. L., Merriam, E. P., Movshon, J. A., & Heeger, D. J. (2008). Maps of visual space in human occipital cortex are retinotopic, not spatiotopic. *Journal of Neuroscience*, *28*, 3988–3999.

Garnham, L., & Sloper, J. J. (2006). Effect of age on adult stereoacuity as measured by different types of stereotest. *British Journal of Ophthalmology*, *90*, 91–95.

Gattass, R., Nascimento-Silva, S., Soares, J. G., Lima, B., Jansen, A. K., Diogo, A. C., et al. (2005). Cortical visual areas in monkeys: Location, topography, connections, columns, plasticity and cortical dynamics. *Philosophical Transactions of the Royal Society B*, *360*, 709–731.

Gilmore, G. C., Wenk, H. E., Naylor, L. A., & Stuve, T. A. (1992). Motion perception and aging. *Psychology and Aging*, *7*, 654–660.

Greenlee, M. W. (2000). Human cortical areas underlying the perception of optic flow: Brain imaging studies. *International Review of Neurobiology*, *44*, 269–292.

Grossman, E. D., Battelli, L., & Pascual-Leone, A. (2005). Repetitive TMS over posterior STS disrupts perception of biological motion. *Vision Research*, *45*, 2847–2853.

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., et al. (2000). Brain areas involved in perception of biological motion. *Journal of Cognitive Neuroscience*, *12*, 711–720.

Hagler, D. J., & Sereno, M. I. (2006). Spatial maps in frontal and prefrontal cortex. *Neuroimage*, *29*, 567–577.

Hess, R. H., Baker, C. L., & Zihl, J. (1989). The "motion-blind" patient: Low-level spatial and temporal filters. *Journal of Neuroscience*, *9*, 1628–1640.

Howard, R. J., Brammer, M., Wright, I., Woodruff, P. W., Bullmore, E. T., & Zeki, S. (1996). A direct demonstration of functional specialization within motion-related visual and auditory cortex of the human brain. *Current Biology*, *6*, 1015–1019.

Huk, A. C., Dougherty, R. F., & Heeger, D. J. (2002). Retinotopy and functional subdivision of human areas MT and MST. *Journal of Neuroscience*, *22*, 7195–7205.

Inman, V. T., Ralston, H., & Todd, J. T. (1981). *Human walking*. Baltimore, MD: Williams & Wilkins.

Ishihara, S. (1962). *Tests for colour blindness*. Tokyo: Kanehara Shuppan.

Kastner, S., & Pinsk, M. A. (2004). Visual attention as a multilevel selection process. *Cognitive, Affective, & Behavioral Neuroscience*, *4*, 483–500.

Kerkhoff, G. (2000). Neurovisual rehabilitation: Recent developments and future directions. *American Journal of Ophthalmology*, *130*, 687–688.

Kerkhoff, G., Schaub, J., & Zihl, J. (1990). Anamnesis of brain-originated vision disorders. *Nervenarzt*, *61*, 711–718.

Konen, C. S., & Kastner, S. (2008). Representation of eye movements and stimulus motion in topographically organized areas of human posterior parietal cortex. *The Journal of Neuroscience*, *28*, 8361–8375.

Kooistra, C. A., & Heilman, K. M. (1989). Hemispatial visual inattention masquerading as hemianopia. *Neurology*, *39*, 1125–1127.

Kourtzi, Z., Krekelberg, B., & Van Wezel, R. J. (2008). Linking form and motion in the primate brain. *Trends in Cognitive Sciences*, *12*, 230–236.

Kustov, A. A., & Robinson, D. L. (1996). Shared neural control of attentional shifts and eye movements. *Nature*, *384*, 74–77.

Lange, J., Georg, K., & Lappe, M. (2006). Visual perception of biological motion by form: A template-matching analysis. *Journal of Vision*, *6*, 836–849.

Lappe, M., Bremmer, F., Pekel, M., Thiele, A., & Hoffmann, K. P. (1996). Optic flow processing in monkey STS: A theoretical and experimental approach. *Journal of Neuroscience*, *16*, 6265–6285.

Logothetis, N. K. (2008). What we can do and what we cannot do with fMRI. *Nature*, *453*, 869–878.

Maunsell, J. H., & Newsome, W. T. (1987). Visual processing in monkey extrastriate cortex. *Annual Review of Neuroscience*, *10*, 363–401.

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

Mesulam, M. M. (1999). Spatial attention and neglect: Parietal, frontal and cingulate contributions to the mental representation and attentional targeting of salient extrapersonal events. *Philosophical Transactions of the Royal Society London B*, *354*, 1325–1346.

