

Challenges to normal neural functioning provide insights into separability of motion processing mechanisms

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ARTICLE INFO

Article history:

Received 28 September 2010

Received in revised form 8 July 2011

Accepted 13 July 2011

Available online 22 July 2011

Keywords:

Visual motion processing

First-order motion

Second-order motion

Ageing

Brain lesions

ABSTRACT

There is a long history of attempts to disentangle different visual processing mechanisms for physically different motion cues. However, underlying neural correlates and separability of networks are still under debate. We aimed to refine the current understanding by studying differential vulnerabilities when normal neural functioning is challenged. We investigated effects of ageing and extrastriate brain lesions on detection thresholds for motion defined by either luminance- or contrast modulations, known as first- and second-order motion. Both approaches focus on extrastriate processing changes and combine distributed as well as more focal constraints. Our ageing sample comprised 102 subjects covering an age range from 20 to 82 years. Threshold signal-to-noise ratios for detection approximately doubled across the age range for both motion types. Results suggest that ageing affects perception of both motion types to an equivalent degree and thus support overlapping processing resources. Underlying neural substrates were further qualified by testing perceptual performance of 18 patients with focal cortical brain lesions. We determined selective first-order motion deficits in three patients, selective second-order motion deficits in only one patient, and deficits for both motion types in three patients. Lesion analysis yielded support for common functional substrates in higher cortical regions. Functionally specific substrates remained ambiguous, but tended to cover earlier visual areas. We conclude that observed vulnerabilities of first- and second-order motion perception provide limited evidence for functional specialization at early extrastriate stages, but emphasize shared processing pathways at higher cortical levels.

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1. Introduction

Advances in modern neuroscience have contributed to a constantly increasing understanding of brain mechanisms underlying perceptual functions. In particular for the visual system, the integration of results from a variety of methods has provided elaborate processing models. The perception of motion probably represents the most intensively studied visual capacity. Combining electrophysiological, behavioural, and functional imaging data, neural correlates of visual motion analysis have been identified and characterized very precisely (see e.g. Andersen, 1997). Remarkably enough, some controversy over basic mechanisms has remained unresolved so far.

The distinction between different functional subsystems for first- and second-order motion perception has attracted sustained attention over many years. Visual motion can be defined either by the spatiotemporal modulation of luminance or by the spatiotemporal modulation of local contrast, texture, or some other properties, respectively labelled as first- or second-order motion (Cavanagh & Mather, 1989; Lu & Sperling, 1995). In contrast to the straightforward physical distinction, it has turned out to be quite debatable whether processing mechanisms are separable and most notably whether they involve specific neural networks (for review see Derrington, Allen, & Delicato, 2004). We propose that differential vulnerabilities of normal functioning allow important insights into underlying neural substrates and thus can be used as an additional window to motion analysis.

Psychophysical studies support that first- and second-order motion are initially analyzed by separate mechanisms in the visual system, but might share later stages of processing. Early evidence came from Ledgeway and Smith (1994) who showed that both motion types are detected separately and cannot be integrated into one single percept. Similarly, work on noise effects, adaptation, and aftereffects demonstrated high specificity and only little cross-order effects (Allard & Faubert, 2007; Nishida, Ledgeway, &

Abbreviations: area MST, medial superior temporal area; area V3, third visual area; area V5/MT, fifth visual area/middle temporal area; CT, computer tomography; fMRI, functional magnetic resonance imaging; MRT, magnetic resonance tomography; SPL, superior parietal lobule; STS, superior temporal sulcus; VF, visual field.

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Edwards, 1997; Nishida & Sato, 1995; Pavan, Campana, Guerreschi, Manassi, & Casco, 2009). Results suggest that separate neural populations are involved in encoding first- and second-order motion. However, processing might converge at higher cortical stages. Campana, Pavan, and Casco (2008) recently reported on cross-order motion priming that lacked sensitivity to spatial position. They proposed that common processing occurs in areas where retinotopicity is no longer maintained. Further evidence of differences in processing mechanisms for first- and second-order motion comes from sensitivity changes across life span. Sensitivity to second-order motion seems to mature more slowly (Elleberg et al., 2004; Kato, de Wit, Stasiewicz, & von Hofsten, 2008; but see Braddick, Atkinson, & Wattam-Bell, 2003) and to be subject to earlier or more pronounced age-related decline in adulthood (Habak & Faubert, 2000; Tang & Zhou, 2009).

Several approaches have aimed to identify critical neural correlates of specialized motion processing, including electrophysiology, functional brain imaging, and lesion studies in patients.

Responses of neurons to first- and second-order motion were recorded in striate and early extrastriate visual areas of cats and monkeys (for review see Baker, 1999). Although neuronal responses to second-order motion have been described as early as in area V1, there is consensus that striate activity induced by this motion type is relatively weak and involves a small proportion of neurons (Mareschal & Baker, 1999; Zhou & Baker, 1996). Recordings in extrastriate areas MT and MST yielded heterogeneous results. Some studies found more than 40% of neurons responsive for both motion types (Albright, 1992; Churan & Ilg, 2001; Geesaman & Andersen, 1996), but other results indicated that only a minority of neurons show these characteristics (O'Keefe & Movshon, 1998).

Findings from functional brain imaging studies in humans support specific sensitivity to first-order motion in area V1 and to second-order motion in area V3 (Smith, Greenlee, Singh, Kraemer, & Hennig, 1998). In addition to specialized processing in early occipital areas further studies also reported dissociated processing in higher cortical areas, e.g. the parietal lobe, and the superior temporal sulcus (STS) (Ashida, Lingnau, Wall, & Smith, 2007; Dumoulin, Baker, Hess, & Evans, 2003; Noguchi, Kaneoke, Kakigi, Tanabe, & Sadato, 2005). In contrast, Nishida, Sasaki, Murakami, Watanabe, and Tootell (2003) questioned dissociated processing pathways. They emphasized the need for controlling the influence of motion-irrelevant information and focused on direction-selective adaptation to first- and second-order motion. Observed pattern of activity in their study indicate shared neural substrates for processing of both motion types as early as in area V1.

A number of observations in brain-lesioned patients have suggested a functional dissociation. In an early study, Plant and colleagues found impaired second-order motion perception in patients with occipito-parietal lesion sites whereas first-order motion perception was preserved (Plant, Laxer, Barbaro, Schiffman, & Nakayama, 1993; Plant & Nakayama, 1993). They speculated that first-order motion mechanisms might be distributed more widely in cortex so that they are less susceptible to focal damage. In disagreement, Rizzo, Nawrot, Sparks, and Dawson (2008) recently reported on a large patient sample with visual cortex lesions in which second-order motion processing appeared much more robust. They considered second-order motion processing to be probably represented at higher central levels. Most recognized evidence for dissociated motion processing mechanisms and specific neural correlates was provided by Vaina and collaborators (Vaina & Cowey, 1996; Vaina, Cowey, & Kennedy, 1999; Vaina, Makris, Kennedy, & Cowey, 1998; Vaina, Soloviev, Bienfang, & Cowey, 2000). In case studies, they described patients with either selective first- or selective second-order motion deficits. Lesion analysis showed specific functional significance of regions near area

V2 for first-order motion processing and of regions in the vicinity of area V5/MT for second-order motion processing. However, there have also been reports on patients suffering from common deficits for first- and second-order motion perception (Braun, Petersen, Schonle, & Fahle, 1998; Greenlee & Smith, 1997; Nawrot, Rizzo, Rockland, & Howard, 2000). Their lesions predominantly affected higher extrastriate areas, e.g. in the occipital-temporal cortex, the superior temporal lobe, or the lateral parietal lobe.

