Rollvection Versus Linearvection: Comparison of Brain Activations in PET

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Abstract: We conducted a PET study to directly compare the differential effects of visual motion stimulation that induced either rollvection about the line of sight or forward linearvection along this axis in the same subjects. The main question was, whether the areas that respond to vection are identical or separate and distinct for rollvection and linearvection. Eleven healthy volunteers were exposed to large-field ($100^\circ \times 60^\circ$) visual motion stimulation consisting of (1) dots accelerating from a focus of expansion to the edge of the screen (forward linearvection) and (2) dots rotating counterclockwise in the frontal plane (clockwise rollvection). These two stimuli, which induced apparent self-motion in all subjects, were compared to each other and to a stationary visual pattern. Linearvection and rollvection led to bilateral activations of visual areas including medial parieto-occipital (PO), occipito-temporal (MT/V5), and ventral occipital (fusiform gyri) cortical areas, as well as superior parietal sites. Activations in the polar visual cortex around the calcarine sulcus (BA 17, BA 18) were larger and more significant during linearvection. Temporo-parietal sites displayed higher activity levels during rollvection. Differential activation of PO or MT/V5 was not found. Both stimuli led to simultaneous deactivations of retroinsular regions (more pronounced during linearvection); this is compatible with an inhibitory interaction between the visual and the vestibular systems for motion perception. *Hum. Brain Mapp.* 21:143–153, 2004.

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INTRODUCTION

Unlike vestibular stimuli, which lead to the sensation of motion or tilt of the body, visual motion stimuli can be interpreted as either self-motion or surround-motion. Vestibular information about self-motion is evoked only through acceleration and deceleration and dies out as the cupulae in the semicircular canals and the otoliths progressively return to their resting position during constant velocity. Visually induced apparent self-motion (vection) [Tschermak, 1931] is essential for the adequate perception of self-motion at constant velocity. The sensation of vection can be induced in a stationary observer by large-field visual motion stimulation [Brandt et al. 1973]. The direction of induced vection is opposite to that of visual pattern motion. Vection

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may result from rotation about one or several of three head axes (x , y , z ; corresponding to roll, pitch, and yaw planes), in which case it is referred to as circularvection (CV). Linear motion along these axes (x : forward/backward, y : right/left, z : upward/downward) is called linearvection (LV). A widely accepted and useful nomenclature for the planes and cardinal axes, as well as the polarity conventions for head-referenced movement have been proposed by Hixson et al. [1966].

In an earlier PET study [Brandt et al., 1998], we found that visually induced vection in roll activated a medial parieto-occipital brain area (PO) bilaterally while simultaneously deactivating the posterior insula and adjacent retroinsular regions (the human homologue of the multisensory parieto-insular vestibular cortex of the monkey, PIVC) [Grüsser et al. 1990a,b; Guldin and Grüsser, 1996]. Deactivations of the vestibular cortex during large-field visual motion stimulation were confirmed by Previc et al. [2000] in PET and by Kleinschmidt et al. [2002] in fMRI. This led to the concept of an inhibitory reciprocal visual-vestibular interaction as a form of sensori-sensory interaction for orientation in space and perception of vection [Brandt et al., 1998]. The functional significance of an inhibitory intersensory interaction is obvious; it allows a potential mismatch between two incongruent and misleading inputs to be suppressed by shifting the sensorial weight to the dominant and more reliable modality.

The differential effects of LV and CV in various planes on activations in visual cortical areas are not yet clear. Beer et al. [2002] compared visually induced vection in roll, yaw, and linear movement in depth and found responses in a posterior-inferior temporal area, in the paralimbic cortex, pulvinar, midbrain tegmentum, and in the kinetic occipital region (KO), an area located on the occipital pole approximately 20 mm behind MT/V5, which is involved in the processing of kinetic borders [Dupont et al., 1997]. In the study by Beer et al. [2002], a region in the right insula appeared to respond specifically to roll motion. Sites apparently selective for linear movement in depth were identified in the right cerebellum and in both temporal lobes. The localization and the extent of activations of cortical areas during apparent roll motion or forward motion vary considerably in brain activation studies [Beer et al., 2002; Brandt et al., 1998; de Jong et al. 1994; Kleinschmidt et al., 2002; Previc et al., 2000]. This makes it difficult to determine the critical cortical structures involved in visually induced self-motion. The incongruity of the data can be explained to some extent by different methodological approaches, such as (1) the choice of the control condition (stationary pattern or coherent or incoherent motion stimulation), (2) the stimulus pattern (i.e., size, density of structures), (3) eye movements induced by motion stimulation, (4) influences of attention, and (5) the extent of the stimulus field, which critically determines the intensity of vection.

