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Cortical Control of Visually Guided Reaching: Evidence from Patients with Optic Ataxia

Hans-Otto Karnath[1] and Marie-Thérèse Perenin[2]

[1]Section Neuropsychology, Department of Cognitive Neurology, Hertie-Institute for Clinical Brain Research, University of Tübingen, Tübingen, Germany and [2]INSERM U371 'Cerveau et Vision', IFR 19 Institut Fédératif des Neurosciences, Université Claude Bernard LyonI, Lyon, France

The dorsal stream of visual information processing connecting V1 to the parietal cortex is thought to provide a fast control of visually guided reaching. Important for this assumption was the observation that in both the monkey and the human, parietal lesions may provoke disturbance of visually goal-directed hand movements. In the human, severe misreaching termed 'optic ataxia' has been ascribed to lesions of the superior parietal lobule (SPL) and/or the intraparietal sulcus. Using new tools for lesion analysis, here we re-evaluated this view investigating the typical lesion location in a large group of unilateral stroke patients with optic ataxia, collected over a time period of 15 years. We found no evidence for the assumption that disruption of visually guided reaching in humans is associated with a lesion typically centering on the SPL on the convexity. In both left and right hemispheres, we found optic ataxia associated with a lesion overlap that affected the lateral cortical convexity at the occipito-parietal junction, i.e. the junction between the inferior parietal lobule (IPL) and superior occipital cortex and — in the left hemisphere even more posteriorly — also the junction between occipital cortex and the SPL. Via the underlying parietal white matter, the lesion overlap extended in both hemispheres to the medial cortical aspect, where it affected the precuneus close to the occipito-parietal junction. These lateral and medial structures seem to be integral to the fast control of visually guided reaching in humans.

Keywords: brain damage, human, optic ataxia, parietal lobe, reaching

Introduction

Ungerleider and Mishkin (1982) proposed two functionally specialized cortical streams of visual processing emanating from V1: a dorsal stream connecting V1 via prestriate areas to the parietal cortex and a ventral, occipito-temporal stream stretching through the visual areas of the temporal lobe. Milner and Goodale (1995) asserted that the dorsal stream was involved in the fast control mechanisms of visually guided eye and arm movements, while the ventral stream embodies the enduring characteristics of objects promoting conscious awareness of the visual world.

Empirical evidence arguing for this dual stream concept has been derived from neurophysiological work, functional imaging and lesion studies. An important milestone was the observation that stroke patients with parietal lesions may show a specific disorder of co-ordination and accuracy of visually goal-directed hand movements, not related to motor, sensory, visual acuity or visual field disorders. This disorder has been termed 'optic ataxia'. Typically such patients are impaired in reaching and grasping for visual objects with both hands in their contralesional visual field, while they have no problem in reaching when allowed to orient their eyes and head towards the object, i.e. under conditions of central vision.

In a series of cases with missile wound injuries, Ratcliff and Davies-Jones (1972) reported that such patients had skull lesions located in the superior part of the parietal region. In line with these early findings were CT scans of single cases with optic ataxia who showed lesions located in the superior parietal lobule (SPL) (Auerbach and Alexander, 1981; Ferro, 1984; Buxbaum and Coslett, 1998). Perenin and Vighetto (1988) investigated a series of 10 patients with optic ataxia after unilateral left- or right-sided lesions. The central 'core' of lesion overlap appeared symmetric in both hemispheres. It always included the intraparietal sulcus (IPS) and either the upper part of the inferior parietal lobule (IPL) or — more often — the medial or the ventral part of the SPL. Based on these early observations many subsequent studies have assumed the SPL (Jeannerod, 1988; Caminiti *et al.*, 1996; Rizzolatti *et al.*, 1997; Milner and Dijkerman, 1998; Wolpert *et al.*, 1998; Galletti *et al.*, 1999, 2003; Battaglia-Mayer and Caminiti, 2002; Milner *et al.*, 2003; Glover, 2003) and/or the IPS (Pierrot-Deseilligny *et al.*, 1986; Perenin and Vighetto, 1988; Milner and Goodale, 1995; Perenin, 1997; Milner and Dijkerman, 1998; Glover, 2003) as the neural correlate of human optic ataxia.

New tools are now available that provide a more precise lesion localization in humans (for a review, see Rorden and Karnath, 2004). These techniques reduce significantly the uncertainty brought in by the procedures used in previous anatomical studies on optic ataxia, where mainly skull landmarks were taken into consideration (Mazzochi and Vignolo, 1978), where only a rather small number of patients was available, and where no direct visual comparison between patients with and without optic ataxia patients via subtraction analysis (cf. Rorden and Karnath, 2004) was carried out. The present study thus readdressed the question of what the critical lesion site that typically disturbs the control of visually guided reaching in humans is. We investigated the typical lesion location in a large group of 16 unilateral stroke patients with optic ataxia, collected over a time period of 15 years, and compared them with 36 stroke patients without that disorder.