The manifold efforts to differentiate between first- and second-order motion perception have refined our understanding of motion analysis (compare Ilg & Churan, 2010). The vast majority of findings suggest separate processing pathways in striate cortex and later occipital areas; however specific functional contributions of higher extrastriate areas have remained controversial. Indeed, a variety of different cortical areas have been shown to be involved in motion analysis (Billino, Braun, Bohm, Bremmer, & Gegenfurtner, 2009; Culham, He, Dukelow, & Verstraten, 2001; Sunaert, Van Hecke, Marchal, & Orban, 1999), though, functional specificity has been elusive. We consider the lack of knowledge on differential first- and second-order motion processing beyond early occipital areas as highly relevant for the notion of higher complexity of second-order motion analysis (compare Faubert, 2002). Is this complexity primarily based on specific early processing steps or do specific functional pathways in higher cortical areas contribute to separability of first- and second-order motion perception? And if so, do these functional pathways comprise completely distinct neural resources or share some common substrates?

Challenges to normal neural processing offer the possibility to study functionally specific consequences and thus to differentiate between functional submechanisms. We were especially interested in how extrastriate processing changes affect first- and second-order motion perception. The combined consideration of normal age effects and deficits after focal brain lesions provides several advantages for this purpose. Both methodological approaches allow insights into specific vulnerabilities and offer a window to underlying neural substrates. The ageing brain is subject to many physiological changes and growing research interest has yielded significant advances in detailed understanding. There is converging evidence that brain regions age at different rates and are differentially prone to volume loss (Pieperhoff et al., 2008; Raz et al., 2005; Sowell et al., 2003). With regard to cortical brain areas, imaging results suggest that the occipital lobes might be the most robust to the effects of ageing. Age-related shrinkage affects the occipital lobes only moderately whereas for temporal, parietal, and particularly frontal areas pronounced volume decline has been observed. Therefore, differential age effects on first- and second-order motion perception are a good candidate to reflect extrastriate processing differences. We supposed that data from a large sample covering a broad age range might enable us to evaluate differential decline reliably. Although age effects on motion perception can be a powerful handle to separability of processing mechanisms, the associated neural correlates remain speculative. Deficits after focal brain lesions represent a convenient complement because they directly point to critical functional areas. Besides uncovering dissociations, they can reveal the complexity of neural networks involved in first- and second-order motion perception. We considered perceptual performance in patients with focal extrastriate lesions localized at diverse cortical areas in order to further qualify separability of processing pathways beyond early occipital cortex.

2. Methods

2.1. Participants

2.1.1. Age sample

We recruited 102 healthy subjects (56 females) ranging in age from 18 to 82 years ($M=52.9$, $SD=19.8$). Subjects were required to show normal or corrected-

to-normal visual acuity and normal contrast sensitivity in standard tests. Ocular diseases, a history of neurological or psychiatric disorders, and medications known to interfere with visual functioning were screened out.

2.1.2. Patient sample

Over a period of 12 months, we considered all ischemic or hemorrhagic stroke patients admitted to the Neurological Clinic Braunfels, a rehabilitation unit cooperating with the University of Giessen. Individual screening sessions were scheduled if (i) medical records described focal cortical lesions visualized by magnetic resonance tomography (MRT) 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 on medications known to interfere with visual functioning. Patients had to accomplish a battery of standard visual tests to assure normal abilities regarding visual acuity, stereo vision, contrast sensitivity, and color perception. Moreover, visual field defects affecting a central radius of 20° and impaired visuospatial attention, i.e. neglect symptoms, were defined as exclusion criteria. We obtained a group of 18 patients (6 females) 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.

2.2. Stimuli

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/m² and .3 cd/m², respectively, resulting in a maximum Michelson contrast of 99%. A gamma correction ensured linearity of gray levels.

We used two types of grating stimuli typically used to differentiate between first- and second-order motion (compare Derrington et al., 2004; Lu & Sperling, 2001). Static versions of the gratings are depicted in Fig. 1A. First-order motion was presented in luminance modulated vertical sinusoidal gratings with a spatial frequency of 0.7 cyc/° and a Michelson contrast of 10%. Gratings could drift either to the right or to the left at a speed of 10.1°/s. Second-order motion was defined by a stationary carrier consisting of vertical light and dark stripes of random widths and a vertical sinusoidal contrast modulation with a spatial frequency of 0.7 cyc/° that moved either to the right or to the left at a speed of 10.1°/s. The Michelson contrast of the carrier was set to 50% and the sinusoidal modulation lowered the contrast to 10% periodically. The relatively high drift speed in both grating types was chosen corresponding to our specific interest in extras-

Table 1
Summary of patients' clinical characteristics and perceptual thresholds in both motion tasks.

Case	Age	Sex	Lesion type	Lesion location	Lesion-test-interval (weeks)	Visual Field	First-order motion		Second-order motion	
							contra	ipsi	contra	ipsi
CH	60	F	ICH	Right: O-T	6	○	30.4	25.6	56.3	68.5
GF	42	M	INF	Right: F	3	○	50.8*	50.0*	60.9	54.2
KE	44	M	ICH	Left: F	9	○	31.1	39.3	59.0	62.0
KK	74	M	INF	Right: T-P	5	●	56.3	40.4	88.1	72.2
KN	27	F	SAH	Bilateral: F	5	○	22.7	30.3	36.4	50.4
KS	50	M	INF	Left: P-T	4	○	53.5*	63.1*	68.2	65.8
LL	40	M	INF	Right: P-F	6	○	20.6	23.0	42.7	57.0
MB	50	M	INF	Right: P	5	○	54.1*	55.0*	64.0	62.0
MS	22	F	SAH, INF	Left: T	7	○	67.8*	↔ 56.9*	70.8*	52.6
PEK	42	F	SAH	Left: T-P	5	○	38.9	34.2	52.4	54.0
PK	38	M	INF	Left: P	4	○	26.7	24.2	49.7	46.2
RL	51	F	SAH	Right: T	5	●	41.9	33.1	73.0	62.1
SB	38	M	ICH	Right: P	6	●	55.6*	↔ 25.1	68.6	↔ 52.4
SS	39	M	INF	Right: P-F	4	●	32.3	32.1	53.4	51.7
UJ	45	M	INF	Left: P	4	○	23.6	27.0	46.2	46.2
UW	53	F	SAH, INF	Right: F	104	○	47.0	46.5	53.0	49.6
WK	56	M	INF	Right: O-P-T	5	○	57.2*	↔ 40.3	84.7*	↔ 58.9
WR	52	M	INF	Right: O-P-T	4	○	46.5	45.4	81.7*	82.5*

Abbreviations. M: male, F: female; INF: infarction; ICH: intracerebral hemorrhage; SAH: subarachnoidal hemorrhage; 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. Detection thresholds are given for the contralesional visual field and the ipsilesional visual field. Thresholds which exceed the upper 90% limit of the age-specific prediction are marked by an asterisk. Differences between psychometric functions for the contra- and ipsilesional visual fields have been evaluated in consideration of threshold and slope differences using Monte Carlo simulations and setting a significance level of .05; || indicates equivalence, ↔ indicates a difference between psychometric functions. Elevated thresholds and differences between psychometric functions are defined as deficits and are highlighted in red.