We conducted a PET study to directly compare the differential effects of large-field visual motion stimulation that induced either CV about the line of sight (rollvection, RV) or

forward LV along this axis in the same subjects. These two vection-inducing stimuli were also compared with a stationary visual pattern. Since RV and LV may have different consequences for ocular motor and postural control, the main questions were: Are the areas that respond to vection identical or separate and distinct for RV and LV? Do deactivations in vestibular cortical regions differ during RV and LV?

SUBJECTS AND METHODS

Subjects

Eleven healthy male subjects (right-handed, 29 to 60 years of age, mean age 37.1 years) without any history of visual (except for corrected refraction anomalies), vestibular, or CNS disorders participated in the study. The laterality quotient for handedness according to the 10-item inventory of the Edinburgh test [Oldfield, 1971; Salmaso and Longoni, 1985] was +100 in eight, +80 in two, and +40 in one subject. Handedness was determined because of the possibility of differential effects due to hemispheric dominance. Informed written consent was obtained from all subjects in accordance with the Declaration of Helsinki after the experimental procedure was explained. The study was approved by the Ethics Committee of the Technical University (Munich) and the local radiation protection authorities.

Visual Motion Stimulation

During PET data acquisition subjects lay supine. Goggles were placed directly over their eyes in order to mount a display with a field of visual stimulation subtending 100° in horizontal and 60° in vertical dimensions (head-mounted display ProView™, Kaiser Electro-Optics). Texture patterns were generated by a Silicon Graphics O2 workstation and rendered onto VHS video tapes. Computer images were presented by a video recorder (VC-MH 761 GM, Sharp). The room with the PET scanner was darkened. Subjects were asked to look at the center of the display and not to follow any moving dots with the eyes. Three stimuli were presented: **STAT (A)**: The stationary optical flow pattern (see B) served as control stimulus. **Linearvection (B)**: An optical flow pattern consisting of about 150 distinguishable green dots (diameter: one pixel, 6° average speed: 8°/sec) on a black background was rendered by perspective projection. Dots seemed to appear in the distance and move toward the subjects. Dots accelerated from the focus of expansion in the center of the screen toward the edge of the screen and increased in size. **Rollvection (C)**: About 250 dots (diameter: 1.3–3.4 degrees) rotated counterclockwise from the subject's viewpoint at a constant angular velocity of 26 degrees/sec. The different amount of dots during LV and RV was chosen in order to generate stimuli that induce vection of similar intensity.

A comparable RV stimulus has been demonstrated elsewhere to elicit a weak, irregular, rotatory nystagmus with a maximum frequency of 0.5–2 Hz, a maximum amplitude of 0.5–3°, and a maximum slow-phase velocity of 1–8 degrees/sec [Brandt et al., 1998].

After the scanning procedure, 7 of the 11 subjects were exposed to the same visual stimuli presented by the same head-mounted display outside the PET scanner in a darkened room while sitting. In order to compare the intensity of forward LV and RV, they were asked to scale vection by assigning arbitrary values from 0 (experience of surround-motion only) to 5 (self-motion only) to each condition [Brandt et al., 1998].

PET Data Acquisition and Analysis

The regional distribution of radioactivity was measured with a Siemens/ECAT EXACT HR+ PET scanner (CTI, Knoxville, TN) operated in 3-D mode with a total axial field of view (FOV) of 15.28 cm and no interplane dead space. Sixty-three slices (matrix: 128×128 , voxel size: $2.06 \times 2.06 \times 2.43 \text{ mm}^3$) covering the whole brain and large parts of the cerebellum were acquired for each subject. Twelve sequential rCBF PET image sets were obtained for each subject using the $\text{H}_2\text{O}-15$ semi-bolus technique. An infusion pump was used to administer a dose of 370 MBq in 20 ml of normal saline solution for each of the 12 scans over 30 sec. Single frames were acquired for 50 sec after the tracer appeared in the brain. There was a 10-min-long interval between the acquisitions. Each condition was presented three times in random order. The visual stimulation started 10 sec prior to the onset of the radioactive bolus injection and continued over the whole scanning time.

