

Functional dissociation of saccade and hand reaching control with bilateral lesions of the medial wall of the intraparietal sulcus: Implications for optic ataxia

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The posterior parietal cortex (PPC) is essential for the integration of visuomotor information during visually guided reaching. Studies in macaque monkeys have demonstrated a functional specialisation around the intraparietal sulcus (IPS) with a more medial representation of hand movements (“parietal reach region”) and a more lateral representation of saccadic eye movements (lateral intraparietal area, LIP). Here we present evidence for the validity of this concept with respect to the human parietal cortex. We recorded isolated and combined goal-directed eye-hand movements in normal control subjects and in a patient with bilateral parieto-occipital lesions and incomplete Balint’s syndrome including severe optic ataxia (misreaching to visual targets). Brain lesions in the patient were caused by acute posterior leucoencephalopathy in association with aortic surgery because of Takayasu’s arteritis. MRI scans showed bilateral line-shaped hemorrhagic lesions, restricted to the cortex at the medial banks of the intraparietal sulcus, but leaving its lateral banks largely intact. In the patient visually guided reaching was significantly dysmetric, whereas the metrics of visually guided saccades were within normal limits. Dysmetria was more pronounced for the right visual field, with a gross hypermetria. Variability of the movement improved when a delay of 5 or 10 s was introduced between target presentation and movement execution. Lesion data support the concept of a functional specialisation around the human IPS: The cortex medial to the IPS predominantly controls rapid goal-directed reaching movements, comparable to the parietal reach region in monkeys, whereas saccadic eye movements appear to be controlled rather by the cortex lateral to the IPS.

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Introduction

To a large extent our actions require a goal-directed arm movement towards a visually defined target in the peripersonal space. The information to perform this kind of task is coded in retinal coordinates that depend on the eye position for a given position in space. This information has to be transformed into space coordinates in which the commands for torques of a large number of joints can be coded. The neural process that accomplishes this transformation is referred to as sensorimotor integration.

One brain area that extensively deals with sensorimotor integration is the posterior parietal cortex. It comprises the superior and the inferior parietal lobes that are separated by the intraparietal sulcus (IPS). Single-cell recordings in monkeys revealed subregions around the IPS (Carey, 2000; Colby and Goldberg, 1999). One of them contains neurons that discharge prior to a behaviourally relevant saccadic eye movement to a visual or remembered target position in space (the lateral intraparietal area, LIP, located at the lateral banks of the IPS; Colby et al., 1996; Gnadt and Andersen, 1988). In another subregion located at the medial bank of the IPS (the medial intraparietal area, MIP) neurons are activated when the monkey reaches for a visual target (Colby and Duhamel, 1991). Furthermore, the anterior intraparietal area (AIP) is involved in grasping of and hand shaping for 3D objects. A fourth area in the depth of the IPS (ventral intraparietal area, VIP) is involved in the representation of perioral space (Colby and Goldberg, 1999). Despite this preponderance of the regions for a certain effector (eye or hand), there is considerable overlap in the activating modality (Snyder et al., 2000).

Much effort has been directed towards clarifying whether monkey and human parietal cortex is homologous. At least regions with homologous functions have been described (Culham and Kanwisher, 2001) although their precise location might differ in particular in the parietal cortex. For the study of human cortical organisation functional imaging and lesion studies in patients are

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used. Both techniques focus on different properties of the underlying neural circuitry and may lead to different results (Pierrot-Deseilligny et al., 2004).

Functional imaging has been performed for visually guided saccades and anti-saccades and hand pointing movements (Connolly et al., 2000), for memory saccades and memory pointing (DeSouza et al., 2000; Medendorp et al., 2005; Medendorp et al., 2003), for visually guided saccades, pointing and attending (Astafiev et al., 2003) and for pointing in a subliminally double-step paradigm (Desmurget et al., 2001). There were several foci of activation along the IPS (both medially and laterally to the IPS; for a summary see Table 1 and Fig. 1). In contrast to findings in the monkey, retinotopically modulated activation has been demonstrated medial to the IPS during memory saccades (Schluppeck et al., 2006; Schluppeck et al., 2005). This activation could correspond to monkey LIP. The same paradigm also leads to an activation medial to the IPS (Serenio et al., 2001). Other authors found foci of fMRI activation by triple-step saccades with LIP-like properties in the lateral wall (Heide et al., 2001) or in the posterior end of the IPS (Konen et al., 2004). At least a recent review of the functional imaging, clinical and anatomical literature comes to the conclusion that “the functional relevance of the IPS for visuomotor tasks involving arm and eye movements and visuospatial attention is similar in humans and macaques” (Grefkes and Fink, 2005).