Subjects and Methods

Sixteen patients with optic ataxia following circumscribed unilateral left ($n = 10$) or right ($n = 6$) hemispheric brain lesions were investigated. Lesions resulted from stroke in all patients except three who underwent a surgical ablation of a benign tumor or abcess. Clinical evaluation of optic ataxia (see below) was performed 45.1 days on average after onset of stroke/surgery. The patients were compared with four different control groups. The first two (control groups A) consisted of 10 left and

six right brain-damaged stroke patients who did not show optic ataxia but who were comparable with respect to age and the frequency of minor additional impairments such as paresis, visual field defects, langage disorders, apraxia or spatial neglect. This ensures that the anatomical substrates provoking these latter deficits are equally represented in the subject groups with and without optic ataxia. Further, to ensure that the control patients did not suffer from optic ataxia even in the acute stage of stroke, clinical evaluation of these patients was performed early after onset (7.1 days on average). Table 1 gives an overview of the demographic and clinical parameters of all subjects.

The second two control groups (control groups B) consisted of 10 left and 10 right brain-damaged stroke patients who did not show optic ataxia but who were selected for having a lesion predominantly located in the parietal lobe. We selected these additional control subjects to characterize the critical area of parietal lesion overlap in patients with optic ataxia in more detail. Table 2 gives an overview of the demographic and clinical parameters of control subjects B.

Table 1
Demographic and clinical data of the patients with optic ataxia suffering from unilateral lesions of the left (L) or the right (R) hemisphere and of those control patients without optic ataxia (control groups A) who were comparable with respect to age and additional impairments such as paresis, visual field defects, langage disorders, apraxia or spatial neglect

	L optic ataxia (n = 10)	L controls A (n = 10)	R optic ataxia (n = 6)	R controls A (n = 6)
Sex	2 female, 8 male	2 female, 8 male	3 female, 3 male	3 female, 3 male
Mean age (years)	52.5 (11.7)[a]	65.2 (11.7)	64.2 (11.8)	64.8 (10.5)
Etiology	6 infarct, 3 hemorrhage, 1 surgery	6 infarct, 4 hemorrhage	2 infarct, 2 hemorrhage, 2 surgery	5 infarct, 1 hemorrhage
Mean time between lesion and clinical evaluation (days)	45.7 (55.7)	7.6 (10.0)	44.2 (55.8)	6.3 (2.3)
Paresis of contralesional side (% present)	10	20	17	50
Somatosensory deficit of contralesional side (touch) (% present)	20	10	33	67
Visual field defect (% present)	10	10	17	17
Spatial neglect (% present)	0	0	17	17
Aphasia (% present)	20	20	0	0
Apraxia (% present)	20	20	0	0

Surgery: 1 meningioma, 1 cavernoma, 1 abscess.
[a]Figures in parentheses are SD.

Table 2
Demographic and clinical data of those control patients without optic ataxia (control groups B) who were comparable to the optic ataxia patients in that their lesions likewise were located in the left (L) or the right (R) parietal lobe but who did not show optic ataxia

	L controls B (n = 10)	R controls B (n = 10)
Sex	5 female, 5 male	4 female, 6 male
Mean age (years)	67.2 (14.3)[a]	67.7 (9.1)
Etiology	8 infarct, 2 hemorrhage	7 infarct, 3 hemorrhage
Mean time between lesion and clinical evaluation (days)	11.9 (16.8)	11.7 (9.5)
Paresis of contralesional side (% present)	40	60
Somatosensory deficit of contralesional side (touch) (% present)	80	70
Visual field defect (% present)	0	20
Spatial neglect (% present)	0	30
Aphasia (% present)	40	0
Apraxia (% present)	10	0

[a]Figures in parentheses are SD.

Clinical Investigation

Patients with optic ataxia perform with large directional errors of the arm and a lack of anticipatory hand shaping, when grasping at objects in peripheral vision (Perenin and Vighetto, 1988). Apart from a few cases where only the hand and finger postural preparation failed (Binkofski *et al.*, 1999), optic ataxia patients are impaired in both the proximal and distal components of prehension (Perenin and Vighetto, 1988). The typical pattern of deficit in patients after unilateral brain lesions is gross misreaching in peripheral vision with spared reaching under foveal vision, mostly with the contralesional hand and in the contralesional space (Rondot *et al.*, 1977; Perenin and Vighetto, 1988). The majority of these ataxic reaches remain uncorrected. Exceptions occur, for example, when patients get tactile cues on object position while reaching, e.g. when the patient's hand hits the object by chance. In this case, they are able to correct the spatial errors. Sporadically, visually corrected errors are also observed.

In the present study, optic ataxia was assessed by video recordings in two reaching conditions. In the first condition, the patient had to fixate the camera lens in front of him and to grasp for an object (a big pencil) that was presented by the experimenter at various locations in the ipsilesional and then in the contralesional hemispace (Fig. 1*a*). First the hand ipsilateral and then the hand contralateral to the lesion was tested. In the second condition, instead of fixating the camera lens, the patient had to orient eyes and head towards the object while reaching for it (Fig. 1*b*). Ten reaches were recorded in each hand/space combination of the two test conditions (peripheral and foveal vision of the target).

The present patients with optic ataxia were selected when they showed the typical pattern, i.e. gross and uncorrected misreaching in peripheral vision with undisturbed reaching under foveal vision (illustrated in Fig. 1). All patients failed in both the proximal and distal components of prehension; Table 3 presents the frequency of those trials in which the patients missed the target and did not correct their errors. Typically, ataxic reaches were performed most frequently with the contralesional hand in contralesional space (Table 3). None of the patients showed any systematic bias of reaching, toward or away from the lesion side, in the present testing condition.