Images were analyzed on an UltraSPARC workstation (Sun Microsystems) using statistical parametric mapping (SPM99; online at <http://www.fil.ion.ucl.ac.uk/spm>) implemented in MATLAB (Mathworks, Sherborn, MA). Two scans of one subject had to be excluded due to technical problems with the visual stimulus. All volumes were re-aligned to the first scan of the series to correct for subject motion. Volumes were spatially normalized into the standard stereotactic space defined by the Montreal Neurological Institute (MNI) template [Friston et al., 1995a]. Resulting images contained 91 planes with voxel sizes of $2 \times 2 \times 2 \text{ mm}^3$. Prior to statistical analysis, each image was smoothed with an isotropic Gaussian kernel of 12 mm (full width at half maximum) to compensate for intersubject gyral variability and to attenuate high-frequency noise. To correct for variations in global flow across subjects and scans, an analysis of covariance (ANCOVA) was applied with global flow as the confounding variable [Friston et al., 1990, 1995b]. Mean rCBF was scaled to an arbitrary level of $50 \text{ ml dl}^{-1} \text{ min}^{-1}$. Statistical parametric maps (SPM) were calculated on a voxel-by-voxel basis using the general linear model [Friston et al., 1995b] and the theory of Gaussian fields [Worsley and Friston, 1995]. Both single-subject and group statistical comparisons were performed. SPMs for the group were created by entering the results from the first level statistics into a *t*-test, according to the random effects model [Friston and Pocock, 1992; Woods, 1996]. In addition to subtraction analyses, a conjunction analysis (LV-STAT and RV-STAT) was performed to identify areas that respond both to LV and RV. Activation and deactivation foci that were significant at

a threshold of $P < 0.001$ in group analyses are reported. For single-subject analyses, a significance level of $P < 0.01$ was accepted for signal changes in brain areas that were expected to show signal changes on the basis of previous PET experiments (a priori hypothesis). Some data of single-subject analyses for $P < 0.001$ are also reported. MNI coordinates as well as defined anatomic landmarks [Naidich and Brightbill, 1996; Stephan et al., 1997; Yousry et al., 1997] and the atlas of Talairach and Tournoux [1988] were used to define anatomical sites of activation and deactivation clusters.

To determine brain areas showing hemispheric predominance, a paired Student's *t*-test was performed on the number of activated voxels per hemisphere ($P < 0.05$). Only right-handed subjects with a laterality quotient for handedness of $+100$ were included ($n = 8$). Additionally, images were flipped along the midsagittal plane and compared with unflipped images using a paired *t*-test model in SPM ($P < 0.001$).

RESULTS

Reported Vection

The optic flow pattern (B) induced LV forward (along the line of sight), while visual motion stimulation about the line of sight (C) induced apparent clockwise self-rotation in roll in the subjects. The stationary dot pattern (A) did not induce any apparent self-motion. Mean magnitude estimates of the intensity of vection were slightly higher for RV (mean 3.36; SD 0.98) than for LV (mean 2.64; SD 0.43), but this difference was not significant ($P = 0.198$; Student's *t*-test).

Rollvection vs. STAT (Group Analysis)

Confluent activations were found bilaterally in the visual cortex (BA 17–BA 19) including PO, V3A [Tootell et al., 1997], and the middle temporal/middle occipital gyri (BA 19/37), representing the motion-sensitive area MT/V5, and extended into the fusiform gyri (BA 37) bilaterally (Figs. 1B, 2B, Table I). Bilaterally, parts of BA 17 and BA 18 on the occipital pole around the calcarine sulcus were spared, and only upper parts of BA 17 showed activations. Bilateral activations were also seen in the posterior superior parietal lobule (BA 7), extending into the intraparietal sulcus (IPS). Two left-sided signal increases were found in the inferior parietal lobule (BA 40). Additional right-sided activations were seen in the precuneus (BA 7), in the precentral (BA 6), and in the middle temporal (BA 21/37) gyri.

In the comparison RV-STAT, a predominance of the left hemisphere for the mediation of clockwise vection in roll was observed (Student's *t*-test; $P = 0.037$). This was primarily due to differential activation of MT/V5 (MNI coordinates x, y, z : $-48, -74, 0$). Notably, an area in the inferior parietal lobule, extending into the supramarginal gyrus (MNI coordinates x, y, z : $62, -42, 30$), showed a stronger activation in the right hemisphere during RV ($P < 0.001$).

Deactivations occurred in the left anterior and posterior cingulate gyri, extending into the left medial frontal gyrus

Figure 1.

Surface rendering of differential brain activations (red) and deactivations (blue) in four subtraction analyses: linearvection compared to a stationary pattern (B vs. A; **A**); rollvection compared to

a stationary pattern (C vs. A; **B**); linearvection-rollvection (B–C; **C**); rollvection-linearvection (C–B; **D**) ($n = 11$, $P < 0.001$). SPMs were projected onto the MNI standard brain template.