Hand movements were mapped to an area that largely overlapped the saccade area medial to the IPS (Medendorp et al., 2005; Medendorp et al., 2003). Thus, fMRI has not yet provided dissociable activation sites medially or laterally of the IPS with respect to saccade and hand movement control.

In addition to single-cell recordings in animals and functional imaging studies in humans, deficits of hand and/or eye movements after brain lesions in humans can be used to localize homologues of areas MIP and LIP, respectively. Lesion studies confined to the medial or lateral bank of the IPS should be appropriate to identify the cortical area which is essential for a certain function (hand reaching or eye movements), among several areas activated during fMRI.

Impaired saccadic eye movements with an increased latency and reduced accuracy have been demonstrated after parietal lesions (Leigh and Zee, 2006; Morrow, 1996; Pierrot-Deseilligny et al., 1991). An impairment of hand movements that is observed with lesions in the region around the IPS is optic ataxia. Optic ataxia is a condition with impaired goal directed hand movements towards visually presented targets (Battaglia-Mayer and Caminiti, 2002; Perenin and Vighetto, 1988). Clinically, hand pointing is tested to peripheral targets while the gaze remains fixating straight ahead. Optic ataxia may be part of a Balint syndrome (Balint, 1909) together with simultanagnosia and oculomotor apraxia (Rizzo and Vecera, 2002).

Both with respect to eye and hand pointing movements the lesions reported were large and do not allow the localization of the human homologue of LIP and MIP, respectively. In optic ataxia, a number of large lesions overlapped medially of the intraparietal sulcus. However, some of the lesions involved the inferior parietal lobule (Perenin and Vighetto, 1988). In contrast, a more recent study using lesion data from 16 patients could not delineate the superior parietal lobe (SPL) as the crucial lesion site. Most of the patients had a lesion overlap in the junction between the inferior parietal lobule and the superior occipital cortex (Karnath and Perenin, 2005).

We characterized the simultaneous pointing hand and saccadic eye movements in a patient with optic ataxia due to circumscribed bilateral parieto-occipital lesions. The patient showed an anatomical dissociation of her lesions along the IPS: the lesion was confined to the medial wall (putative MIP homologue) but left the lateral wall largely intact (putative LIP homologue). In accordance with the functional anatomy of cortical areas around the IPS we postulate that this anatomical dissociation will be reflected in a functional dissociation: Reaching arm movements to visual targets were expected to be impaired as opposed to saccadic eye movements.

It has been claimed that eye movements are not impaired in optic ataxia on the basis of clinical observation. There are only a few reports which tried to record eye movements to analyse saccade metrics. With respect to their accuracy, however, results

Table 1
Regions in the PPC activated in fMRI studies

Study	Region	Position relative to IPS	Paradigm	Right			Left		
				x	y	z	x	y	z
Connolly et al., 2000	pSPR (eye)	Medial	Saccades	12	-73	39	-11	-73	39
	pSPR (hand)	Medial	Pointing	17	-64	41	-12	-68	38
	aSPR (hand)	Medial	Pointing	-	-	-	1	-47	44
	ISPR (hand)	Medial	Pointing	-	-	-	-35	-49	33
	aIPR (eye)	Lateral	Saccades	39	-47	42	-41	-43	41
	aIPR (hand)	Lateral	Pointing	38	-50	41	-44	-32	33
Schluppeck et al., 2005	IPS1	Medial	Memory-guided saccades	21	-76	27	-21	-76	27
	IPS2	Medial	Memory-guided saccades	18	-71	52	-18	-71	52
Serenio et al., 2001		Medial	Memory-guided saccades	32	-68	46	-	-	-
		Medial	Memory-guided saccades	24	-65	53	-	-	-
		Medial branch of the IPS	Memory-guided saccades and pointing	-25	-59	41	20	-58	42
Medendorp et al., 2005	retIPS	Medial branch of the IPS	Memory-guided saccades	-19	-63	46	21	-62	42
Medendorp et al., 2003	retIPS	Medial branch of the IPS	Memory-guided pointing	-19	-62	46	22	-64	44
Astafiev et al., 2003	aIPS	Lateral		-31	-49	51	-	-	-
	pIPS	Lateral		-30	-61	49	-	-	-

Regions: pSPR, aSPR, ISPR: posterior, anterior and lateral superior parietal region; aIPR: anterior intraparietal region. IPS1, IPS2: intraparietal regions; aIPS, pIPS: anterior and posterior intraparietal region; retIPS: retinotopic intraparietal region.