Michels, L., Lappe, M., & Vaina, L. M. (2005). Visual areas involved in the perception of human movement from dynamic form analysis. *Neuroreport*, *16*, 1037–1041.

Moore, T., & Fallah, M. (2001). Control of eye movements and spatial attention. *PNAS*, *98*, 1273–1276.

Morrone, M. C., Tosetti, M., Montanaro, D., Fiorentini, A., Cioni, G., & Burr, D. C. (2000). A cortical area that responds specifically to optic flow, revealed by fMRI. *Nature Neuroscience*, *3*, 1322–1328.

Newsome, W. T., & Pare, E. B. (1988). A selective impairment of motion perception following lesions of the middle temporal visual area (MT). *Journal of Neuroscience*, *8*, 2201–2211.

Oldfield, R. C. (1971). The assessment and analysis of handedness: The Edinburgh inventory. *Neuropsychologia*, *9*, 97–113.

Orban, G. A., Fize, D., Peuskens, H., Denys, K., Nelissen, K., Sunaert, S., et al. (2003). Similarities and differences in motion processing between the human and macaque brain: Evidence from fMRI. *Neuropsychologia*, *41*, 1757–1768.

Orban, G. A., Sunaert, S., Todd, J. T., Van Hecke, P., & Marchal, G. (1999). Human cortical regions involved in extracting depth from motion. *Neuron*, *24*, 929–940.

Orban, G. A., Van Essen, D., & Vanduffel, W. (2004). Comparative mapping of higher visual areas in monkeys and humans. *Trends in Cognitive Sciences*, *8*, 315–324.

Pasternak, T., & Merigan, W. H. (1994). Motion perception following lesions of the superior temporal sulcus in the monkey. *Cerebral Cortex*, *4*, 247–259.

Pavlova, M., Birbaumer, N., & Sokolov, A. (2006). Attentional modulation of cortical neuromagnetic gamma response to biological movement. *Cerebral Cortex*, *16*, 321–327.

Pelli, D. G., Robson, J. G., & Wilkins, A. J. (1988). The design of a new letter chart for measuring contrast sensitivity. *Clinical Vision Science*, *2*, 187–199.

Peuskens, H., Sunaert, S., Dupont, P., Van Hecke, P., & Orban, G. A. (2001). Human brain regions involved in heading estimation. *Journal of Neuroscience*, *21*, 2451–2461.

Plant, G. T., Laxer, K. D., Barbaro, N. M., Schiffman, J. S., & Nakayama, K. (1993). Impaired visual motion perception in the contralateral hemifield following unilateral posterior cerebral lesions in humans. *Brain*, *116*, 1303–1335.

Posner, M. I., & Dehaene, S. (1994). Attentional networks. *Trends in Neurosciences*, *17*, 75–79.

Ptito, M., Kupers, R., Faubert, J., & Gjedde, A. (2001). Cortical representation of inward and outward radial motion in man. *Neuroimage*, *14*, 1409–1415.

Rizzo, M., Nawrot, M., & Zihl, J. (1995). Motion and shape perception in cerebral akinetopsia. *Brain*, *118*, 1105–1127.

Rizzolatti, G., & Craighero, L. (2004). The mirror-neuron system. *Annual Review of Neuroscience*, *27*, 169–192.

Rizzolatti, G., Matelli, M., & Pavesi, G. (1983). Deficits in attention and movement following the removal of postarcuate (area 6) and prearcuate (area 8) cortex in macaque monkeys. *Brain*, *106*, 655–673.

Robinson, D. A., & Fuchs, A. F. (1969). Eye movements evoked by stimulation of frontal eye fields. *Journal of Neurophysiology*, *32*, 637–648.

Rorden, C., & Brett, M. (2000). Stereotaxic display of brain lesions. *Behavioural Neurology*, *12*, 191–200.

Rorden, C., & Karnath, H. O. (2004). Using human brain lesions to infer function: A relic from a past era in the fMRI age? *Nature Reviews Neuroscience*, *5*, 813–819.

Royden, C. S., & Vaina, L. M. (2004). Is precise discrimination of low level motion needed for heading discrimination? *Neuroreport*, *15*, 1013–1017.

Rudolph, K., & Pasternak, T. (1999). Transient and permanent deficits in motion perception after lesions of cortical areas MT and MST in the macaque monkey. *Cerebral Cortex*, *9*, 90–100.