(BA 10), in the left precuneus (BA 7), right basal ganglia, and in the right middle temporal (BA 21, BA 39) and left superior temporal (BA 22) gyri. A right-sided cluster located in the basal ganglia (putamen, claustrum) was adjacent to the posterior insula.

Single-subject analyses showed deactivations of the posterior insula or retroinsular regions in six subjects on the right side and in four subjects on the left side ($P < 0.01$). However, group analysis failed to demonstrate a significant deactivation in these vestibular areas, due most likely to considerable interindividual anatomical variation.

Linearvection vs. STAT (Group Analysis)

Bilateral confluent activations were seen in the visual cortex (BA 17–BA 19) including the middle temporal/middle occipital gyri (BA 19/37; MT/V5), V3A, and KO [Van Oostende et al., 1997] and extended bilaterally into PO dorsally and into the fusiform gyri (BA 37) ventrally (Figs. 1A, 2A, Table I). Contrary to the comparison RV-STAT, all parts of BA 17 showed signal changes. The number of activated voxels in the visual cortex (including MT/V5) was 9,734 during LV, i.e., nearly double that of RV (5,978 voxels). We

Figure 2.

Differential brain activations (red) and deactivations (blue) in the comparisons: linearvection–stationary pattern (B–A; **A**); rollvection–stationary pattern (C–A; **B**); linearvection–rollvection (B–C;

C); rollvection–linearvection (C–B; **D**) ($n = 11$, $P < 0.001$). SPMs were superimposed onto transverse sections of the MNI standard brain template.

also saw rCBF increases in the right parahippocampal gyrus (BA 34) and in the left inferior parietal lobule extending to the precuneus (BA 7/40). No left-hemispheric dominance was observed in this comparison (Student's t -test, $P = 0.55$). The comparison of flipped and unflipped images showed stronger activation of visual occipital areas (BA 18 and MT/

V5) on the left side and of two areas in the medial and inferior frontal gyri on the right side ($P < 0.001$).

Deactivations were seen bilaterally in the anterior and posterior cingulate gyri. Right-sided deactivations were found in the superior frontal (BA 9) and superior temporal (BA 38) gyri, putamen and claustrum, adjacent to the pos-

TABLE I. Differential rCBF changes comparing LV, RV, and STAT*

Brain area	BA	RV – STAT			LV – STAT			LV – RV		
		<i>x, y, z</i>	Voxels	<i>t</i>	<i>x, y, z</i>	Voxels	<i>t</i>	<i>x, y, z</i>	Voxels	<i>t</i>
Visual cortex	17–19, 37	–2,–88,18	5105 ^a	11.13	–20,–92,0	9725 ^a	15.34			
	17/18							12,–92,–4	2556	10.03
	18							32,–84,0	53	5.74
	19		5105 ^a					12,–50,0	20	–5.12
MT/V5	19/37	46,–76,0	5105 ^a	9.14	42,–82,–2	9725^a	8.04			
PO	19	–22,–90,26	5105 ^a	6.24	–20,–80,24	9725 ^a	5.31			
PO	19	16,–82,28	5105 ^a	10.29	14,–82,34	9725 ^a	8.76			
MT/V5	19/37	–48,–76,2	873	10.14	–50,–76,–6	9725 ^a	6.91			
GF	37	42,–78,–20	27	6.64						
PCu	7	12,–52,58	35	7.45	–4,–50,42	26	–5.47	22,–58,46	20	–5.50
PCu	7	–4,–52,44	13	–4.56				–12,–74,52	17	–4.68
LPi/PCu	7/40				–24,–48,38	13	5.00			
LPs/IPS	7	30,–58,56	249	7.65				–32,–42,74	38	–7.52
LPs/IPS	7	–32,–58,60	126	5.87						
LPi	40	–50,–34,42	26	6.06	–32,–44,72	24	–7.33	–36,–42,62	29	–6.60
LPi	40	–26,–50,38	25	5.93	–54,–18,22	9	–5.79	–52,–42,50	18	–4.92
LPi	40				–70,–46,30	38	–5.24			
GTs	22	–72,–38,16	6	–5.91				56,–30,20	26	–5.39
GTs	22							–70,–8,2	24	–5.00
GTs	38				42,18,–22	36	–8.10	40,18,–18	23	–4.90
Ga/Gsm	39							38,–52,32	29	–5.24
GTm	21/37	54,–58,4	7	4.95						
GTm	21	60,–16,18	42	–7.17						
GTm	39	46,–72,28	5	–4.58						
Ins p								–36,–16,–2	5	–4.83
Ins p								–38,–38,12	2	–3.29
GFs	8/9				20,48,30	11	–5.04	18,36,54	12	5.65
GFi	47							50,36,–4	8	–5.00
GPrC	6	48,–8,42	82	8.16						
Th a/NC					–18,–2,14	38	–7.34			
Th p					4,–20,12	6	–4.66			
GPi/Pu		30,–12,8	26	–4.84						
Pu/Cl		32,–8,2	4	–4.28	26,–2,8	54	–7.18			
GH	34				24,–8,–22	94	7.79	12,–50,0	20	–5.12
GH	28				–32,8,–28	19	–5.44			
GCa	24				12,0,46	17	–5.33			
GCa	24				6,26,24	175	–7.76			
GCa/GFd	24/32/10	–12,48,0	255	–11.46	–16,46,4	214	–9.05			
GCp	23/30	–2,–52,14	30	–5.36	–8,–50,14	11	–5.24			
Brainstem					2,–30,–12	18	–5.11			