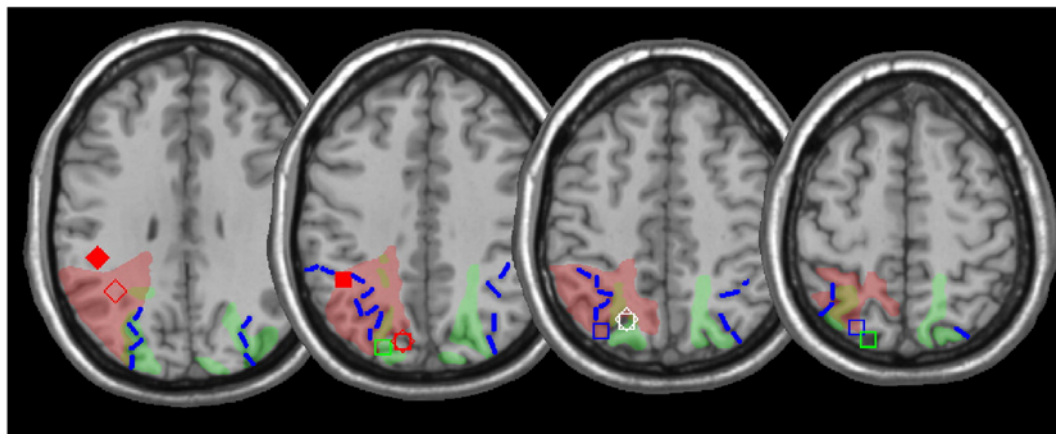


Fig. 1. Peak activations in the parietal region in fMRI studies of pointing and reaching. Red symbols: (Connolly et al., 2000), green symbols: (Schluppeck et al., 2005), blue symbols: (Serenio et al., 2001), white symbols: (Medendorp et al., 2003). Squares: activation by saccades, diamonds: activation by pointing. Filled symbols: foci lateral of the IPS, open symbols: foci medial of the IPS. Images were generated using MRIcro (Rorden and Brett, 2000). Activation sites according to Talairach coordinates as reported in studies listed in Table 1. Green regions: lesion of patient AP. Red regions: lesions in patients with optic ataxia as reported by Karnath and Perenin, 2005. The z coordinates of the slices are 32, 40, 48 and 56, respectively.

may be inaccurate and misleading with techniques which do not use head-restrained systems or additionally record head movements. Thus, conclusions drawn from previous reports with EOG recordings (Khan et al., 2005a; Perenin and Vighetto, 1988) may not suffice to answer this question appropriately. We therefore tested the precision of saccadic eye movements with the magnetic search coil technique and pointing movements to the same targets.

In our paradigm the target initially appears peripherally. Unfortunately, due to simultanagnosia, our patient was not able to point to the peripheral target while keeping the eyes on the central fixation point. So we had to let her perform a saccade towards the target. Thus the target became foveal after a saccade had been made towards it. As reaching errors in optic ataxia have been shown to depend on remapped, not viewed target locations (Khan et al., 2005b), the foveal target position might be more relevant for the pointing error than the initial peripheral position. The fovea (as contrasted to the peripheral visual field) has a bilateral representation in the posterior parietal cortex (Khan et al., 2005b; Merriam et al., 2003). This implies that in unilateral lesions the deficit is well compensated with central fixation of the target. In bilateral lesions like in our patient, we expected gross pointing errors with respect to left and right targets, so the foveal target position should not be critical.

Methods

Patient characteristic

The right handed patient AP suffered from Takayasu's arteritis at the age of 39. At that time she was pregnant in the 26th week of gestation and developed signs of an aortic insufficiency due to an aortic aneurysm including the aortic valve. Two weeks after a cesarean section in the 31st week of gestation an aortic root repair was performed. In the resection specimen the diagnosis of Takayasu's arteritis was proven. Postoperatively the weaning was difficult. When the patient recovered, she was left with a visual field defect for the lower hemifield. A visual field 12 weeks after the lesion showed a field defect for the lower right quadrant (10°

and 20° for the right and left eye, respectively). The horizontal meridian and 20° below were normal.

Initially she showed Balint's syndrome including oculomotor apraxia that later disappeared. In addition, she continued to show left–right disorientation, stereoagnosia, acalculia and severe optic ataxia. Pointing to own body parts was normal on clinical testing but was not formally measured. As a test for simultanagnosia we presented either one or two red spots on a computer screen. In this task the patient missed 3 of 20 simultaneous presentations as a sign of mild simultanagnosia at the time of recording. She also was spatially disoriented even in familiar places such as her own apartment. Recordings of hand pointing and eye movements could not be obtained earlier than 35 weeks after the lesion when the patient had completed rehabilitation and a first course of immunosuppressive therapy.