Visual fields were investigated by standardized neurological examination and/or Tübingen perimetry. Clinical testing for aphasia and apraxia was carried out using Hécean's test batteries (Hécean, 1968; Hécaen and Albert, 1978); spatial neglect was tested with the line cancellation test (Albert, 1973) and a copying task (house, bicycle).

Lesion Analysis

All 52 patients had circumscribed unilateral right or left hemisphere brain lesions due to stroke or surgery in three cases (caverenoma in two, abscess in one). Magnetic resonance imaging (MRI, including diffusion-, T1- and T2-weighted MRI) or computerized tomography (CT) was carried out in each subject, both with high resolution using a slice thickness between 1 and 4 mm for anatomical analysis. The MR scans were oriented along the bicommissural plane; the CT scans along the glabella-inion plane, which is virtually parallel to the later (Tokunaga *et al.* 1977). The mean time between

Figure 1. Reaching for a target in an exemplary patient with optic ataxia who was selected for the present study. The left brain-damaged patient showed gross and uncorrected misreaching for a target in peripheral vision (when he had to fixate the camera lens in front of him) (*a*) and normal reaching under foveal vision (when he had to orient eyes and head towards the object while reaching for it) (*b*). Typically, such ataxic reaches were performed most frequently with the contralesional hand in contralesional space.

Table 3
Mean percentage (and standard deviations) of uncorrected reaching errors in the 16 left or right brain-damaged optic ataxia patients

Peripheral vision				Foveal vision			
Contralesional hand		Ipsilesional hand		Contralesional hand		Ipsilesional hand	
CHS	IHS	CHS	IHS	CHS	IHS	CHS	IHS
64.6 (20.9)	27.5 (27.4)	21.1 (18.3)	2.5 (6.4)	5.7 (13.4)	2.1 (8.0)	1.4 (3.6)	0

The patients reached for objects presented in the contralesional vs. the ipsilesional hemispace (CHS/IHS), with the contralesional vs. the ipsilesional hand, with peripheral vs. foveal vision of the target. Ten reaches were recorded in each of the 8 test conditions.

stroke/surgery and imaging used for the anatomical analysis of lesion location was 60.7 months (SD 75.1). In 11 out of the 16 patients with optic ataxia, MRI images were available, seven in digital format. In these latter cases, the boundary of the lesion was delineated directly on the individual MRI image for every single transversal slice using MRIcro software (Rorden and Brett, 2000) (http://www.mricro.com). Both the scan and lesion shape were then mapped into stereotaxic space using the spatial normalisation algorithm provided by SPM2 (http://www.fil.ion.ucl.ac.uk/spm/). For determination of the transformation parameters, cost-function masking was employed (Brett *et al.*, 2001). In those cases where MRI data were not available in digital format or where CT had been performed, MRIcro software was used to map the lesion on transversal slices of the T1-template MRI from the Montreal Neurological Institute (www.bic.mni.mcgill.ca/cgi/icbm_view) that likewise is aligned with stereotaxic space and is distributed with MRIcro. The template scan provides various anatomical landmarks for precisely plotting size and localization of the lesion. Lesions were mapped onto the slices that correspond to Talairach *Z*-coordinates –24, –16, –8, 0, 8, 12, 16, 20, 24, 28, 32, 36, 40, 44, 48, 52, 56, 60, 64, 68, 72 and 76 mm by using the identical or the closest matching transversal slices of each individual. Automatic three-dimensional rendering of the lesion data was carried out using MRIcro (Rorden and Brett, 2000).

Results

In the group of 10 optic ataxia patients with left-sided lesions, we found (part of) the postcentral gyrus involved in 30%, the SPL in 60%, IPL in 70%, occipital gyri in 50%, superior and middle temporal gyri in 30%, the insula in 10%, the precuneus in 60%, the cuneus in 20%, parietal white matter in 90% and the temporo-occipital white matter in 50%. In the group of six optic ataxia patients with right-sided lesions, (part of) the postcentral gyrus was involved in 17%, the SPL in 50%, IPL in 100%, occipital gyri in 33%, superior and middle temporal gyri in 33%, the insula in 10%, the precuneus in 67%, parietal white matter in 83%, and the temporo-occipital white matter in 50%. Figure 2 illustrates lesion overlay plots for the two groups of optic ataxia patients. The number of overlapping lesions is illustrated by different colors coding increasing frequencies from violet (n = 1) to red (n = max. number of subjects in the respective group).

However, in order to identify the structures that are typically associated with optic ataxia, it is not sufficient to only divide cortex into discrete anatomical sections, calculate the frequency of their involvement in the patients' individual MR or CT lesions, and illustrate the overlap of the lesions (as above). It is also essential to contrast these sites directly with those of patients who also suffer from brain lesions in the same hemisphere but who do not exhibit optic ataxia. Without this comparison, observed regions of involvement and their overlap — as listed above and illustrated in Figure 2 — may reflect vulnerability of certain regions to injury (e.g. due to the vasculature of these regions) rather than any particular involvement with optic ataxia. A technique that provides such a comparison is the lesion subtraction method (reviewed in Rorden and Karnath, 2004). This enables the illustration of the center of overlap in patients with optic ataxia in direct visual contrast to those cortical areas that do not induce optic ataxia when lesioned. It was used for the following analyses.