* Brain areas showing activations (bold letters, positive *t*-value) or deactivations (not bold, negative *t*-value) comparing linearvection (LV), rollvection (RV), and a stationary dot pattern (STAT) (subtraction analyses: *n* = 11, *P* < 0.001). Brodman areas (BA), MNI coordinates, cluster sizes, and *t*-values for voxels showing maximum significance are listed. Positive *x, y*, and *z* coordinates indicate locations right, anterior, and superior to the middle of the anterior commissure, respectively.

^aThis cluster is confluent with another listed cluster; the voxel number represents the entire cluster.

G, gyrus; F, frontal; O, occipital; T, temporal; LP, parietal lobule; GC, cingulate gyrus; s, superior; m, middle; i, inferior; d, medial; a, anterior; p, posterior; Ins, insula; GPi, globus pallidus internus; NC, caudate nucleus; Pu, putamen; Cl, claustrum; PCu, precuneus; Ga, angular gyrus; Gsm, supramarginal gyrus; GH, parahippocampal gyrus; GPrC, precentral gyrus; PO, parieto-occipital; IPS, intraparietal sulcus.

Figure 3.

Example of bilateral deactivations in the posterior insula and adjacent retroinsular regions in one subject during apparent linearvection compared to a stationary pattern ($P < 0.01$). The deactivated areas correspond to the parieto-insular vestibular cortex (PIVC). The left-sided cluster extends from $z = -4$ to $z = 16$ (285 voxels); the right-sided cluster extends from $z = 0$ to $z = 10$ (50 voxels).

terior insula, and in the posterior thalamus. There was also an rCBF decrease in the right brainstem, which was located in or close to the inferior colliculus. Left-sided deactivations appeared in the inferior parietal lobule (BA 40), precuneus (BA 7), anterior thalamus (extending to the caudate nucleus), as well as in the parahippocampal gyrus (BA 28).

Single-subject analyses showed deactivations of the posterior insula or retroinsular regions in six subjects on the right side and in three subjects on the left side ($P < 0.01$), an example of which is shown in Figure 3.

TABLE II. Local maxima in the conjunction analysis RV - STAT and LV - STAT ($n = 11$, $P < 0.001$)

Brain area	BA	MNI coordinates (x,y,z)	Voxels	t
Visual cortex	17–19, 37	16,–82,30	16,442 ^a	7.92
PO	19	16,–82,30	16,442 ^a	7.92
PO	19	–22,–88,24	16,442 ^a	5.53
MT/V5	19/37	46,–76,0	16,442	6.43
MT/V5	19/37	–50,–74,–2	16,442 ^a	6.85
GF	37	–44,–66,–18	16,442 ^a	5.16
GF	37	38,–78,–18	16,442 ^a	4.89
Cerebellar hemisph.		12,–60,–26	16,442 ^a	3.29
GF	20	38,–20,–26	29	2.47
LPs/IPS	7	–22,–70,64	118	2.98
LPs/IPS	7	32,–60,60	250	4.01
LPi/PCu	40/19	–26,–48,40	115	4.20
PCu	7	2,–66,62	65	2.52
GH	34	18,0,–20	235	3.18
GPrC	4/6	–58,0,22	102	2.79
GFi	45	56,24,18	58	2.77

^aThis cluster is confluent with another cluster; the voxel number represents the entire cluster.