Stimuli

The subjects (patient AP and three controls) were seated in a wooden chair with their head fixed in a chin rest in a completely darkened room and 41 cm from a screen with targets for hand and eye movements. Targets and fixation point (red diodes) were at the height of the subjects' eyes.

The targets were presented in a pseudorandom sequence 8° and 12° left or right to the central fixation point, respectively. At trial begin the subjects looked at the fixation point straight ahead and rested with their right hand on a table in front of the target screen. To ensure the same starting position the hand rested in a mould which was easy to find without visual guidance.

Two tasks were studied: the “foveal” and the “peripheral/delay” task (Fig. 2).

In the “foveal” task, the subjects were instructed to simultaneously point to the target and to make a saccade towards it, as soon as the fixation point was shut off and the target was shut on. The label “foveal” in this case refers to the fact that at the beginning of hand pointing the targeting saccade has already shifted the peripheral target's image onto the “fovea”. 40 trials were presented. In the “peripheral/delayed” pointing task the fixation point was visible throughout the whole trial. The target

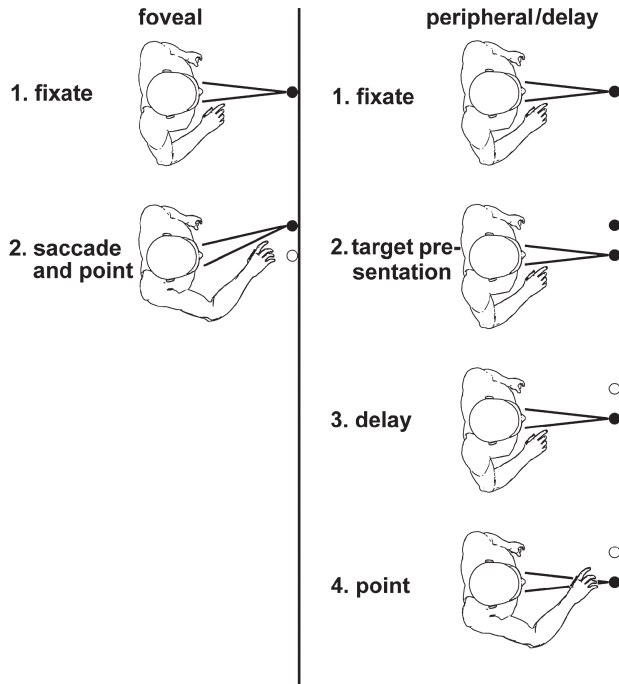


Fig. 2. Illustration of the foveal task (left) and the peripheral/delay task (right). The filled circles symbolize diodes shut on, open circles symbolize diodes shut off.

was visible for 2 s while the subjects kept fixating centrally. Then the target was shut off. In 4 different sessions (each of them with 20 trials), the patient was supposed to point as the fixation point changed its colour with a delay of 0, 2, 5 and 10 s (session A to D) with respect to the shut off of the target, respectively, while holding eye fixation on the central fixation point. The four sessions were carried out in a sequence ABCDDCBA to exclude any influence of fatigue.

Trials in the foveal condition had to be excluded from further analysis if the patient did not complete the pointing movement while the target was on or in case of prolonged eyelid closure (blink artefact). In the peripheral/delay condition trials were excluded if the subjects made a saccade towards the target. The patient successfully completed 12 trials in the foveal task and between 23 and 36 trials in the peripheral/delayed task.

Recording techniques

Eye movements were recorded 35 to 65 weeks after the lesion with one of two systems: the magnetic search-coil technique or the infrared reflection oculography.

A head-mounted infrared reflection oculography device (IMT, Dresden) was used for the peripheral/delay paradigm because it does not cause any discomfort for the patient. However, with this technique head movements interfere with the measurement. This is avoided with search coil recordings which were performed in the “foveal” task.

For the search coil recording, the CNC system (size $2 \times 2 \times 2$ m; CNC Engineering, Seattle, WA, USA) was used with standard annulus search coils (Skalar, Delft, The Netherlands). Data were stored in binary format at a 500 Hz sampling rate on a PC. For an

in vivo calibration subjects performed 10° saccades to the left and to the right (Helmchen et al., 2003).

Hand movements were recorded with the ZEBRIS system (Zebris Medizintechnik GmbH, Isny, Germany) at a sampling rate of 200 Hz and filtered with a 20 Hz Gauss filter. The system determines the position [specified by three values $v=(x, y, z)$] of an ultrasound-emitting marker relative to an array of three receivers. The calibration and the following analysis of the hand data were done using a program written with the Matlab programming language (The Mathworks, Natick, MA, USA). To record pointing movements, a marker was taped to the tip of the index finger. To obtain positions relative to the target screen, data were recorded for the positions of eight markers with known positions in 3D space and coefficients were determined for a linear transformation of the measured data to the known 3D positions in order to minimize error. Details can be found elsewhere (Trillenberget al., 2006).

