



Research report

Revisiting the cortical system for peripheral reaching at the parieto-occipital junction

Jason A. Martin, Hans-Otto Karnath and Marc Himmelbach*

Division of Neuropsychology, Center of Neurology, Hertie-Institute for Clinical Brain Research, Eberhard Karls University, Tübingen, Germany

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ABSTRACT

Optic ataxia (OA) is a neurological disorder that is characterised by misreaching to targets in the visual periphery. The anatomy of OA thus provides important information for the neural representation of visually guided reaching in humans. In 2005 a lesion mapping analysis of OA localised the critical lesion site at the parieto-occipital junction (POJ) (Karnath & Perenin, 2005). This work was accompanied by the discovery of a peripheral reaching module at the POJ in an fMRI study (Prado et al., 2005). The ostensible overlap between the territory typically affected in patients with OA and the findings of Prado et al. (2005) had a tremendous influence on the search for a cortical peripheral reaching module. However, a close inspection of the functional Magnetic Resonance Imaging (fMRI) study revealed that a comparison between reaching towards visible targets in the peripheral visual field and reaching to visible targets in the central visual field – which is the key aspect in clinical examinations of OA – was not conducted. Moreover, whereas main effects of reaching overlapped with the OA lesion site, specific interaction effects did not overlap. We performed a direct comparison between reaching to visible peripheral targets and reaching to visible central targets to address the inconsistencies between the aforementioned studies. Our analysis shows that Prado et al.'s study cannot be taken as evidence for a delineated module for peripheral reaching. In contrast to Prado et al. we found a combined system of POJ, IPS and SPL areas – the posterior human 7A, mIPS, V6A and the posterior IPS – with increased signals during reaching to peripheral targets.

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1. Introduction

1.1. Optic ataxia (OA): a deficit of peripheral reaching

In 1909 Rudolph Bálint described a patient who showed large errors when searching for an object with the right hand in

visual space; when asked to grasp an object with his right hand, the patient regularly missed it and would only find it when his hand touched the object; but he had no such difficulty with the left hand (Bálint, 1909; Balint & Harvey, 1995, p. 273; Harvey & Milner, 1995). Bálint called this particular visuomotor deficit 'Optische Ataxie' (optic ataxia), one of three

* Corresponding author. Division of Neuropsychology, Centre for Neurology, Hoppe-Seyler-Str. 3, 72076 Tübingen, Germany.

E-mail address: marc.himmelbach@uni-tuebingen.de (M. Himmelbach).

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components of the Bálint syndrome. The first report that OA could appear in distinct isolation from Bálint's syndrome was published by [Garcin, Rondot, and de Recondo \(1967\)](#). In the following years, only a few single cases that resembled Bálint's description of OA have been reported, typically showing unilateral or bilateral lesions that covered large parts of the posterior parietal cortex (for reviews please see [Battaglia-Mayer & Caminiti, 2002](#); [Jeannerod, 1986](#); [Pisella, Ota, Vighetto, & Rossetti, 2008](#); [Rossetti, Vighetto, & Pisella, 2003](#)).

[Perenin and Vighetto \(1988\)](#) and [Blangero et al. \(2010\)](#) presented two group studies of brain damaged patients with OA after unilateral lesions. Both reports converged on one behavioural observation: visuomotor performance in each single case of OA was worst in the contralesional field with the contralesional hand, which suggested a lateralisation of visuomotor control with respect to the visual field of target presentation as well as with respect to the hand being used. [Perenin and Vighetto \(1988\)](#) reconstructed the lesions drawn from CT scans in 8 of their 10 patients and found an overlap at the PPC, always including the intra-parietal sulcus and either the superior part of the inferior parietal lobule or more often extending into the superior parietal lobule. This pattern pretty much overlapped with the lesions sites later reported for the patients of [Blangero et al. \(2010\)](#). However, neither of these two lesion analyses included a brain damaged control group without OA. A direct comparison of a group of OA patients with a group of brain damaged patients without OA localised critical brain structures more precisely in the region of the parieto-occipital junction (POJ; [Karnath & Perenin, 2005](#)).