Conjunction Analysis (LV-STAT and RV-STAT)

The conjunction analysis (group analysis of LV-STAT and RV-STAT) showed bilateral activations in the posterior superior parietal lobule extending into the IPS (BA 7; Fig. 4, Table II). The latter is consistent with the activation in Figure 2B at $z = 58$. Visual cortical activation spared the occipital pole bilaterally around the calcarine sulcus, i.e., parts of BA 17 and BA 18 were spared. Only the upper parts of BA 17 showed signal increases. MT/V5, V3A, area LOS [Sunaert et al., 1999], PO, and the lingual gyri (BA 18) showed bilateral activations. The activation extended into the right cerebellar hemisphere and into the fusiform gyri (BA 37) bilaterally. Two frontal areas, one in the right inferior frontal gyrus (BA

Figure 4.

Conjunction analysis LV-STAT and RV-STAT ($n = 11$, $P < 0.001$). Common activations during linearvection and rollvection are located in occipital (BA 17–19), temporo-occipital (BA 19/37), and superior parietal (BA 7) cortical areas bilaterally. The two clusters in the superior parietal lobule extend into the intraparietal sulcus.

45) and one in the left precentral gyrus (BA 4/6), also responded to both motion stimuli. Further clusters were seen in the right precuneus (BA 7), right parahippocampal gyrus (BA 34), and left inferior parietal lobule (BA 40/19).

Linearvection vs. Rollvection

In the comparison LV-RV (group analysis; Figs. 1C, 2C, Table I), a bilateral cluster was seen in the visual cortex around the calcarine sulcus on the occipital pole ($z < 7$; BA 17, BA 18). BA 19 was spared. No clusters were seen in V3A, MT/V5, PO, or in the fusiform gyri (BA 37). A small right-sided activation appeared in the superior frontal gyrus (BA 8). In the group analysis, no activation was located in the posterior insula. Single-subject analyses revealed a right-sided cluster in the posterior insula in one subject ($P < 0.001$).

In the contrast RV-LV (group analysis; Figs. 1D, 2D, Table I) a weak widespread pattern of small activations occurred. The largest activation with a cluster size of 38 voxels was situated in the left superior parietal lobule (BA 7). Bilateral activations appeared in the precuneus (BA 7) and superior temporal gyri (BA 22, BA 38). Right-sided activations were seen in the inferior frontal (BA 47), lingual (BA 19), parahippocampal (BA 34), and angular (BA 39) gyri. Left-sided activations were seen in the inferior parietal lobule (BA 40), posterior insula, and adjacent retroinsular regions. The size of the activation cluster in the posterior insula increased from 5 to 112 voxels at a lower significance threshold ($P < 0.01$, group analysis). In single-subject analyses three subjects showed a cluster in the right posterior insula, and one subject showed a cluster in the left posterior insula ($P < 0.001$).

DISCUSSION

Common Signal Changes During Linearvection and Rollvection

During LV and RV, *signal increases* were found in striate and extrastriate visual cortical areas including V1 and V2 as well as area V3A, LOS, the lingual gyri, and the motion-sensitive area MT/V5 bilaterally. Signal increases extended bilaterally into PO as well as into the fusiform gyri (conjunction analysis RV-STAT and LV-STAT; Fig. 4, Table II). The region on the occipital pole around the calcarine sulcus was spared bilaterally, and only parts of BA 17 and BA 18 showed signal changes. PO has previously been shown to respond to coherent motion stimulation in roll, which induced RV [Brandt et al., 1998]. LOS and V3A have been demonstrated to be motion-responsive in humans [Sunaert et al., 1999]. De Jong et al. [1994] observed bilateral activations in the fusiform gyri, when subjects viewed an optic flow stimulus. Peuskens et al. [2001] also used an optic flow stimulus in a heading task and found responses in the right

fusiform gyrus, suggesting it may be an inferior satellite of MT/V5 and a human homologue of monkey MSTd.

Other common activations were seen in the left inferior parietal lobule and bilaterally in the posterior superior parietal cortex (extending into the IPS; Fig. 4, Table II). The posterior parietal cortex is known to participate in visuo-spatial tasks, e.g., perception of space, optic flow, and motion-in-depth, and in the control of visuo-spatial attention [for review see Bremmer et al., 2000; Greenlee, 2000]. The bilateral activations in the IPS correspond well to DIPSL described earlier in activation studies using visual motion stimulation [Goebel et al., 1998: apparent motion stimulation and motion imagery, fMRI; Sunaert et al., 1999: small-field motion stimulation, fMRI; Peuskens et al., 2001: optic flow and heading estimation, PET and fMRI]. The maxima observed in our study are located slightly postero-lateral to the maximum of DIPSL described by Sunaert et al. [1999] (x, y, z : 25, -54, 62). Peuskens et al. [2001] suggested that DIPSL/DIPSM is a possible homologue of monkey area 7a, which is thought to be involved in the representation of space, self- and object-motion in the macaque [Phinney and Siegel, 2000; Read and Siegel, 1997]. Activations along the dorsal lip of the IPS in response to coherent visual motion stimulation have also been reported by Dupont et al. [1997], Dieterich et al. [1998], and Cornette et al. [1998].