Analysis

Gains of the hand pointing movement were calculated as hand distance from midline divided by target distance from midline (both measured in cm). The gain of the eye movement was calculated likewise from the endpoint of the last saccade [eye eccentricity and target eccentricity measured in degrees ($^\circ$)] if correctives saccades were present. Our gains therefore reflect final gaze and pointing errors, respectively.

Since a few trials with large gains occurred, the data are right skewed. In this situation large outliers strongly influence the mean. We therefore used medians instead of means. To characterize the width of the distribution of the data we report median absolute differences from the median (Sachs, 1982).

Results

Lesion topography

Cortical lesions are shown 35 weeks after the onset of symptoms in axial MRI slices (Fig. 3). MR-tomography showed symmetrical bilateral wedge-shaped lesions that spared the occipital pole and lower occipital regions but included more rostral parts of the visual cortex (V2 and V3). In the more apical parts of the lesion near the IPS there was cortical hemorrhagic transformation (in terms of a T1-hyperintense cortical band) of the medial but not the lateral bank of the IPS. The fundus of the IPS was not affected. The lesions were most likely caused by posterior leukoencephalopathy.

Results of pointing experiments

We will present results of the pointing experiments for the “foveal” task first and will then describe delayed pointing.

Traces of hand and eye movements in one trial of the “foveal” task are shown in Fig. 4, the results are summarized in Fig. 5. The recording was carried out 65 weeks after the lesion. The hand pointing movement was hypermetric and showed a greater variability when compared with the saccadic eye movement (median gain hand: 1.26, median gain eye: 1.02; *t*-test: $p < 0.002$, *F*-Test for variability hand vs. eye: $p < 0.05$). As compared with the controls the hand pointing movement, but not the saccadic eye movement of AP was hypermetric (*t*-test: $p < 0.001$).

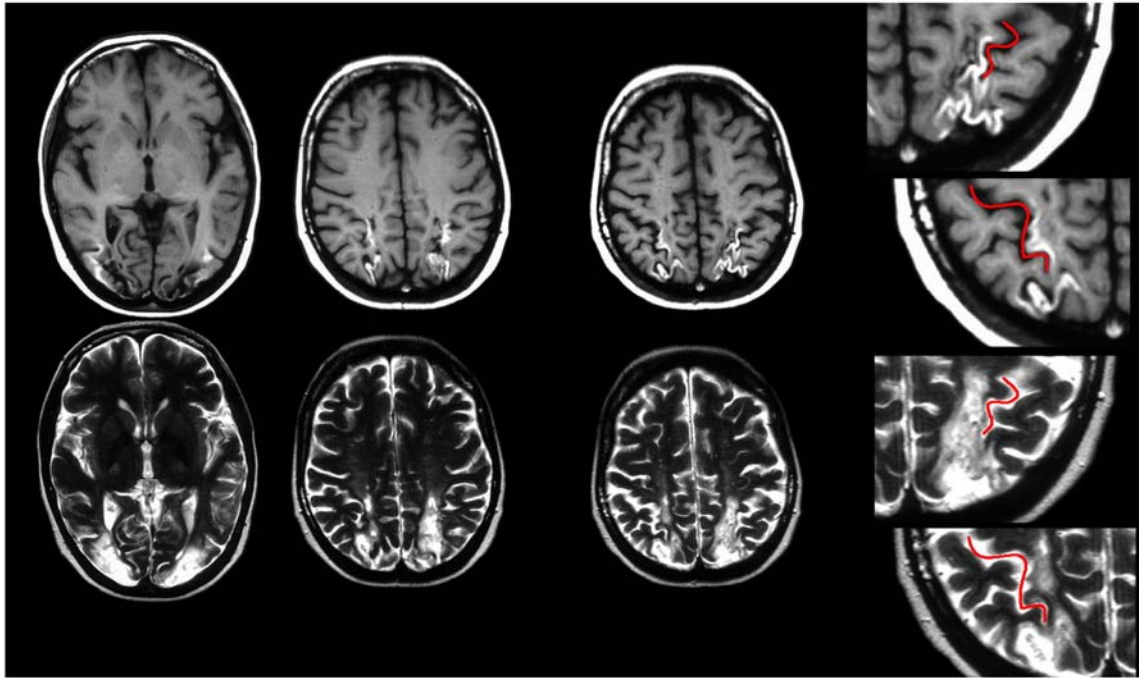


Fig. 3. MRI scan of patient AP 34 weeks after the lesion. T1-weighted images without contrast (upper panels) and T2-weighted images (lower panels). The intraparietal sulcus is indicated by a red line in the two inserted parieto-occipital scans.