1.2. fMRI and OA lesions: [Prado et al. \(2005\)](#)

It was proclaimed that the lesion overlap reported in [Karnath and Perenin \(2005\)](#) showed a remarkable resemblance with parieto-occipital activation foci presented from an fMRI study that addressed visually guided reaching to peripheral targets ([Prado et al., 2005](#)). [Prado et al. \(2005\)](#) claimed to provide evidence for the existence of a dedicated cortical system for peripheral reaching and presented the POJ as its crucial node. However, there is some disagreement with later fMRI studies that failed to find clear cut evidence for gaze-centred target encoding or for a specificity for foveated versus extrafoveal targets at the POJ ([Beurze, Toni, Pisella, & Medendorp, 2010](#); [Filimon, Nelson, Huang, & Sereno, 2009](#)). Furthermore, the negative conclusion of [Prado et al. \(2005\)](#) that other regions like the medial intraparietal sulcus (MIP) play no specific role during peripheral reaching was contradicted by [Hwang, Hauschild, Wilke, and Andersen \(2012\)](#) who reported a mis-reaching in macaques, resembling that of OA in humans, induced by pharmacological inhibition of MIP.

A closer look at the study of [Prado et al. \(2005\)](#) helps to resolve these apparent contradictions. [Prado et al. \(2005\)](#) used an elaborated design primarily controlling for the execution of saccades and the visibility of the targets during the reaching movement. The participants saw a fixation cross at the centre of a screen. After the fixation cross was extinguished, a peripheral target appeared either 5° or 10° to either the right or to the left of the fixation position. Subjects performed three different reaching conditions: (1) reaching to a peripheral

target after a saccade to the target position, the target being visible throughout the whole trial (Visible Target/Saccade execution, VT/Se); (2) executing a saccade and reaching to a peripheral target, with the target becoming invisible 150 msec after target presentation onset (Invisible Target/Saccade execution, IT/Se); (3) reaching to a visible peripheral target without a saccade, the participants kept their gaze at the fixation cross during reaching (Visible Target/No Saccade execution, VT/NSe). These three reach conditions were matched by three control conditions in which participants did not reach; (4) executing a saccade to a visible peripheral target (Visible Target/Saccade control, VT/Sc); (5) executing a saccade to an invisible peripheral target (Invisible Target/Saccade control, IT/Sc); or (6) execute a covert shift of attention to a visible peripheral target (VT/NSc).

[Prado et al. \(2005\)](#) reported three different POJ peak signal locations for the left hemisphere based on three different contrasts between the abovementioned conditions ([Fig. 1](#)). One resulted from the contrast between reaching to a target in the peripheral visual field under fixation and the peripheral presentation of the target without a hand movement under central fixation [(MNI coordinates $x = -16, y = -78, z = 44$); VT/NSe – VT/NSc; [Fig. 1A](#)]. A second POJ peak in their report was associated with the execution of a hand and eye movement to a target in the peripheral visual field that vanished before movement execution contrasted with the execution of just an eye movement to the invisible target [(-18 -84 42); IT/Se – IT/Sc; [Fig. 1B](#)]. The third POJ location reported in [Prado et al. \(2005\)](#) resulted from an interaction analysis that, in the words of the authors, revealed the “effect of the peripheral position of the target during reaching” or the “effect of the retinal position of the target” (see [Prado et al., 2005](#) p. 852; their [Figure 3](#) and [Table 2](#), respectively). The contrast with visible peripheral targets during eye and/or hand movement execution was subtracted from the contrast with peripheral targets that became invisible before eye and/or hand movement execution [(IT/Se – IT/Sc) – (VT/Se – VT/Sc)] ([Fig. 1C](#)). In both contrasts of this interaction analysis a hand movement always accompanied an eye movement to the same spatial locations and thus no peripheral reaching under central fixation was included in this analysis. Despite their complex design the most straightforward comparison, namely that of reaching for a peripheral target under fixation versus reaching for a foveated target – which is the key aspect in pathological OA behaviour – was never conducted in this study. Instead, the authors reported POJ signal increases from two contrasts without a control of reaching per se: (1) VT/Se versus VT/Sc, (2) IT/Se versus IT/Sc ([Fig. 1A, B](#)). In both contrasts, the execution of an eye and hand movement was compared to an eye movement only. It is obvious that neither of these two contrasts accounted for signal changes induced by reaching per se. Nevertheless, the figures illustrating the associated signal increases became the most prominent parts of this report (please see [Prado et al., 2005](#), [Figure 2b, c](#) and [Figure 3b](#)), presumably because of their remarkable similarity with the previous lesion analysis of OA stroke patients ([Karnath & Perenin, 2005](#)).

Because of the lack of a control condition that could isolate the effects of peripheral reaching in the abovementioned two contrasts, the main conclusion of [Prado et al. \(2005\)](#) on a dedicated system for peripheral reaching must be based on

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