Common *signal decreases* were seen in the superior temporal and cingulate gyri, left precuneus, in the basal ganglia (putamen, claustrum) of the right hemisphere, as well as in the posterior insula (Table I). Deactivations of the posterior insula (and retroinsular regions) reached the level of significance only in single-subject analyses, probably due to inter-individual anatomical variations. This fits previous findings using galvanic stimulation in fMRI [Bense et al., 2001]. The central part of the vestibular cortex is situated here (PIVC), and the current data confirm a previous PET study showing that large-field visual motion stimulation leads to rCBF decreases in the PIVC [Brandt et al., 1998]. Areas in the superior temporal gyrus, the anterior cingulate gyrus, and the putamen have also been demonstrated to respond to vestibular stimuli in humans [Bense et al., 2001; Bottini et al., 1994; Fasold et al., 2002]. The observation that vestibular stimulation activates the vestibular cortex and simultaneously deactivates the visual cortex, while visual motion stimulation activates the visual cortex and simultaneously deactivates the PIVC, is consistent with the concept of a reciprocally inhibitory visuo-vestibular interaction [Brandt et al., 1998; Deutschländer et al., 2002]. This interaction has functional significance, since it allows the sensorial weight to be shifted to the modality more reliable for the mode of stimulation and thereby avoids perceptual ambiguity [Brandt and Dieterich, 1999]. Deactivation of the vestibular cortex duringvection was originally not expected, since vestibular neurons also carry visual input at the level of the vestibular nuclei, which is transmitted to multisensory cortical areas via thalamic nuclei by trisynaptic afferent pathways [de Waele et al., 2001].

Differential Signal Changes for Linearvection vs. Rollvection

The number of activated voxels in visual areas was considerably higher during LV than during RV (compared to STAT), and the *subtraction LV–RV* revealed contiguous bilateral signal increases in visual areas on the occipital pole (large areas of BA 17 and BA 18, including KO), which extended up to 7 mm above the AC–PC line, but spared BA 19. KO is thought to be involved in processing kinetic boundaries and extracting 2-D shape from motion [Orban et al., 1995; Van Oostende et al., 1997]. KO has also been reported to be sensitive to optical expansion [de Jong et al., 1994], particularly in combination with depth information [Greenlee, 2000; Rutschmann et al., 2000], both of which are characteristics of the LV stimulus used in our study.

Due to the experimental paradigm, dot density in the center of the field of view as well as dot speed in the periphery differed between LV and RV stimulus, and this may have influenced activation patterns. Moreover, LV was inevitably associated with depth perception; subjects reported the illusion of moving forward through a three-dimensional space. Changes in the perception of depth did not occur during RV. Activation studies on horizontal binocular disparities, which provide critical cues for seeing depth, stress the importance of the polar visual cortex for the discrimination of stereo-optic depth information [Gulyas and Roland, 1994]. Backus et al. [2001] observed that activity in the early visual cortex (V1, V2, V3, V3A) was larger for static stimuli with stereoscopic depth induced by binocular disparities than for a single flat plane, while responses in MT/V5 did not significantly differ.

The *subtraction RV–LV* showed a widespread pattern of small rCBF changes in the parietal cortex (inferior and superior parietal lobules), precuneus, and in temporal, frontal, and parahippocampal regions (Table I). A left-sided cluster was seen in the posterior insula, compatible with a stronger deactivation of the PIVC during LV than during RV. The level of activation of MT/V5 as well as of PO did not significantly differ between LV and RV. Thus, our paradigm was not suitable for separating distinct cortical representations of linear and rotational vection in these two areas.

We observed a stronger deactivation of vestibular areas (left posterior insula and retroinsular regions) during LV than during RV. Areas in the superior temporal gyrus (BA 22) and inferior parietal lobule (BA 40) also showed lower activity levels during LV. With regard to functional significance, one can speculate that during visually induced forward LV, vestibular input elicited by head movements may be distressing and interfere with the perceived direction of vection. RV (in upright stance) requires postural reflexes to prevent falls in the roll plane, and, therefore, vestibular suppression appears less desirable. However, subjects in our study lay supine and relaxed without the above-mentioned demands for postural control in the gravitational field. It must remain open as to whether there are differential effects on the activation patterns for RV and LV, which depend on body posture.