To identify the cortical structures that are commonly damaged in patients with optic ataxia but are typically spared in patients with left or with right hemisphere lesions but no optic ataxia, a first analysis contrasted the optic ataxia patients with control groups A. These groups were comparable with respect to age and the frequency of additional impairments such as paresis, visual field defects, langage disorders, apraxia or spatial neglect. This ensures that the anatomical substrates provoking these latter deficits are represented equally in the group contrasts. We subtracted the superimposed lesions of control groups A (Fig. 3*a*) from the overlap images of the optic ataxia groups (Fig. 2) revealing percentage overlay plots. Figure 4 illustrates these results and highlights the anatomical structures that were affected more frequently in the patient groups with optic ataxia than in controls A. In both hemispheres, the lesion overlap laterally centered on the IPL, and in the left hemisphere also included the posterior occipito-parietal junction, i.e. the junction between superior occipital gyrus and

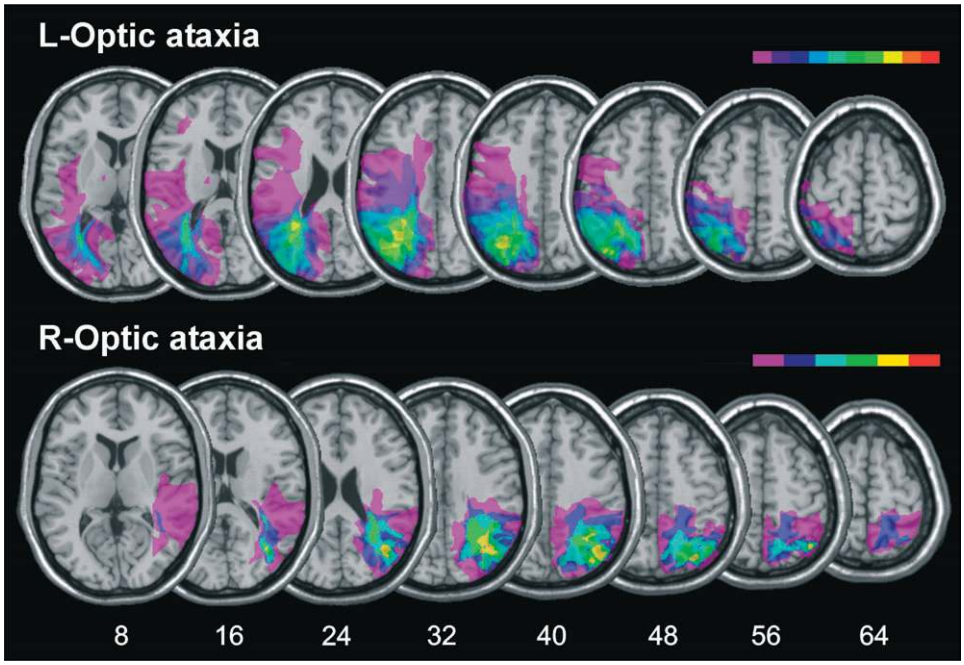

Figure 2. Overlay lesion plots of the patients with optic ataxia [$n = 10$ with left-sided lesions (L), $n = 6$ with right-sided lesions (R)]. The number of overlapping lesions is illustrated by different colors coding increasing frequencies from violet ($n = 1$) to red (n = max. number of subjects in the respective group). Talairach *z*-coordinates (Talairach and Tournoux, 1988) of the transverse sections are given.

the SPL (Fig. 4). Via the underlying parietal white matter, the lesion overlap extended medially to the left and the right precuneus, close to the occipito-parietal junction (Fig. 4).

Further, to determine the critical area associated with optic ataxia within the parietal lobe in more detail, we performed a second subtraction analysis. Two additional control groups were selected (control groups B), consisting of 10 left and 10 right brain-damaged patients who had stroke lesions predominantly located in the parietal lobe (like the patients with optic ataxia) but who did not show optic ataxia. Figure 3*b* shows the lesion overlay plots for these two additional control groups. To identify the parietal structures that are commonly damaged in patients with optic ataxia but are typically spared in those patients with parietal lesions but no optic ataxia, we subtracted the superimposed lesions of control groups B from the overlap images of the optic ataxia groups (Fig. 2), revealing percentage overlay plots (Fig. 5). In the right hemisphere, the lesion overlap centered laterally on the IPL and extended (via the underlying parietal white matter) to the precuneus on the medial aspect of the hemisphere close to the occipito-parietal junction (Fig. 5). In the left hemisphere, we found the same medial location of the center of lesion overlap on the precuneus. On the lateral convexity of the left hemisphere, the lesion overlap included the posterior occipito-parietal junction, i.e. the junction between superior occipital gyrus and the SPL, and extended into the IPL (Fig. 5).

Discussion

Our analysis of 52 unilateral stroke patients with and without optic ataxia revealed no evidence for the assumption that optic ataxia in humans is associated with a lesion typically centering on the SPL[1] (Jeannerod, 1988; Caminiti *et al.*, 1996; Rizzolatti *et al.*, 1997; Milner and Dijkerman, 1998; Wolpert *et al.*, 1998; Galletti *et al.*, 1999, 2003; Battaglia-Mayer and Caminiti, 2002; Milner *et al.*, 2003; Glover, 2003). In both hemispheres, we found the center of lesion overlap affecting the lateral cortical convexity at the occipito-parietal junction, i.e. the junction between the IPL and the superior occipital cortex and — in the left hemisphere even more posteriorly — also the junction between the occipital cortex and the SPL. Via the underlying parietal white matter, the lesion overlap extended to the medial cortical aspect, where it affected the precuneus close to the occipito-parietal junction.