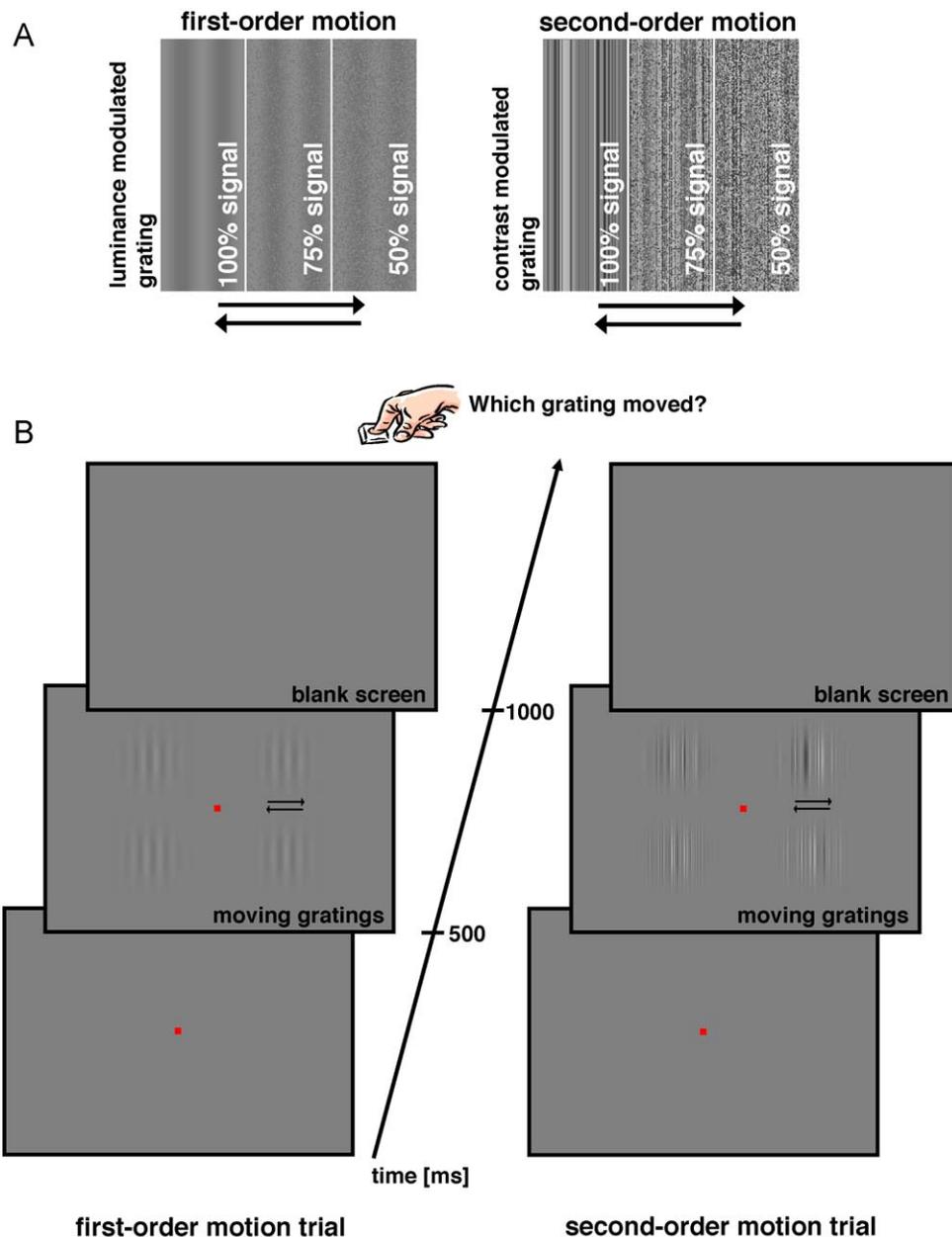


Fig. 1. Static representation of motion stimuli. (A) Gratings. First-order, luminance modulated grating and second-order, contrast modulated grating. For illustration gratings are shown at signal-to-noise levels of 100%, 75%, and 50%. Modulation could move either to the right or to the left. (B) Procedure. Detection thresholds were determined in a 4-alternative forced choice (4AFC) paradigm. After a fixation period, four gratings masked by Gaussian envelopes were presented simultaneously, one in each quadrant of the screen. Three gratings were stationary whereas one drifted either to the right or to the left. Signal intensity varied across five different noise levels. Participants had to detect the location of the drifting grating and gave responses without temporal constraints.

triate motion processing. Extrastriate areas have been shown to be particularly involved in processing of higher velocities (compare Rodman & Albright, 1987). In addition, according to the classification of motion systems by Lu and Sperling (2001) a drift speed of $10.1^\circ/s$ provided an adequate input to the first- and second-order motion systems, but excluded possible contributions of the third-order motion system which is sensitive to lower temporal frequencies. Signal intensity of motion gratings was varied by a dynamic gray-scale noise mask. A certain percentage of stimuli's pixels were replaced randomly by either light or dark pixels. The noise mask had a Michelson contrast of 50% and was set new every tenth frame.

2.3. Procedure

Prior to the testing session informed consent was given by all participants according to the Declaration of Helsinki (World Medical Association, 2004). Methods and procedures were approved by the local ethics committee.

Subjects were seated in a darkened room in front of the monitor at a distance of 60 cm. Viewing was binocular and the head was stabilized by a chinrest. The back-

ground screen was set to gray of mean luminance. A red fixation square, subtending $0.3^\circ \times 0.3^\circ$, was provided 500 ms before stimulus onset and persisted during stimulus presentation. Subjects were instructed to fixate at the centre of the screen and not to move their eyes. 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, which occurred very rarely, trials were immediately rejected and repeated. Subjects were reminded to refixate.

First- and second-order motion stimuli were presented in spatial 4-alternative forced choice paradigms. In each trial, four gratings, one in each quadrant of the screen, appeared simultaneously for 500 ms. Gratings were shifted from the fixation square diagonally to an eccentricity of 7.1° and masked by a Gaussian envelope with a standard deviation of 2.3° . One grating drifted whereas the other three were static. Subjects had to detect the location of the drifting grating. The procedure is illustrated in Fig. 1B. Responses were entered without temporal constraints directly on the keyboard after stimulus presentation. No feedback was given. The next trial was started by pressing the space bar.

Before obtaining threshold data, sufficient practice trials were given so that all subjects got used to each task and could 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.

2.4. Psychophysical data analysis

Thresholds were obtained by fitting the percentage of correct responses with a Weibull function for a performance level of 63%. We used the *psignifit* toolbox in Matlab (Wichmann & Hill, 2001a, 2001b) and summary statistics yielded a good fit between the model and the data. Since it is known that perceptual thresholds for second-order motion lie well above thresholds for first-order motion (see Manahilov, Simpson, & Calvert, 2005), we transformed absolute thresholds into log units in order to provide an appropriate comparison.

Age-related changes in first- and second-order motion perception were characterized by correlation and regression analyses. For comparison between the cross-sectional developmental trajectories of both perceptual measures we conducted a repeated measures ANCOVA with age as covariate (compare Thomas et al., 2009).

Patients' perceptual thresholds were evaluated with reference to performance of our age sample. Individual age-specific thresholds were predicted using determined regression coefficients and for each prediction the upper 90% limit was calculated. Due to possible retinotopic deficits in patients, their thresholds were determined separately for the contra- and ipsilesional visual hemifields. Differences between psychometric functions for both visual hemifields were analyzed by Monte Carlo simulations of the distribution of threshold and slope differences. Observed differences in threshold and slope were tested simultaneously with a level of significance of $\alpha = .05$. Patients' performance was considered as deficient either when thresholds exceeded the 90% limit of the age-specific prediction or when significant differences between psychometric functions for both visual hemifields were asserted.

2.5. Lesion analysis in patients

Anatomical analysis of cortical lesion location in patients was based on MRT (including diffusion-weighted, T1, and T2 weighted MRT) scans for 9 patients and on CT (spiral CT) scans for 9 patients. Median time between lesion and considered imaging was 2 days (range between 1 and 29 days) for MRT scans and 9 days (range between 1 and 63 days) for CT scans. The MRICron software was used for detailed lesion analysis (Rorden & Brett, 2000; Rorden, Karnath, & Bonilha, 2007). Patients' lesions were transferred 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 specific deficit, i.e. selective or common deficits were overlaid to highlight 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 (for discussion compare Rorden & Karnath, 2004). We therefore qualified simple overlay plots by a masking procedure. Patients without a specific deficit 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 the given deficit. A more sophisticated statistical analysis of the association between lesion location and probability of a specific deficit was not applicable due to the small group sizes.