Influences of eye movements on the observed activation patterns cannot be fully excluded. During RV, an irregular infrequent torsional nystagmus with low amplitude and low slow-phase velocity could not be suppressed by fixation. To keep optokinetic eye movements as minimal as possible, we chose forward LV and discarded LV stimuli in the frontal plane (upright/downward, right/left). During LV, fixation of the stationary center of the expanding stimulus suppressed optokinetic nystagmus. In an fMRI study by Dietrich et al. [2003], small-field horizontal OKN induced deactivations in vestibular cortical areas (posterior insula, superior temporal gyrus, inferior parietal lobule) and activations in the visual cortex. Since OKN was only seen during RV in our study, stronger deactivations of vestibular areas and stronger activations of visual areas during LV cannot be explained by ocular motor bias.

Neural Substrates for Mediation of Vection and Coherent Motion Perception

The distinct neural substrates that mediate vection are still in dispute [Cumming, 2002]. Cells selective for optic flow have been demonstrated in monkey MSTd, area 7a, in the ventral intraparietal area, the anterior superior temporal polysensory area (STP), and in the superior temporal sulcus [Anderson and Siegel, 1999; Andersen et al., 1990; Baizer et al., 1991; Sakata et al., 1985]. The interpretation of brain signal changes during various visual motion stimulations is complicated by uncertainty as to whether these changes are predominantly due to the coherence of motion stimuli or perception of depth or vection. In addition, other visual stimulus characteristics may influence activation patterns, such as the total area of visual motion stimulation, the location of the stimulus in the visual field, texture, luminance, and direction of gratings. In the following, we compare four activation studies with respect to differential effects of induction of vection, coherence, and perception of depth.

In a PET study by de Jong et al. [1994], coherent optical flow stimulation of the lower visual field ($30^\circ \times 40^\circ$), which induced apparent forward motion over a flat surface and perception of depth, was compared with an incoherent control stimulus. Activations in the right dorsal cuneus (V3), right superior parietal lobule (BA 7), and in the fusiform gyri bilaterally, i.e., in ventral and dorsal visual pathways, were seen during optical flow stimulation. These foci of activation have also been identified in our study (conjunction analysis). Previc et al. [2000] used large-field visual motion stimulation in roll in PET and found deactivations in vestibular areas (posterior insula, parieto-temporal cortex) during coherent and incoherent stimulation. The coherent stimulus induced compelling vection. In a subsequent PET study, Beer et al. [2002] compared large-field coherent visual motion stimuli, which simulated three different kinds of observer movement (roll, yaw, and linear movement in depth), to three corresponding incoherent visual motion stimuli, which were designed to induce weaker vection in the same planes and directions. Brain areas in the left temporal lobe (BA 37), the

pulvinar, amygdala, anterior cingulate gyrus, midbrain tegmentum, and KO were interpreted to respond specifically to the coherence of the stimuli. Furthermore, vestibular areas appeared to be selective for roll motion (right insula, right putamen) as well as for linear movement (right superior and left transverse temporal gyrus). Kleinschmidt et al. [2002] compared BOLD signal changes during the sensation of object motion and self-motion in the roll plane. During vection, early motion-sensitive visual areas (V1, V3, V3A, V4, V5) and the PIVC were deactivated. Thus, the finding of vestibular deactivation during visually induced vection was confirmed. They furthermore demonstrated that higher order visual areas (medial parieto-occipital cortex and V5A/MST) displayed transient activations in response to perceptual switches.

Despite several brain activation studies on vection, coherence, and 3-D processing, the precise location of areas involved in vection along different spatial axes remains uncertain. In conclusion, our PET study on RV and LV using large-field visual motion stimulation showed activations of striate and extrastriate visual areas including PO, V3A, MT/V5, and ventral occipital areas, as well as superior parietal sites extending into the IPS. Activations in the visual cortex around the calcarine sulcus on the occipital pole ($z < 7$; BA 17, BA 18; including KO) were larger and more significant during LV. Areas in the precuneus (BA 7), parietal (BA 7, BA 40), and temporal (BA 22, BA 38, BA 39) cortex showed higher activity levels during rotational than during linear vection. Our study did not show differential activation of PO and MT/V5 during LV and RV. Both stimuli led to simultaneous deactivations of vestibular areas including the posterior insula, which were more pronounced during LV than during RV. Deactivation of vestibular areas was described earlier for roll motion, indicating an inhibitory interaction between the visual and the vestibular systems.

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