Functional imaging in healthy human subjects has shown various parietal regions activated in goal-directed movements including both parietal lobules, the IPS and the precuneus, as well as other cortical regions (e.g. Desmurget *et al.*, 2001; Culham *et al.*, 2003). The large variability in the topography of activations may be partly due to the various tasks requirements. However, when considering the peak of activation in the parieto-occipital region, two main foci can be distinguished. In the early PET studies (Kawashima *et al.*, 1995; Grafton *et al.*, 1996; Inoue *et al.*, 1998; Desmurget *et al.*, 2001), natural reaching movements were performed with the eyes free to move at the visual target. Among varying foci in these studies a constant observation was activation in the IPS. In the more recent fMRI experiments (DeSouza *et al.*, 2000; Connolly *et al.*, 2000, 2003; Simon *et al.*, 2002; Medendorp *et al.*, 2003; Astafiev *et al.*, 2003), pointing movements were usually carried out in peripheral vision and after a delay of several seconds. In addition to the former, a second focus of activation was observed by most authors in a more dorsal and medial region located in the precuneus, just in front of the parieto-occipital sulcus, which could represent the homologue of the so-called 'parietal reach region' of the monkey (Connolly *et al.*, 2003). Although most studies have focused on the preparation phase of reaching movements, when the whole time-course of activation was considered, it was observed that the level of activation was even higher during movement execution than preparation (Astafiev *et al.*, 2003).