3. Results

3.1. Effects of ageing

We found strong evidence for an age-related sensitivity decline for both first- and second-order motion. Fig. 2A illustrates age effects by psychometric functions of single subjects who showed representative detection thresholds according to their specific age (compare Fig. 2B). For both motion types, rightward shifted functions of the senior subject indicated an age-related threshold increase.

Exemplary differences between single subjects were validated by analysis of threshold data for the complete sample. Correlations between thresholds in log units and age are given in Fig. 2B. Visual inspection of data suggested a linear increase of detection thresholds with age for both motion types. Distributions provided no evidence of task-related ceiling or floor effects. Whereas

first-order motion perception correlated with age only moderately ($r(102) = .497, p < .01$), a strong correlation was found between second-order motion perception and age ($r(102) = .728, p < .01$). We explored a possible influence of visual acuity or contrast sensitivity on these correlations by partial correlation analyses. Controlling for both basic parameters yielded very similar results, i.e. $r(102) = .336, p < .01$, and $r(102) = .610, p < .01$, for first- and second-order motion respectively. Linear regression models predicted cross-sectional developmental trajectories with differential quality. For first-order motion thresholds, we derived the prediction equation $y = .004x + 1.299, s_e = 0.038$, with age explaining about 25% of interindividual variance in perceptual performance. For second-order motion perception, the prediction equation, $y = .004x + 1.596, s_e = 0.020$, resulted in 53% explained variance. Thus, sensitivity for both motion types appeared to rely on at least partially different functional substrates which differ in their susceptibility to age effects. However most notably regression analysis indicated an equivalent age-related increase of thresholds for both motion types. Considering absolute units, thresholds almost doubled from age of 20 to age of 80 for first-order motion (from 23.9% to 41.6%) as well as for second-order motion (from 47.4% to 82.4%).

For further comparison between the developmental trajectories of first- and second-order motion perception we carried out a repeated-measures ANCOVA with age as covariate. Results revealed significant main effects of motion type, $F(1, 100) = 68.95, p < .001, \eta^2 = .408$, and age, $F(1, 100) = 76.77, p < .001, \eta^2 = .434$. The within-subjects effect supported that thresholds for second-order motion detection were consistently higher than for first-order motion detection. Furthermore, the strong main effect of age reflected the overall age-related threshold increase. There was no interaction between age and motion type, $F(1, 100) = .026, p = .873, \eta^2 < .001$, indicating an equivalent age-related sensitivity decline for first- and second-order motion.

Analyzing the intercorrelation between thresholds for first-order and second-order motion detection provided further qualification of results. The left part of Fig. 3 illustrates the raw correlation between thresholds for both motion types. We determined a strong correlation ($r(102) = .582, p < .01$). Thresholds for first- and second-order motion shared 34% of their variance. When we controlled for age, though, shared variance dropped to 14%. The right part of Fig. 3 shows the correlation corrected for age ($r(102) = .370, p < .01$).

Taken together results supported an equivalent sensitivity decline for first- and second-order motion. Processing mechanisms of both motion types thus involve functional resources that are similarly prone to ageing which is suggestive of a significant overlap of neural substrates. However, we also found some evidence of functional resources that do not vary with age and that are more specific to motion type.

3.2. Effects of cortical brain lesions

Perceptual thresholds for first- and second-order motion detection in our patient sample are summarized in Table 1. Impaired performance was determined in 7 patients and is highlighted in red. Deficits were either selective for first- or second-order motion or concerned both motion types. Selective impairment uniformly involved elevated thresholds for both visual hemifields without significant psychometric differences. In contrast, common deficits for both motion types appeared more heterogeneous. Elevated thresholds were mostly present for the contralesional visual hemifield only and psychometric functions differed between both visual hemifields. A detailed description of individual deficits is given in the following.

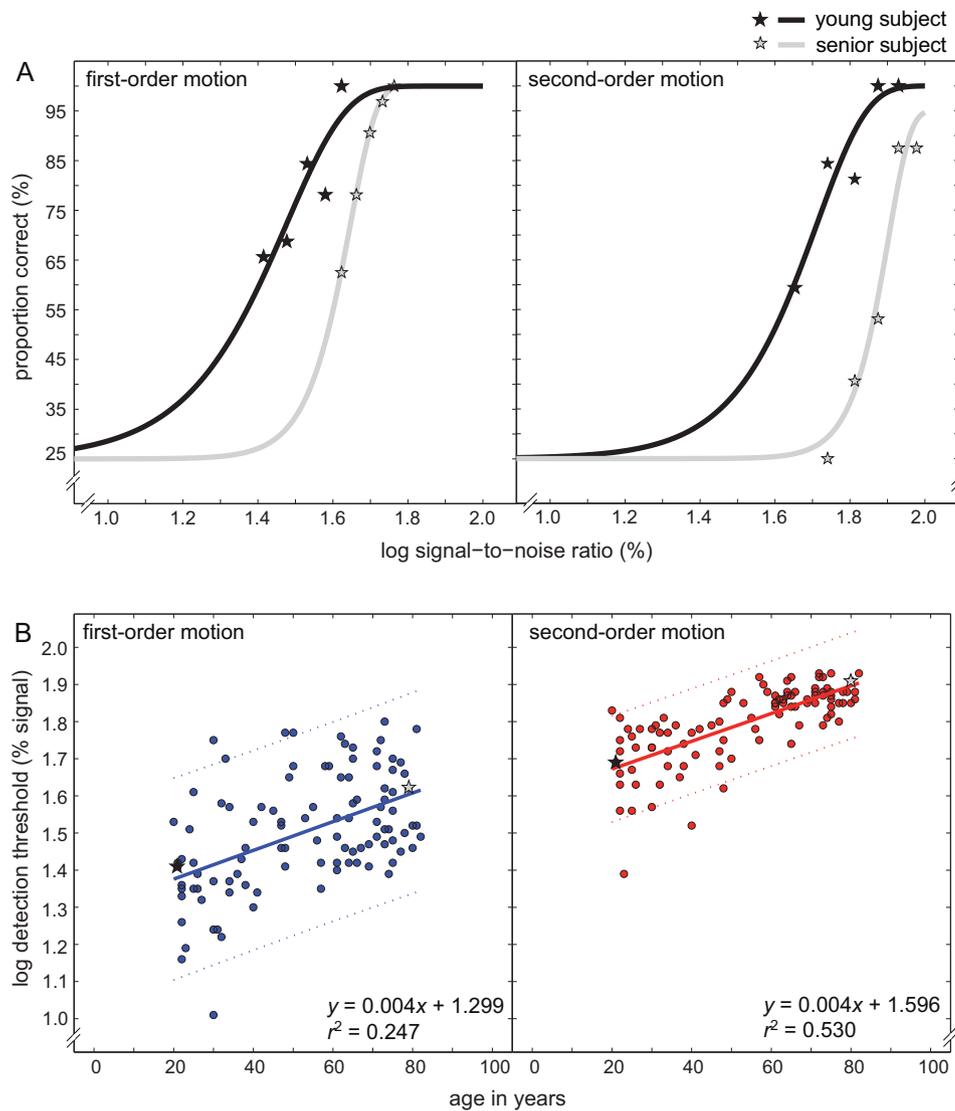


Fig. 2. Age effects on first-order and second-order motion perception. (A) Typical psychometric functions. Detection accuracy is plotted as function of signal intensity given. For each motion type, performance of typical young subject (black line, black stars) and a typical senior subject (gray line, gray stars) with representative thresholds referring to their age group (see part (B)) is shown. (B) Detection thresholds plotted as a function of age. Black and gray stars correspond to the young subject and the senior subject whose psychometric functions are shown in part (A). Regression lines (solid lines), 95% confidence intervals (dashed lines), and model coefficients are given.

3.2.1. Selective first-order motion deficits

3.2.1.1. *Psychophysical data.* Table 1 lists three patients with selective deficits for first-order motion perception. Their performance is illustrated on the left part of Fig. 4.