With respect to hand pointing movements, recordings earlier after the lesion (35 to 54 weeks after the lesion) showed the same results in the foveal task (findings not reported in detail). At that time the patient was not able to complete the “peripheral/delay” task, probably because she had difficulty to see the peripheral target due to simultanagnosia.

The results of the “peripheral/delayed” task are shown in Fig. 6. Results for AP are shown with filled symbols, the results for age matched controls with open symbols. The middle panel shows the

influence of the delay on the gain of hand pointing. In AP pointing was hypermetric for any delay and either sides (gain larger than 1) and considerably larger than in the age-matched controls ($p < 0.02$, U -test). The gains differed significantly between left and right for immediate pointing, but not for pointing with delay (U -test: $p = 0.021$ for delay 0, n.s. for delay > 0). The gain for pointing to right targets did not change with a delay. However, for left targets there was an increase of hand pointing gains with increasing delay (t -test: $p < 0.02$ delay 0/delay 5, $p < 0.005$ delay 0/delay 10; U -test:

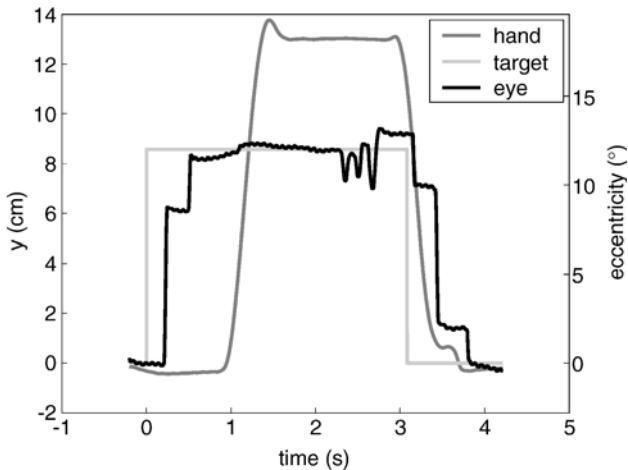


Fig. 4. A single trial of the “foveal” paradigm. The distance of the hand position from the midline is shown (y , in cm). Target and eye eccentricity are shown in deg ($^{\circ}$). Note that y is converted into angular eccentricity approximately by a scaling factor since the tangent is linear for small angles. Target: light grey line; eye: black line; hand: dark grey line.

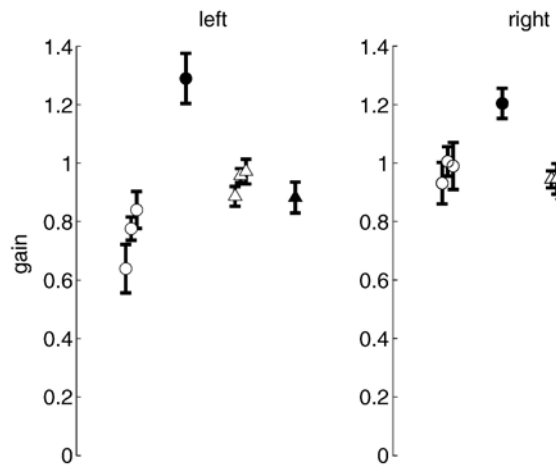


Fig. 5. Pointing hand movements and saccadic eye movements in “foveal” tasks for patient AP (filled symbols) and three controls (open symbols). Right panel: movements to the right; left panel: movements to the left. The hand pointing gain is shown with circles, saccadic gain is shown with triangles.

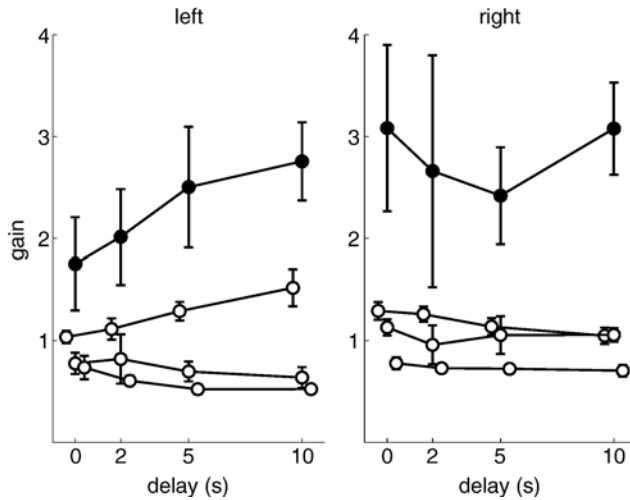


Fig. 6. Results of the “peripheral/delayed” task. Results for AP are shown with filled symbols, the results for the controls with open symbols. The left panel shows the results for pointing to the left, the right panel for pointing to the right. AP was always hypermetric in this task (for both sides and every delay). Variability decreased for the right targets with increasing delay.