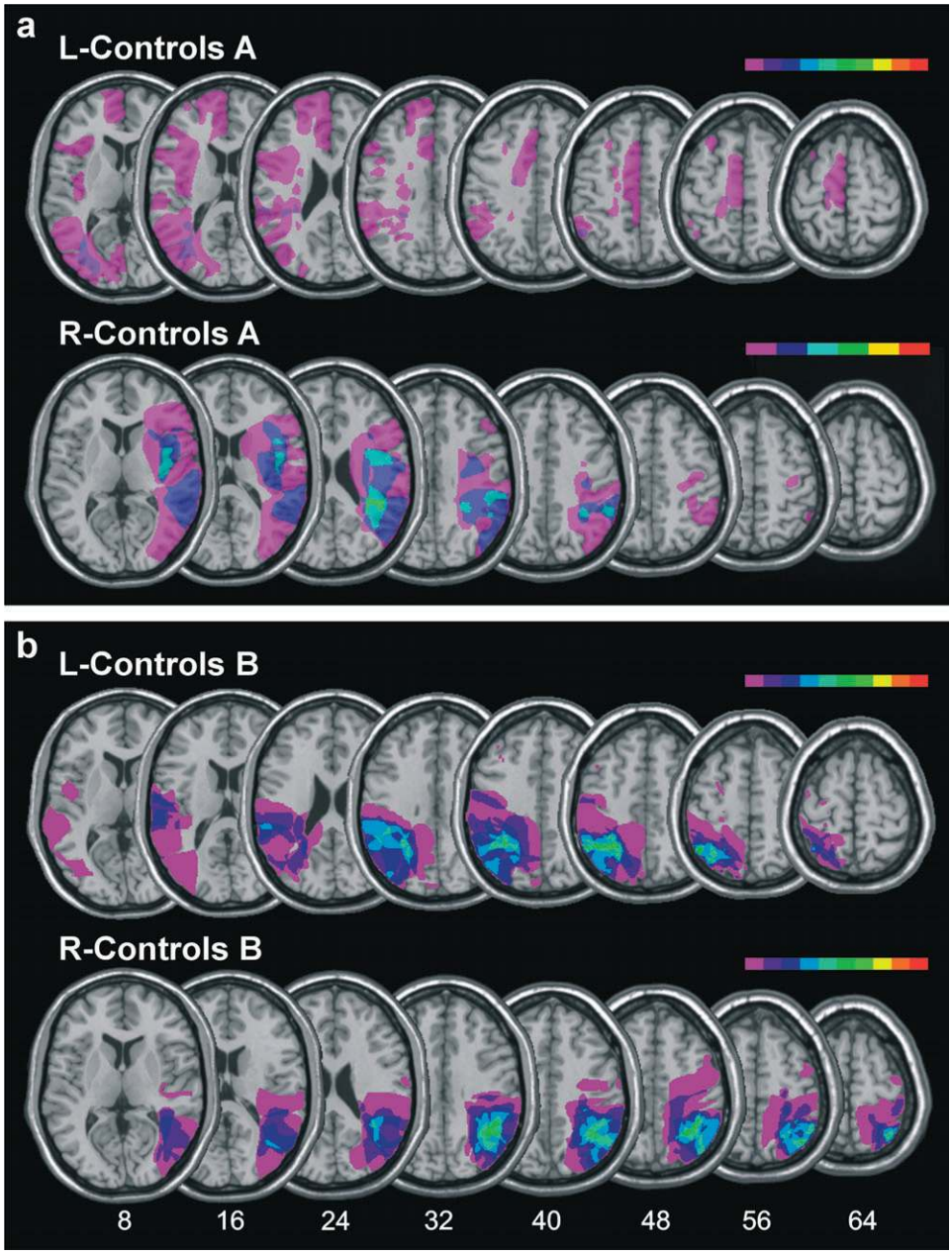

Figure 3. (*a*) Overlay lesion plots of control patients A without optic ataxia [$n = 10$ with left-sided lesions (L), $n = 6$ with right-sided lesions (R)] who were comparable to the patients with optic ataxia with respect to demographic variables and additional neurological and neuropsychological impairments. (*b*) Overlay lesion plots of control patients B without optic ataxia [$n = 10$ with left-sided lesions (L), $n = 10$ with right-sided lesions (R)] who had brain lesions predominantly located in the parietal lobe. The number of overlapping lesions is illustrated by different colors coding increasing frequencies from violet ($n = 1$) to red (n = max. number of subjects in the respective group). Talairach *z*-coordinates (Talairach and Tournoux, 1988) of the transverse sections are given.

Further, several recent studies carried out in monkeys have reported that areas MIP in the medial wall of the intraparietal sulcus and V6A at the junction between medial occipital and precuneate regions take part in processes related to limb action. It was observed that neurons in the 'parietal reach region', which overlaps areas MIP and V6A, responded preferentially before monkey reaches in peripheral vision (Andersen and Buneo, 2002). Moreover, Galletti *et al.* (1997) and Fattori *et al.* (2001) have shown that neurons located medial to MIP, in area V6A at the junction between the medial occipital and parietal cortex, are modulated during arm reaching movements. Further studies investigating neuronal activity in areas 5 (PE or PEc), MIP and V6A have hypothesized a role in coding arm movement direction, and in the transformation of sensory input into reference frames that can be used to guide limb action (Kalaska *et al.*, 1983; Ferraina and Bianchi, 1994; Lacquaniti *et al.*, 1995; Batista *et al.*, 1999; Buneo *et al.*, 2002; Galletti *et al.*, 2003).

The present results suggest that these regions are also involved in the control of visually guided reaching in humans. Compatible with the view from both electrophysiology in the monkey and fMRI in normal humans, namely that a homologous 'parietal reach region' is involved in planning (Andersen and Buneo, 2002; Connolly *et al.*, 2003) and in executing reaching