Patients GF, KS, and MB homogeneously showed elevated thresholds for first-order motion perception. In contrast, their thresholds in the second-order motion task lay well below the critical age-specific predictions. No significant differences between psychometric functions for both hemifields were observed in the first- as well as in the second-order motion task.

3.2.1.2. *Lesions.* The upper part of Fig. 5A depicts the individual lesions of patients GF, KS, and MB. Patients GF and MB suffered from right-sided lesions, whereas patient KS' lesion affected the left hemisphere. Lesions of the three patients did not overlap. Patient GF exhibited a lateral lesion in the posterior frontal lobe. In contrast, lesions of patients KS and MB primarily touched the parietal lobes. Patient KS showed a lateral parietal lesion bordering marginally the

superior temporal lobe. Patient MB's lesion exclusively affected a small medial parietal area.

Lesions of the 15 patients who did not show a selective deficit for first-order motion perception can be superimposed and applied as a mask to lesions of patients GF, KS, and MB. The masked overlay plot is given in the lower part of Fig. 5A. The masking procedure left only small spots of cortical damage. This indicated that lesions in the control patient group overlapped substantially with lesions associated with selective first-order motion deficits. Functional relevance of the described lesion sites therefore has to be considered with caution and remains ambiguous.

3.2.2. Selective second-order motion deficit

3.2.2.1. *Psychophysical data.* We found a selective deficit for second-order motion perception only in patient WR. His performance is shown in the middle part of Fig. 4. Detection thresholds for first-order motion lay in the normal range, but for second-order motion perception he showed threshold elevations for the contra-

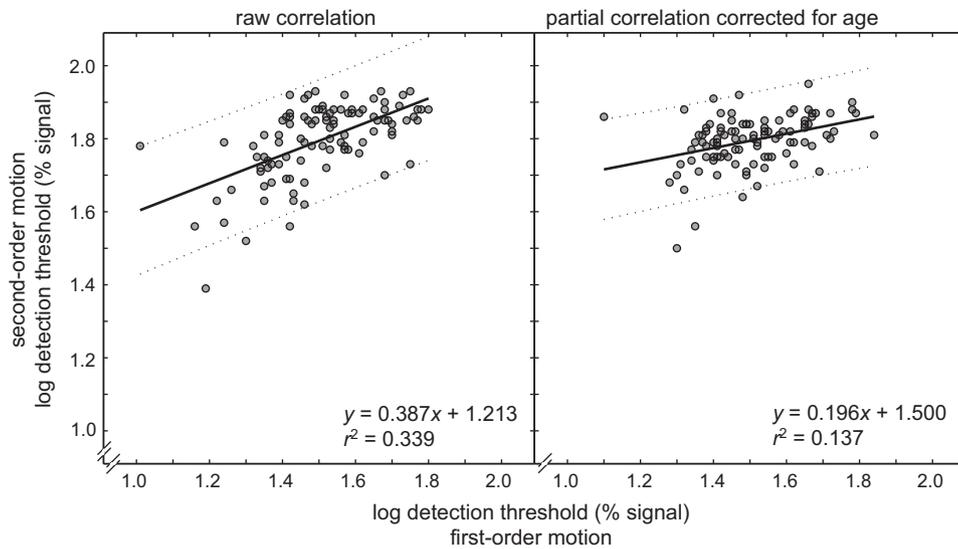


Fig. 3. Correlation between detection thresholds for first-order and second-order motion. The left figure shows the raw correlation, the right figure shows the correlation corrected for the age. Regression lines (solid lines), 95% confidence intervals (dashed lines), and model coefficients are given.

as well as for the ipsilesional visual hemifield. Psychometric functions for both hemifields did not differ in both motion tasks.

3.2.2.2. Lesion. Fig. 5B illustrates patient WR's lesion. It was located in the area of the right occipito-temporo-parietal junction, but affected primarily the occipital lobe. Masking the lesion with the superimposed lesions of the 17 patients without a selective second-order motion deficit supported the functional specificity of the identified area. The masked lesion is shown in the lower part of Fig. 5B. Lesions of the control patients scarcely overlapped with the lesion of patient WR. His critical lesion site was shown to be unique to the selective second-order motion deficit.

3.2.3. Common first- and second-order motion deficits

3.2.3.1. Psychophysical data. Common deficits in both motion tasks were found by three patients, however, deficit profiles differed

somewhat between individual patients as is illustrated on the right part of Fig. 4. Patient MS showed elevated detection thresholds for first-order motion in the contra- as well as in the ipsilesional visual hemifield. However, there also was a significant difference between psychometric functions for both hemifields, indicating a lateralization of perceptual impairment. Her threshold for second-order motion was elevated for the contralesional, but not for the ipsilesional visual hemifield. Although this asymmetry again indicated a lateralization of perceptual impairment, psychometric differences between both hemifields failed to reach significance. Patients SB and WK presented consistent deficit profiles. In both motion tasks, their perceptual thresholds in the contralesional visual hemifield exceeded or just met the critical age-specific prediction while thresholds in the ipsilesional visual hemifield were normal. Significant differences between psychometric functions for both hemifields supported a lateralized deficit in both motion tasks.

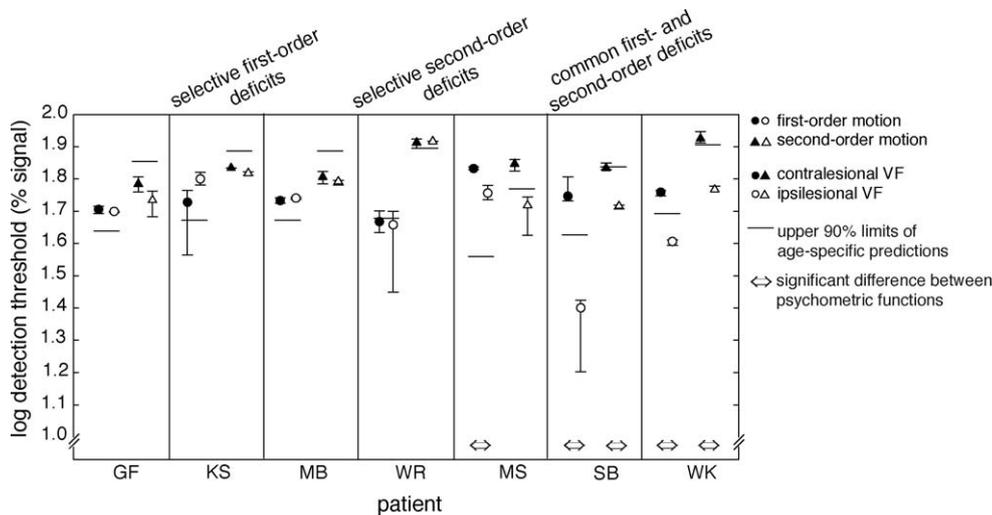


Fig. 4. Deficits for first-order and second-order motion perception. On the x-axis, patients who showed elevated thresholds or differences between psychometric functions for both visual hemifields are presented. The y-axis indicates detection threshold in % signal for a performance level of 63%. Circles symbolize thresholds for first-order motion; triangles symbolize thresholds for second-order motion. Solid symbols represent performance in the contralesional visual field; open circles symbols represent performance in the ipsilesional visual field. Error bars depict 95% confidence intervals of thresholds. For each patient, the bold black horizontal lines indicate the upper limit of the age-specific prediction. Significant differences between psychometric functions for both visual hemifields are marked by ↔.