$p < 0.02$ and $p < 0.01$). The variability of the pointing movement decreased with the delay for pointing to the right (cf. error bars in Fig. 6, *U*-test delay 2 vs. delay 10: $p < 0.05$).

Discussion

The major finding revealed profound hand reaching errors in the absence of severe saccadic gain deficits.

Eye movements and lesion sites

Our patient’s data confirmed the functional predictions obtained from areas MIP and LIP in the monkey.

With the head unrestrained, the metrics of eye movements are difficult to measure for technical reasons because subjects tend to move their head. For head-mounted devices, such as infrared reflection oculography and electrooculography (Heide et al., 1999) the signal for a given gaze direction is diminished by the amplitude of any head movement. Therefore head movements could simulate gaze errors while in fact gaze direction accurately matches target position. Our study is the first to use the magnetic search coil technique in optic ataxia that avoids this complication and shows that final eye position accurately matches target position.

The lesion in our patient involved the medial but largely spared the lateral wall of the IPS and thus seemed to spare the putative homologue of monkey area LIP. In another patient a very focal lesion medial to and at the fundus of the IPS has been reported (Roy et al., 2004). Eye movements were not recorded in this patient. In addition there only was a slight hand movement impairment in this patient: Only an absence of influence of manipulandum size on grasping kinematics but no misreaching could be demonstrated. The lesion site in our patient as well as that in the latter patient extend earlier findings in a larger group of optic ataxia patients with (in part large) lesions that overlapped in the intraparietal sulcus (Perenin and Vighetto, 1988). Both areas medial and lateral to the IPS were involved. In a more recent analysis the overlap of larger lesions of patients with optic ataxia

was compared to lesions in patients with other symptoms (Karnath and Perenin, 2005). As a candidate region for optic ataxia the junction between the inferior parietal lobule and the superior occipital cortex was suggested, thus a region lateral to the IPS.

In normal controls, functional imaging has also been used to localize the LIP homologue: activations during memory-guided saccades have been demonstrated in two regions medial of the IPS (Schluppeck et al., 2005). Their activation is mapped retinotopically to the respective regions, and the authors speculate that one of them could correspond to MIP and the other to LIP. Arm movements were not used in their protocol. In single-cell recordings in the monkey, however, there is a percentage of neurons that respond in relation to saccadic eye movements in area MIP, and in area LIP there is a percentage of neurons that respond in relation to hand movements. Thus, in a functional imaging study both areas could be activated. Overlapping activation by hand movements and saccades was also demonstrated by other authors (Medendorp et al., 2005; Medendorp et al., 2003).

In another study in controls both hand pointing and saccadic eye movements were mapped (Connolly et al., 2000). Their results were closer to our results with two foci of activation medial to the IPS that were activated during pointing only. Three foci were activated by both saccades and pointing, two of them lateral and one medial of the IPS.

Thus, our lesion data indicate that regions in the medial rather than in the lateral bank of the IPS are responsible for impaired visually guided reaching movements with preserved saccadic metrics.

Hand pointing

Hand pointing movements were characterized by (1) a marked asymmetric overshoot (hypermetria), (2) marked variability that increased to remembered (as opposed to visible) targets and decreased with delay, and (3) a marked directional difference for small delays in the delayed pointing paradigm.

Hand pointing dysmetria

The overshoot in our patient was pronounced. Much smaller overshoots in hand pointing may physiologically occur when pointing hand movements are carried out alone as opposed to a combination with a saccade (van Donkelaar and Staub, 2000). Second, pointing to remembered (as opposed to visible) targets is hypermetric in controls whether or not a combined saccade is executed (Henriques and Crawford, 2000; Henriques et al., 1998).

As a caveat one has to consider that our range of target eccentricities may be too small to fully explore metrics in delayed pointing. For example, hand pointing in patient AT in another study (Rossetti et al., 2005) was hypometric in immediate pointing to 10°, 20° and 30° targets; delayed pointing was hypermetric at 10° but continued to be hypometric at 20° and 30° (see Fig. 2 in Rossetti et al., 2005). Similar (much smaller) changes of gain with eccentricity have been demonstrated in controls (Prablanc et al., 1979a; Prablanc et al., 1979b). However a direct comparison of these results with our findings is precluded by different target heights which imply different mechanical constraints. Also, a systematic overestimation of target eccentricity in pointing was later demonstrated for a different paradigm in the same patient AT (Khan et al., 2005a), similar to our results.