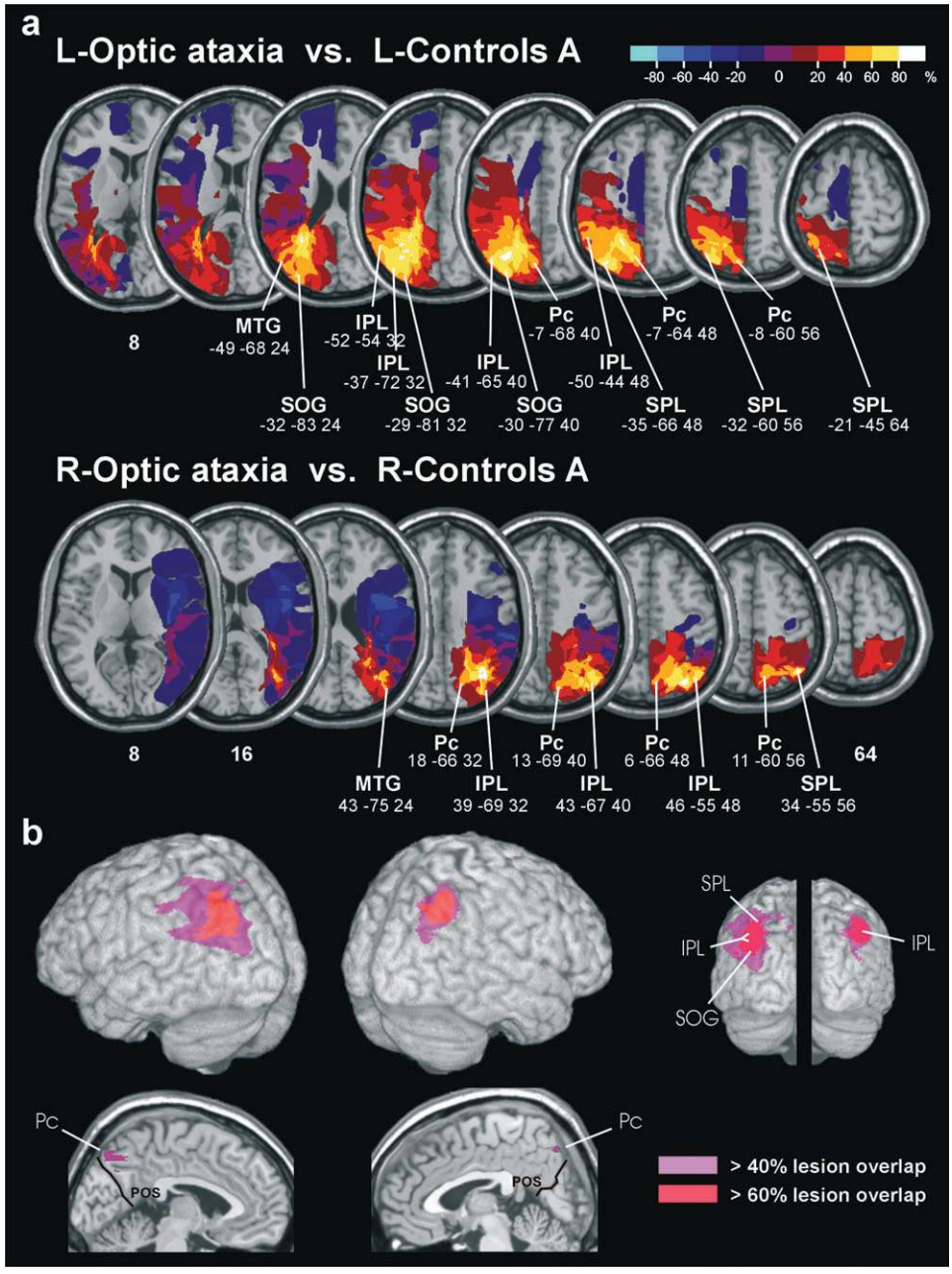

Figure 4. (*a*) Overlay plots of the subtracted superimposed lesions of the left (L) and of the right (R) brain-damaged optic ataxia groups minus control groups A. The percentage of overlapping lesions of the optic apraxia groups after subtraction of the controls A is illustrated by five different colors coding increasing frequencies from dark red (difference = 1–20%) to white (difference = 81–100%). Each color represents 20% increments. The different colors from dark blue (difference = −1 to −20%) to light blue (difference = −81 to −100%) indicate regions damaged more frequently in control groups A than the optica ataxia groups. Purple (middle of the color bar) designates regions where there is an identical percentage of lesions in both groups (= 0%). Talairach coordinates (Talairach and Tournoux, 1988) of the locations marked are given. MTG, middle temporal gyrus; SOG, superior occipital gyrus; IPL, inferior parietal lobule; SPL, superior parietal lobule; Pc, precuneus. (*b*) Lateral and medial surface views of the center of lesion overlap. The parieto-occipital sulcus (POS) is marked by a black contour.

movements (Astafiev *et al.*, 2003), we found optic ataxia typically associated with a lesion overlap at the hemispheres' medial cortical aspect centering on the precuneus, close to the occipito-parietal junction.

For a greater understanding of the role of areas 5, MIP and V6A in the monkey, it would be interesting to study the consequences of temporary or permanent inactivation. A complete lesion or inactivation of the 'parietal reach region' in the monkey may produce a deficit very similar to optic ataxia in the human. Unfortunately, so far nothing is known about the effects of such extensive damage of this area. However, few studies selectively inactivated areas 5, MIP and/or V6A. The data obtained so far seem to suggest that inactivation of these areas do not provoke severe disruption of visually guided reaching. Cooling of monkey SPL (lateral area 5) failed to provoke misreaching in the natural free gaze condition, but rather gave rise to a restricted disturbance of object manipulation and tactile discrimination with the contralateral hand (Stein, 1978).