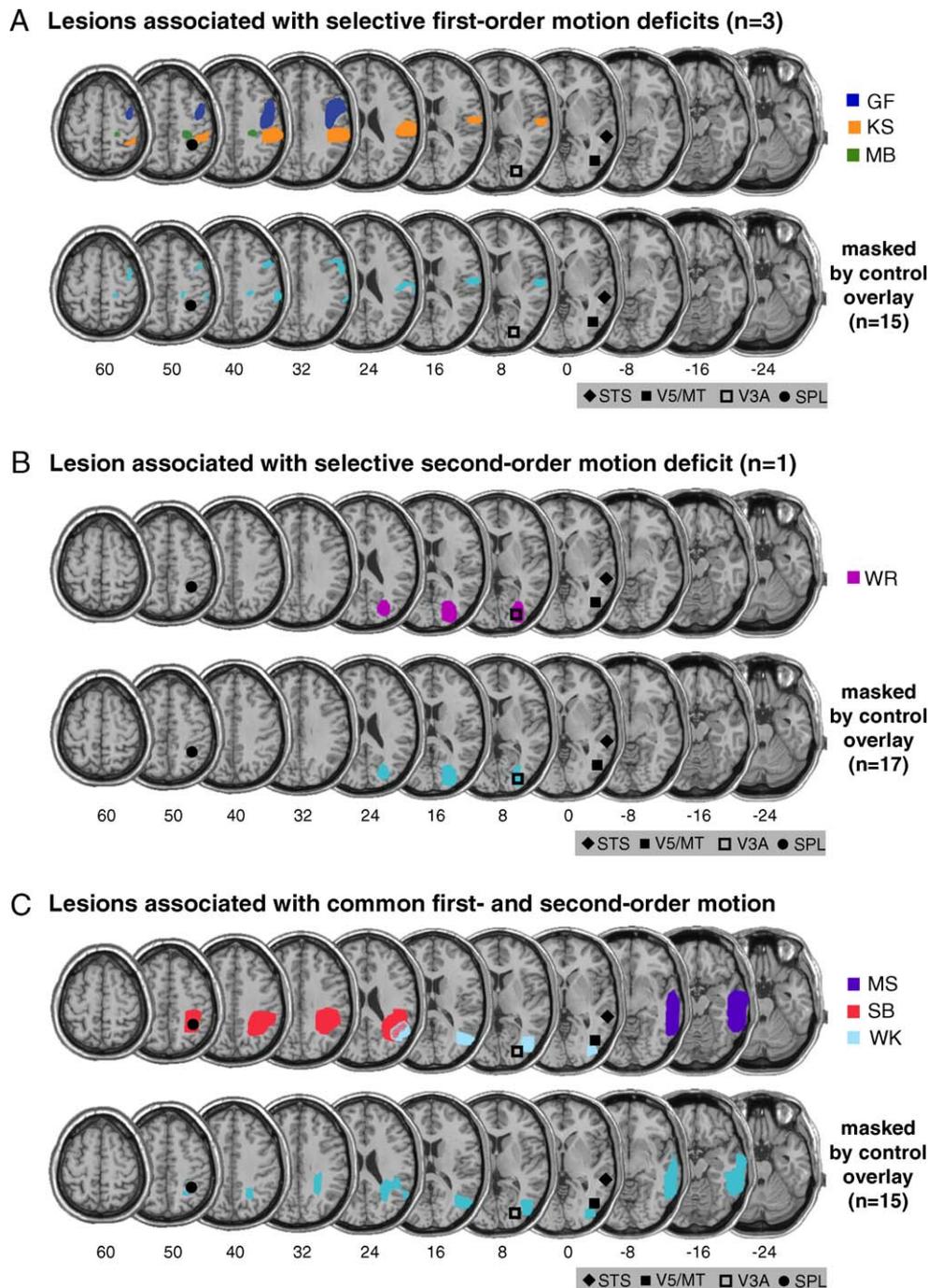


Fig. 5. (A) Lesion plot of patients who showed a selective deficit for first-order motion perception ($n=3$). The upper panel gives the lesions of individual patients in different colors. The lower panel gives the overlay lesion plot of these patients masked by superimposed lesions of control patients ($n=15$). (B) Lesion plot of the patient who showed a selective deficit for second-order motion perception ($n=1$). The upper panel gives the lesion of the patient in magenta. The lower panel gives the overlay lesion plot of this patient masked by superimposed lesions of control patients ($n=17$). (C) Lesion plot of patients who showed a common deficit for first-order and second-order motion perception ($n=3$). The upper panel gives the lesions of individual patients in different colors. The lower panel gives the overlay lesion plot of these patients masked by superimposed lesions of control patients ($n=15$). Talairach z-coordinates (Talairach & Tournoux, 1988) of each transverse section are given. Suspected critical functional locations are marked: STS, superior temporal sulcus (Noguchi et al., 2005); V5/MT, fifth visual area/middle temporal area (Watson et al., 1993); V3A, visual area 3A (Smith et al., 1998); SPL, superior parietal lobule (Dumoulin et al., 2003).

3.2.3.2. Lesions. Fig. 5C illustrates the lesions of patients showing a common deficit for first- and second-order motion perception. Lesions of patients SB, and WK were located in the right hemisphere, whereas patient MS exhibited a left-sided lesion. Lesions overlapped only to a minor degree. Patient MS suffered from a temporal lesion that affects the anterior superior and medial temporal lobe. In contrast, lesions of

patients SB and WK touched the temporal lobe only marginally. Patient SB's lesion was located in the high parietal lobe and bordered ventrally the superior temporal lobe. Patient WK showed a lesion that was predominantly located in the occipital lobe, but also covered the occipito-temporo-parietal junction. Lesions of both patients overlapped in the temporo-parietal area.

Superimposed lesions of the 15 patients without common first- and second-order motion deficits were applied as a mask. The lesion sites that remained after the masking procedure are shown in the lower part of Fig. 5C. There was only moderate overlap between the lesions of patients MS, SB, and WK and lesions of the control patients. The mask reduced the functionally specific areas in the parietal lobe substantially, but left critical occipito-temporo-parietal and temporal areas almost unchanged. Damage to these areas thus appeared unique to common impairment of first- and second-order motion perception.

4. Discussion

We attempted to use challenges to neuronal functioning as a window to analysis of first- and second-order motion. Probably no other visual attribute has attracted more efforts to unveil the precise relationship between its perception and the underlying neuronal circuitry than motion (for review see Albright & Stoner, 1995; Nakayama, 1985). Indeed, detailed understanding of motion processing in striate and early extrastriate areas up to area V5/MT has been achieved. The vast majority of findings support specialized processing pathways for first- and second-order motion in early visual areas. Although there is substantial evidence for functional contributions of higher cortical areas to motion processing (compare Billino et al., 2009; Culham et al., 2001; Sinaert et al., 1999), their involvement in first- and second-order motion perception has remained elusive. Some functional imaging studies suggested specialized processing beyond early visual areas (Dumoulin et al., 2003; Noguchi et al., 2005), but in contrast patient studies rather indicated shared resources (Braun et al., 1998; Greenlee & Smith, 1997; Nawrot et al., 2000). Thus, knowledge still appears fragmentary and further elaboration is needed. We studied functional vulnerabilities due to normal ageing and focal cortical brain lesions in order to advance our understanding of higher cortical contributions to first- and second-order motion processing. Our study includes one of the largest samples in which age effects on first- and second-order motion perception have been studied so far. Our clinical sample including 18 patients adds to results from case studies and stands out due to its consideration of more widely distributed lesion localizations.

4.1. Perceptual decline during normal ageing

We measured motion detection thresholds using luminance modulated as well as contrast modulated gratings in a large sample of 102 subjects ranging in age between 20 and 82 years. Sample size and age range was supposed to yield detailed insights into the association between age and perceptual thresholds. Our data provided strong support for an age-related sensitivity decline for first- and second-order motion. Decline appeared to develop continuously over age. Most notably we determined equivalent vulnerabilities for both motion types.