Saito, H., Yukie, M., Tanaka, K., Hikosaka, K., Fukada, Y., & Iwai, E. (1986). Integration of direction signals of image motion in the superior temporal sulcus of the macaque monkey. *Journal of Neuroscience*, *6*, 145–157.

Saygin, A. P. (2007). Superior temporal and premotor brain areas necessary for biological motion perception. *Brain*, *130*, 2452–2461.
Saygin, A. P., & Sereno, M. I. (2008). Retinotopy and attention in human occipital, temporal, parietal, and frontal cortex. *Cerebral Cortex*, *18*, 2158–2168.
Saygin, A. P., Wilson, S. M., Hagler, D. J., Bates, E., & Sereno, M. I. (2004). Point-light biological motion perception activates human premotor cortex. *Journal of Neuroscience*, *24*, 6181–6188.
Schaafsma, S. J., & Duysens, J. (1996). Neurons in the ventral intraparietal area of awake macaque monkey closely resemble neurons in the dorsal part of the medial superior temporal area in their responses to optic flow patterns. *Journal of Neurophysiology*, *76*, 4056–4068.
Schaafsma, S. J., Duysens, J., & Gielen, C. C. (1997). Responses in ventral intraparietal area of awake macaque monkey to optic flow patterns corresponding to rotation of planes in depth can be explained by translation and expansion effects. *Visual Neuroscience*, *14*, 633–646.
Schafer, R. J., & Moore, T. (2007). Attention governs action in the primate frontal eye field. *Neuron*, *56*, 541–551.
Schenk, T., & Zihl, J. (1997a). Visual motion perception after brain damage. I. Deficits in global motion perception. *Neuropsychologia*, *35*, 1289–1297.
Schenk, T., & Zihl, J. (1997b). Visual motion perception after brain damage. II. Deficits in form-from-motion perception. *Neuropsychologia*, *35*, 1299–1310.
Schenkenberg, T., Bradford, D. C., & Ajax, E. T. (1980). Line bisection and unilateral visual neglect in patients with neurologic impairment. *Neurology*, *30*, 509–517.
Serences, J. T., & Yantis, S. (2007). Spatially selective representations of voluntary and stimulus-driven attentional priority in human occipital, parietal, and frontal cortex. *Cerebral Cortex*, *17*, 284–293.
Shipp, S. (2004). The brain circuitry of attention. *Trends in Cognitive Sciences*, *8*, 223–230.
Shipp, S., de Jong, B. M., Zihl, J., Frackowiak, R. S., & Zeki, S. (1994). The brain activity related to residual motion vision in a patient with bilateral lesions of V5. *Brain*, *117*, 1023–1038.
Siegel, R. M., & Read, H. L. (1997). Analysis of optic flow in the monkey parietal area 7a. *Cerebral Cortex*, *7*, 327–346.
Silvanto, J., Lavie, N., & Walsh, V. (2006). Stimulation of the human frontal eye fields modulates sensitivity of extrastriate visual cortex. *Journal of Neurophysiology*, *96*, 941–945.
Smith, A. T., Wall, M. B., Williams, A. L., & Singh, K. D. (2006). Sensitivity to optic flow in human cortical areas MT and MST. *European Journal of Neuroscience*, *23*, 561–569.
Snowden, R. J., & Kavanagh, E. (2006). Motion perception in the ageing visual system: Minimum motion, motion coherence, and speed discrimination thresholds. *Perception*, *35*, 9–24.
Sunaert, S., Van Hecke, P., Marchal, G., & Orban, G. A. (1999). Motion-responsive regions of the human brain. *Experimental Brain Research*, *127*, 355–370.
Talairach, J., & Tournoux, P. (1988). *Co-planar stereotaxic atlas of the human brain: 3-dimensional proportional system—An approach to cerebral imaging*. New York, NY: Thieme.
Tran, D. B., Silverman, S. E., Zimmerman, K., & Feldon, S. E. (1998). Age-related deterioration of motion perception and detection. *Graefes Archive for Clinical and Experimental Ophthalmology*, *236*, 269–273.
Trick, G. L., & Silverman, S. E. (1991). Visual sensitivity to motion: Age-related changes and deficits in senile dementia of the Alzheimer type. *Neurology*, *41*, 1437–1440.
Vaina, L. M., Cowey, A., Eskew, R. T., LeMay, M., & Kemper, T. (2001). Regional cerebral correlates of global motion perception: Evidence from unilateral cerebral brain damage. *Brain*, *124*, 310–321.
Vaina, L. M., Cowey, A., Jakab, M., & Kikinis, R. (2005). Deficits of motion integration and segregation in patients with unilateral extrastriate lesions. *Brain*, *128*, 2134–2145.