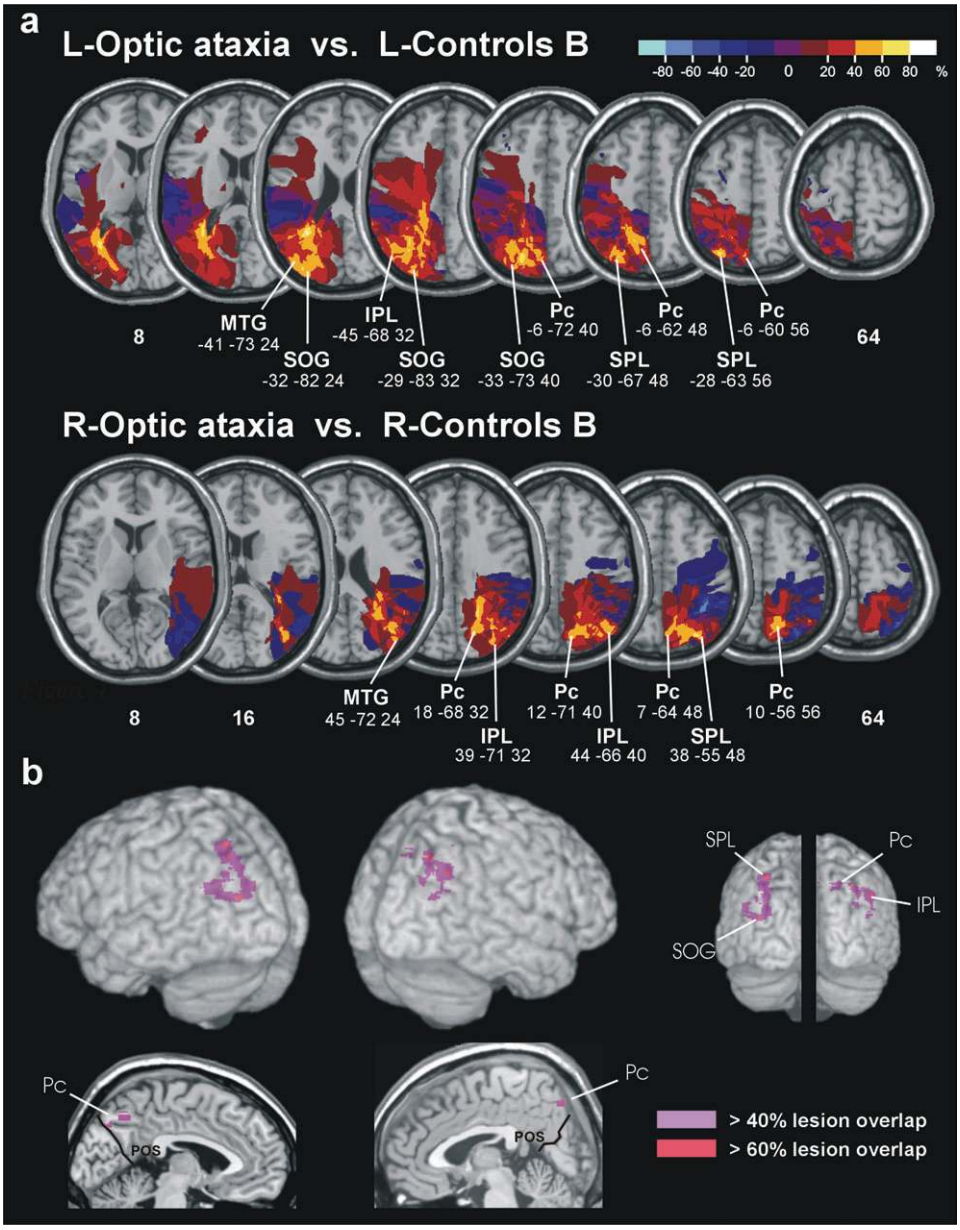

Figure 5. (*a*) Overlay plots of the subtracted superimposed lesions of the left (L) and of the right (R) brain-damaged optic ataxia groups minus control groups B. The percentage of overlapping lesions after subtraction and all other labeling is illustrated as described in Figure 4. Talairach coordinates (Talairach and Tournoux, 1988) of the locations marked are given. (*b*) Lateral and medial surface views of the center of lesion overlap.

Instead, misreaching was observed following cooling of the IPL (lateral area 7) (Stein, 1978). In line with these findings is that lesion of area V6A provoked abnormal wrist and hand rotation (leading to severe disturbance of grasping targets), while misreaching of target positions in peripersonal space was only minimal (Battaglini *et al.*, 2002). Moreover, Rushworth *et al.* (1997a,b, 1998) found that although cells in areas 5, MIP and 7b had spatially tuned activity during movements, lesions in these areas did not disrupt visually guided reaching. They found the relation between hand position and limb postural configuration disturbed (Rushworth *et al.*, 1998). Visual misreaching rather was observed with lesions in the monkey posterior IPL (BA 7a/7ab) and lateral IPS (Rushworth *et al.*, 1997a). This fits with early cell recording studies showing reach-related activities in the IPL, some of them specific for the contralateral arm and/or a particular direction of space (e.g. Mountcastle *et al.*, 1975; Leinonen *et al.*, 1979; see also MacKay, 1992).

Further studies have observed misreaching in monkeys occurring with lesions of the posterior IPL. In correspondence with our present findings that revealed the center of lesion overlap on the lateral cortical convexity typically at the occipito-parietal junction, they suggest that the posterior IPL is also involved in processes controlling visually guided reaching. Unlike in humans, the defect associated with such lesion occured when the monkeys were free to look at the target, i.e. under natural conditions of central vision. For a few days the animals lose the ability to correctly reach for targets located in peripersonal space, independent of primary visual, motor or sensory disorders (Faugier-Grimaud *et al.*, 1978, 1985; Stein 1978; Deuel and Farrar, 1993; Gallese *et al.*, 1994; Watson *et al.*,

1994; Rushworth *et al.*, 1997a). Longer-lasting misreaching has been observed following lesions of both Brodmann areas 5 and 7, encompassing the IPS (Lamotte and Acuña, 1978).

In conclusion, our results raise the question what is the function of the largest fraction of human superior parietal lobule if it is not the decisive area for visually guided reaching (in the sense of disrupting this function if lesioned). Functional imaging in healthy human subjects have shown that the SPL is activated in tactile object exploration (Seitz *et al.*, 1991; Binkofski *et al.*, 1999). SPL activation has further been reported for visuomotor tracking (Grafton *et al.*, 1992), motor imagery of rotatory hand movements (Wolbers *et al.*, 2003), changes in visual awareness (probably related to attentional switching mechanisms) (Rees *et al.*, 2002) and body part localization processing (Felician *et al.*, 2004). In addition, the SPL may also play a significant role in tactile recognition of objects. Lesions involving the SPL in stroke patients can evoke 'tactile agnosia', i.e. an inability to recognize everyday objects by tactile exploration (Binkofski *et al.*, 2001; Bohlhalter *et al.*, 2002). Moreover, repetitive transcranial magnetic stimulation over SPL has been observed to lead to impaired evaluation of the temporal congruency of pheripheral/central signals associated with self-generated movements (MacDonald and Paus, 2003).

Notes

[1] In terms of human macroanatomy [e.g. Nieuwenhuys *et al.*, 1988; Tzourio-Mazoyer *et al.* 2002], SPL and precuneus designate respectively the lateral and medial aspects of the upper parietal cortex. In part in conflict with this nomenclature, many neurophysiological studies encompass with the term "superior parietal lobule (SPL)" both the lateral and medial sides of same or similar cytoarchitectonic areas in front or at the parieto-occipital junction (i.e. area 5 or PE, PEm and Pec, the caudalmost part of area 19 and area PO/V6 and V6A).

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Address correspondence to Professor Hans-Otto Karnath, Center of Neurology, University of Tübingen, Hoppe-Seyler-Str. 3, D-72076 Tübingen, Germany. Email: Karnath@uni-tuebingen.de.

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