Detection thresholds for first-order motion correlated moderately with age, $r(102) = .497$. This correlation concurs well with previous reports on age-related sensitivity decline for luminance-defined motion information. Using random dot kinematograms, studies found correlations between age and detection thresholds ranging between $r = .37$ and $r = .51$ (Billino, Bremmer, & Gegenfurtner, 2008; Tran, Silverman, Zimmerman, & Feldon, 1998; Trick & Silverman, 1991). For second-order motion perception a stronger correlation with age was found, $r(102) = .728$. The association between second-order motion perception and a broad range of age has only been described in the study of Tang and Zhou (2009). They reported similar results for a linear model, with $r = .882$. Differential correlations indicated that both motion types are asso-

ciated with age to a different degree. Whereas age explained 25% of variance in first-order motion perception, 53% of variance in second-order motion perception could be attributed to age differences. However, slope of threshold increase turned out to be equivalent for both motion types. Thresholds approximately doubled from age of 20 to age of 80. Findings suggested that first- and second-order motion perception share functional substrates that show age-related decline. We also found support for common processing resources that were not affected by age, but they made a minor contribution.

What can be learnt from the observed age effects about separability of processing mechanisms for first-order and second-order motion? Our results emphasize similar vulnerabilities and thus suggest common functional pathways for processing of both motion types. Ageing offers the opportunity to observe the behavioural consequences of changing neuronal substrates. Findings from neurochemical and structural imaging have provided evidence that specific brain regions age at quite different rates (e.g. Grachev & Apkarian, 2000, 2001; Kochunov et al., 2005; Pieperhoff et al., 2008; Raz et al., 2005; Sowell et al., 2003). Given that first- and second-order motion perception showed an equivalent age-related decline, we suppose that processing of both motion types involves overlapping neuronal resources. We further assume that shared substrates are in particular located in higher cortical areas. Age-related changes in volume and morphology have been found to be much more pronounced in parietal, temporal, and frontal areas than in the occipital lobes where only moderate decline has been observed.

Our findings deviate from previous results reported by Habak and Faubert (2000) and Tang and Zhou (2009). Both studies found differential age effects on first- and second-order motion perception. Furthermore, Tang and Zhou (2009) described an exponential decline of first- and second-order motion perception whereas we observed threshold increases that appear to emerge gradually during ageing. These divergent results call for a critical consideration of methodological differences between our study and both earlier studies.

We consider the drift speed of our stimuli as a major issue that might have contributed to our differing findings. In contrast to previously used paradigms, we chose a relatively high drift speed of $10.1^\circ/s$. We were particularly interested in extrastriate motion processing which especially supports perception of higher velocities (compare Rodman & Albright, 1987). Both previous studies focused on lower velocities ranging between $1^\circ/s$ and $8^\circ/s$. Allard and Faubert (2008) recently proposed that first- and second-order motion mechanisms are distinct at low, but common at higher velocities. Our data unfortunately do not allow for a detailed differentiation between these supposed second-order motion processing mechanisms dependent on temporal frequency. Since an extensive variation of drift speeds lay beyond the scope of our study, our conclusions clearly have to remain limited. However, we suggest that the discrepancy between previous results on age effects on second-order motion perception and our own findings might expand evidence of different processing mechanisms specific to velocity. Due to the higher drift speed our stimuli might have tapped a different second-order processing mechanism that shares neuronal substrates with first-order motion processing resulting in comparable age effects.

Another potentially relevant detail of our study concerns the procedure how motion perception thresholds were determined. In contrast to earlier studies, we considered motion detection rather than motion direction identification. Bennett, Sekuler, and Sekuler (2007) suggested a divergence between motion detection and motion discrimination measures. Modeling motion perception in a sample with a broad age range they found that detection and direction identification are constrained by different mechanisms

during ageing. We decided to focus on motion detection as the more direct measure of motion sensitivity. Thus, we propose that our findings complement previous results which cover direction identification and emphasize the need to differentiate between ageing of motion detection and motion discrimination.

With regard to the course of threshold increase during ageing we attach significant importance to our specific manipulation of signal intensity. We refrained from manipulating contrast to derive perceptual thresholds, but chose a manipulation of signal-to-noise ratios in our stimuli. We supposed that this procedure could reflect age-related threshold differences due to processing changes beyond early striate cortex more clearly. Motion information was carried by gratings of low spatial frequency to preferably avoid the confounding effect of age-related contrast sensitivity decline which is present for higher spatial frequencies (Owsley, Sekuler, & Siemsen, 1983). Previous studies concerned with ageing and motion perception that directly varied signal-to-noise ratios also reported linear threshold increases (Billino et al., 2008; Tran et al., 1998; Trick & Silverman, 1991). It should be noted that linear threshold increases parallel the linear volume loss that has been described for most brain regions except the hippocampus (Raz et al., 2004). Exponential decline reported by Tang and Zhou (2009) might be explained by manipulation of contrast which confounds the differentiation between central ageing processes and age-related changes of the optics of the eye.

Finally, significance of two further technical issues of our study should be considered. Whereas both previous studies on ageing of first- and second-order motion processing presented single stimuli in the central visual field, our stimuli were presented in 4-alternative forced choice paradigms extending to an eccentricity of 7.1°. We suggest that both extra-foveal presentation and more complex spatial configuration of our stimuli do not interfere with the interpretation of our results. Sensitivity to first- and second-order motion falls off with increasing eccentricity at comparable rates (Smith, Hess, & Baker, 1994; Smith & Ledgeway, 1998). Furthermore, we assume that spatially distributed stimulus presentation, which is supposed to emphasize age-related losses (compare Faubert, 2002), puts equivalent attentional demands on processing of both motion types. We cannot definitely exclude that observed age effects on motion perception are to a certain degree confounded with age-related decline of attentional capacity. However, we found no evidence of enhanced age-related perceptual decline due to spatial complexity of stimuli. Correlations with age for first- and second-order motion agreed well with findings from previous studies which used much simpler spatial stimulus configurations (see above). In summary, it appears rather unlikely that the specific spatial characteristics of stimulus presentation have biased our results and could explain their disagreement with results reported by Habak and Faubert (2000) and Tang and Zhou (2009).

Our findings provide support for shared processing pathways for first- and second-order motion. We are aware of the discussed methodological issues that might limit our conclusion and deserve careful consideration. However, we propose that our data allow for a valid comparison between specific age effects on motion perception. Results from our large sample covering a broad age range clearly show equivalent age-related vulnerabilities that point to overlapping neural substrates. Faubert (2002) proposed a theory of visual perception and ageing that strongly emphasizes the complexity of functional networks. Vulnerability of a function is considered to be primarily determined by the number of necessary processing steps. In consideration of recent converging evidence of regional specificity of age-related changes in the brain we propose to complement this perspective by more direct associations between functional decline and differential ageing of critical brain areas.

4.2. Perceptual deficit profiles in brain-lesioned patients

We investigated first- and second-order motion perception in patients with focal cortical lesions in order to get further insights into functionally involved neural substrates. There is a history of neuropsychological case studies concerned with the processing mechanisms of both motion types (e.g. Plant & Nakayama, 1993; Vaina & Cowey, 1996; Vaina et al., 1998). However, the majority of previous studies included only few patients and focused on lesions in early occipital cortex (but see Braun et al., 1998; Greenlee & Smith, 1997; Rizzo et al., 2008). We collected threshold data from 18 stroke patients who had lesions located at widely distributed cortical regions. Comparison with age-specific threshold predictions yielded significant deficits in seven patients. We found selective deficits for first-order motion in three patients, for second-order motion in one patient, and common deficits for both motion types in three patients. Behavioural data and lesion analysis particularly indicated that there are shared mechanisms for first- and second-order motion perception. Evidence for specific processing steps was less pronounced. Thus, patient data supported that our motion stimuli activated differential processing systems, but pointed to substantial overlap of functional substrates.

Although patients' lesions were carefully analyzed, we are aware that quality of clinical scans and interindividual differences limit the detailed identification of critical brain areas. However, our findings clearly confirmed that many cortical areas are involved in motion analysis (compare Billino et al., 2009; Culham et al., 2001; Rizzo et al., 2008; Sunaert et al., 1999). Lesions in our patients showing deficits for motion perception were determined near the well-studied motion complex in occipito-temporo-parietal junction, but also in the temporal, parietal, and frontal lobes.