Vaina, L. M., Cowey, A., LeMay, M., Bienfang, D. C., & Kikinis, R. (2002). Visual deficits in a patient with 'kaleidoscopic disintegration of the visual world'. *European Journal of Neurology*, *9*, 463–477.
Vaina, L. M., & Gross, C. G. (2004). Perceptual deficits in patients with impaired recognition of biological motion after temporal lobe lesions. *PNAS*, *101*, 16947–16951.
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. *Visual Neuroscience*, *5*, 353–369.
Vaina, L. M., Solomon, J., Chowdhury, S., Sinha, P., & Belliveau, J. W. (2001). Functional neuroanatomy of biological motion perception in humans. *PNAS*, *98*, 11656–11661.
Vaina, L. M., & Soloviev, S. (2004a). First-order and second-order motion: Neurological evidence for neuroanatomically distinct systems. *Progress in Brain Research*, *144*, 197–212.
Vaina, L. M., & Soloviev, S. (2004b). Functional neuroanatomy of heading perception in humans. In L. M. Vaina, S. A. Beardsley, & S. K. Rushton (Eds.), *Optic flow and beyond* (pp. 109–137). Norwell, MA: Kluwer Academic Publishers.
Van Essen, D. C. (2005). Corticocortical and thalamocortical information flow in the primate visual system. *Progress in Brain Research*, *149*, 173–185.
Wall, M. B., & Smith, A. T. (2008). The representation of egomotion in the human brain. *Current Biology*, *18*, 191–194.
Warren, W. H., Blackwell, A. W., & Morris, M. W. (1989). Age differences in perceiving the direction of self-motion from optical flow. *Journal of Gerontology*, *44*, 147–153.
Watson, J. D., Myers, R., Frackowiak, R. S., Hajnal, J. V., Woods, R. P., Mazziotta, J. C., et al. (1993). Area V5 of the human brain: Evidence from a combined study using positron emission tomography and magnetic resonance imaging. *Cerebral Cortex*, *3*, 79–94.
Weintraub, S. (2000). Neuropsychological assessment of mental state. In M.-M. Mesulam (Ed.), *Principles of behavioral and cognitive neurology* (pp. 121–173). New York, NY: Oxford University Press.
Wichmann, F. A., & Hill, N. J. (2001a). The psychometric function. I. Fitting, sampling, and goodness of fit. *Perception and Psychophysics*, *63*, 1293–1313.
Wichmann, F. A., & Hill, N. J. (2001b). The psychometric function. II. Bootstrap-based confidence intervals and sampling. *Perception and Psychophysics*, *63*, 1314–1329.
Wojciechowski, R., Trick, G. L., & Steinman, S. B. (1995). Topography of the age-related decline in motion sensitivity. *Optometry and Visual Science*, *72*, 67–74.
World Medical Association (2004). Ethical principles for medical research involving human subjects. Retrieved November 15, 2007, from http://www.wma.net/e/policy/b3.htm.
Wunderlich, G., Marshall, J. C., Amunts, K., Weiss, P. H., Mohlberg, H., Zafiris, O., et al. (2002). The importance of seeing it coming: A functional magnetic resonance imaging study of motion-in-depth towards the human observer. *Neuroscience*, *112*, 535–540.
Xiao, Q., Barborica, A., & Ferrera, V. P. (2006). Radial motion bias in macaque frontal eye field. *Visual Neuroscience*, *23*, 49–60.
Yamasaki, D. S., & Wurtz, R. H. (1991). Recovery of function after lesions in the superior temporal sulcus in the monkey. *Journal of Neurophysiology*, *66*, 651–673.
Zaroff, C. M., Knutelska, M., & Frumkes, T. E. (2003). Variation in stereoacuity: Normative description, fixation disparity, and the roles of aging and gender. *Investigative Ophthalmology & Visual Science*, *44*, 891–900.
Zeki, S. (1991). Cerebral akinetopsia (visual motion blindness). A review. *Brain*, *114*, 811–824.
Zeki, S. M. (1974). Functional organization of a visual area in the posterior bank of the superior temporal sulcus of the rhesus monkey. *Journal of Physiology*, *236*, 549–573.
Zihl, J., von Cramon, D., & Mai, N. (1983). Selective disturbance of movement vision after bilateral brain damage. *Brain*, *106*, 313–340.
Zihl, J., von Cramon, D., Mai, N., & Schmid, C. (1991). Disturbance of movement vision after bilateral posterior brain damage. Further evidence and follow up observations. *Brain*, *114*, 2235–2252.