Another condition that differs between our paradigm and earlier pointing experiments is the presence of ambient light. Studies in controls (as mentioned above) are usually performed in complete darkness. Patient studies, however, have been performed with ambient light due to technical reasons, e.g. due to the analysis of hand movements with films (Perenin and Vighetto, 1988), or the visual control of central fixation by the experimenter observing the patient (Rossetti et al., 2005). Since we wanted to compare our results with physiological studies we also recorded in complete darkness. In this situation, target positions cannot be related to additional stationary cues in the recording room that might help to locate the target. Also there is no visual control of the hand position while the hand approaches the target. This may cause considerable pointing errors even in controls and could explain why our patient exhibited drastic pointing errors even when she was allowed to fixate the target. Usually, pointing errors of patients with optic ataxia are small in foveal vision. Nevertheless, pointing deficits do occur with central vision with ambient light (Battaglia-Mayer and Caminiti, 2002).

Directional differences in pointing errors

In optic ataxia due to unilateral lesions pointing variability is larger for targets located in the hemifield contralateral to the lesion, or when carried out with the contralesional hand (Perenin and Vighetto, 1988; Revol et al., 2003). This has been called “field effect” and “hand effect”, respectively. Our patient had bilateral lesions. Clinically, however, our patient showed larger pointing dysmetria to the right (ipsilateral to the pointing hand). If one assumes that only the remapped target position after the saccade is relevant for the pointing errors (Khan et al., 2005b) the target will become a foveal target (in the “foveal” condition) irrespective whether it is presented at the left or right. If one further assumes that foveal targets are processed bilaterally in the parietal lobe (Khan et al., 2005b; Merriam et al., 2003) pointing errors should not differ for left and right targets in contrast to our observation. However, remapping of receptive fields to compensate for saccade-associated shifts of the retinal image was probably not intact in our patient, due to her large posterior parietal lesion, and then the field effect should be relevant also in the “foveal” condition. In addition, there was also a strong field effect for the right hemifield in the “peripheral” condition without delay, which disappeared at a delay of 5 or 10 s. This indicates that the field effect with grossly hypermetric pointing occurred only in case of rapid goal-directed responses, without delay, which are assumed to be mediated by the cortex around the IPS (“dorsal” projection system; Milner et al., 1999; Rossetti et al., 2005), whereas the slower “ventral” system (mediated by the inferior parietal lobule and the temporo-parietal junction) was affected less severely, without showing a field effect. Thus, the anatomical cause for the right visual field effect could be a larger size of the lesion in the left medial IPS, as compared to the right IPS, and indeed, this is the case with respect to the extent of T2 lesions in Fig. 3.

Clearly, pointing trajectories to left and right targets differ in geometry. In our patient direction differences in pointing errors occurred only for small delays but disappeared for larger delays in the delayed pointing paradigm. Since geometry and mechanics are not influenced by the delay, the asymmetry of errors reflect a field effect that occurred before remapping and is an argument against an exclusive determination of pointing errors by remapped position, or alternatively for impaired remapping in our patient.

Variability and metrics vs. delay

As shown in our patient, a delay between target presentation and the pointing hand movement has been demonstrated to improve pointing errors in patients with optic ataxia (Milner et al., 1999; Rossetti et al., 2005). This was explained with the temporal properties of the two “streams” for the processing of visual information. The dorsal stream that is affected in optic ataxia provides fast information for on line movement control. In contrast, the ventral stream provides slower analysis of visual information for cognition. A delay enables patients with optic ataxia to use their intact ventral stream for movement control.

With respect to pointing variability our patient does show an improvement with increasing delay thus following the pattern described earlier. However, no improvement can be demonstrated in our patient if absolute errors are studied. This is a consequence of systematic errors that occur with increasing delay: For targets to the right the systematic errors double increasing the delay from 0 to 10 s (gain 2 at delay 0, gain 3 at delay 10 s). This cannot be compensated by a reduction of variable errors (gain variability 1 at delay 0; 0.5 at delay 10 s), therefore the overall error does not decrease. Thus, the ventral stream does not seem to be particularly well suited for movement control (as one can see from the severe dysmetria that is even increased as the delay increases) but at least works with less variable results if there is severe damage to the dorsal stream.

In summary, we were able to show that saccadic eye movements in a patient with optic ataxia were normometric. Since the lesions in this patient were located medially to the IPS this is an argument that the putative homologue of monkey area LIP is localized more laterally, in contrast to imaging studies that demonstrate activation medially to the IPS. Pointing movements in the patient were markedly hypermetric even to foveal targets. Side differences in misreaching even towards a foveal target are an argument that the field effect in optic ataxia does not exclusively depend on the remapped but also on the original target position.

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