Lesion analysis in our patients with selective first-order motion deficits was hindered because their lesions overlapped substantially with lesions in the control patient group. Functional relevance of their lesions in the posterior frontal and the parietal lobes therefore remained ambiguous. Affected regions might simply have been more susceptible to damage due to vasculature (see Rorden & Karnath, 2004). Functionally specific areas identified by the masking procedure appeared rather small and little focused. Their primary localization in the parietal lobes might support a specialization of the dorsal pathway for first-order motion processing that has been proposed by Vaina and Soloviev (2004). However, conclusions should be drawn with caution. Data above all suggested that first-order motion processing mechanisms show a cortical distribution and do not exclusively rely on early visual cortex. Plant and Nakayama (1993) speculated that a wide distribution could make these mechanisms more robust to brain damage, but their study included only patients with occipital lesions. In our patient sample with distributed lesion sites first-order motion perception did not seem to be less vulnerable than second-order motion perception. Finally, previous lesion and functional brain imaging studies determined specific functional relevance for first-order motion perception primarily in early occipital cortex (Dumoulin et al., 2003; Rizzo et al., 2008; Smith et al., 1998; Vaina et al., 1998). Our study though focused on extrastriate lesions. We might have failed to identify specific functional areas simply because our sample did not include the critical lesion localizations.

Results on second-order motion perception confirmed that patients with a selective deficit are rare (compare Rizzo et al., 2008; Vaina & Soloviev, 2004). A selective deficit was found only in one patient who had a lesion near the occipito-parietal junction. This lesion did not overlap with lesions of control patients so that it can be considered as functionally specific. Although lesion localization had to remain coarse due to the above mentioned restriction, we suppose that it covered relatively early visual areas including area V3. A specific functional contribution of area V3 to second-order

motion processing was shown before by Smith et al. (1998) using functional magnetic resonance imaging (fMRI). In addition, selective deficits in patients reported by Plant and Nakayama (1993) were also associated with occipital lesions. Our lesion data did not support a specific contribution of higher extrastriate areas to second-order motion processing. We thus cannot confirm a critical functional role of parietal and temporal areas for which recent imaging studies reported significant involvement, i.e. the superior parietal lobule and the superior temporal sulcus (Dumoulin et al., 2003; Noguchi et al., 2005). Vaina et al. (1999) likewise attributed the selective second-order motion deficit in their case study to damage in the superior temporal sulcus. However, in consideration of the close proximity of different functional areas in the occipito-temporo-parietal junction and interindividual variations in their exact localizations, we consider our case report rather complementary than contradictory. In this context, it appears noteworthy that our patient's thresholds for first-order motion perception lay just under the upper 90% limit of the age-specific prediction. This might indicate that his lesion is closely adjacent to areas in the occipito-temporo-parietal junction that are known to be involved first-order motion processing, i.e. the V5/MT complex (see e.g. Dumoulin et al., 2000; Plant et al., 1993; Schenk & Zihl, 1997; Zihl, von Cramon, & Mai, 1983).

We observed common deficits for first- and second-order motion in three of our patients. Their lesions covered parietal and temporal areas as well as the occipito-temporo-parietal junction. Applying the control mask reduced primarily critical areas in the high parietal lobe, but supported that damage to the other areas is specifically associated with the given deficit profile. These findings were in line with previous reports on patients showing first- and second-order deficits. Braun et al. (1998) found three of those patients whose lesions overlapped near the occipito-temporo-parietal junction, putatively covering area V5/MT. Although another single case study by Nawrot et al. (2000) just described transient common deficits, it congruently emphasized the functional relevance of the occipito-temporal region for the perception of both motion types. We assume that the lesion in our patient WK also affected the motion complex V5/MT. Indeed, his lesion appeared very similar to the lesion of our patient with a selective deficit for second-order motion, but was located more anterior. This similarity again pointed to the extraordinary density of functionally specialized areas in the occipito-temporo-parietal junction. Greenlee and Smith (1997) reported on three different patient groups who all showed deficits for both motion types, namely patients with inferior temporal damage, with inferior parietal damage, and with damage to the superior temporo-occipital border region. Lesions in our two patients with parietal and temporal lesions, respectively, coarsely fit in the former groups.

With regard to retinotopy of motion deficits studies have yielded heterogeneous results. Unilateral lesions have been associated with disturbed motion processing in both visual hemifields as well as with deficits restricted to the contralesional visual field (compare e.g. Braun et al., 1998; Rizzo et al., 2008; Schenk & Zihl, 1997). We found significant differences between psychometric functions for both visual hemifields only in our patients with common first- and second-order motion deficits. Since their lesions affected higher cortical areas, we consider retinotopic deficits as implausible. We suppose that attentional biases might have contributed to higher thresholds in the contralesional visual hemifield. Our screening procedure ruled out pronounced attentional deficits, in particular biases in spatial attention, but more subtle biases might have remained unnoticed.

Lesion studies in patients offer a unique possibility to improve our understanding of the association between brain activity and specific functions. Although patient studies allow only insuffi-

cient control on lesion location and plasticity processes, they offer the important advantage that they provide insights into critically required, not just involved, functional substrates. They thus represent an important complement to functional imaging studies. Our findings in patients emphasized convergence of processing pathways for first- and second-order motion at higher cortical levels. Support for separability of processing mechanisms in early extrastriate cortex remained ambiguous, but this might have been due to our focus on lesions affecting higher cortical areas. Our data on motion perception deficits after damage to a variety of different brain regions draws further attention to cortically distributed networks for motion processing (compare Billino et al., 2009; Culham et al., 2001; Sunaert et al., 1999). An exclusive focus on the V5/MT complex as motion area seems no longer justified. Motion processing deficits might actually be present in many patients with quite different brain lesions, but they are probably underestimated in clinical practice (see also Kerkhoff, 2000). We determined motion deficits in almost 40% of our patients.

4.3. General conclusion

We used challenges to normal neural processing in order to get insights into the separability of mechanisms underlying first- and second-order motion perception. Age-related changes in specific perceptual performance provided clear evidence of equivalent vulnerabilities. We consider this as suggestive of overlapping functional substrates that are prone to age-related decline. Since imaging studies have pointed out that volume loss during ageing is more pronounced in higher cortical areas than in early visual cortex (e.g. Sowell et al., 2003), we further suppose that shared functional substrates are in particular located in higher cortical areas. Deficits for first- and second-order motion processing in patients with focal lesions located at widely distributed cortical areas complemented our data on ageing. Lesion analyses emphasized convergence of processing pathways at higher cortical levels, but left possible specific contribution of earlier visual areas ambiguous. It should be noted that distribution of functionally relevant lesions supported a large network of areas involved in motion processing. Contributions of specific areas remain to be clarified. We are aware that dissociated vulnerabilities would allow for stronger conclusions about underlying functional systems. Given the ongoing debate on the differentiation between first- and second-order motion processing, however, our converging findings of commonality between perception of both motion types contribute significantly to a more detailed understanding. Although some non-invasive techniques like transcranial magnetic stimulation allow for temporary disruption of brain activity, neuronal functioning in humans generally defies direct experimental control. The combination of different approaches focusing on given damage or naturally occurring functional changes offers an excellent opportunity to study the neuronal mechanisms underlying specific perceptual or behavioural capacities in humans. Our study supports the recently growing interest in interindividual differences as a window to biological mechanisms (compare also Wilmer, 2008).

Acknowledgement

This research was supported by the research training group 'Neuronal Representation and Action Control – NeuroAct' (German Research Foundation, DFG 885/1). We are indebted to the Generation Research Program of the Ludwig-Maximilians-Universität München, the Neurologische Klinik Braunfels, and all participants for support during data collection.

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