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# The effect of goals and vision on movements: A case study of optic ataxia and limb apraxia

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ABSTRACT

Normally we can perform a variety of goal-directed movements effortlessly. However, damage to the parietal cortex may dramatically reduce this ability, giving rise to optic ataxia and limb apraxia. Patients with optic ataxia show clear misreaches towards targets when presented in the peripheral visual field, whereas limb apraxia refers to the inability to use common tools or to imitate simple gestures. In the present paper we describe the case of a left-brain damaged patient, who presented both symptoms. We systematically investigated both spatial and temporal parameters of his movements, when asked to reach and grasp common objects to move (Experiment 1) or to use them (Experiment 2), presented either in the central or peripheral visual field. Different movement parameters changed in relation to the goal of the task (grasp to move vs. grasp to use), reflecting a normal modulation of the movement to accomplish tasks with different goals. On the other hand, grip aperture appeared to be more affected from both task goal and viewing condition, with a specific decrement observed when CF was asked to use objects presented peripherally. On the contrary, a neat effect of the viewing condition was observed in the spatial distribution of the end-points of the movements, and of the horizontal end point in particular, which were shifted towards the fixation point when reaching towards peripheral targets. We hypothesized that optic ataxia and limb apraxia have a differential effect on the patient's performance. The specific presence of optic ataxia would have an effect on the movement trajectory, but both symptoms might interact and influence the grasping component of the movement. As a 'cognitive side of motor control impairment', the presence of limb apraxia may have increased the task demands in grasping to use the objects thus exacerbating optic ataxia.



## 1. Introduction

In everyday life, we are used to perform series of fine and complex movements automatically and with high precision. Our abilities range from simple pointing movements, as to press the button of the elevator, to more complex actions involving the use of objects. After damage to parietal and/or premotor areas, these abilities can be compromised leading to two well-known neuropsychological symptoms: optic ataxia (OA) (Perenin & Vighetto, 1988) and limb apraxia (LA) (Kertesz & Ferro, 1984).

First described as one of the symptoms of Balint's syndrome (Balint, 1909), OA is often observed as a consequence of a bilateral lesion in the superior parietal lobe (Karnath & Perenin, 2005; Milner, Dijkerman, McIntosh, Rossetti, & Pisella, 2003; Pisella et al., 2000, 2004). However, it can also be observed following unilateral lesions of either the right or left hemisphere (Blangero et al., 2010; Karnath & Perenin, 2005; Perenin & Vighetto, 1988), or involving more inferior parts of the parietal lobe (Meek, Shelton, & Marotta, 2013; Perenin & Vighetto, 1988). First described as one of the symptoms of Balint's syndrome (Balint, 1909), OA is often observed as a consequence of a bilateral lesion in the superior parietal lobe (Karnath & Perenin, 2005; Milner et al., 2003; Pisella et al., 2000, 2004). However, it can also be observed following unilateral lesions of either the right or left hemisphere (Blangero et al., 2010; Karnath & Perenin, 2005; Perenin & Vighetto, 1988), or involving more inferior parts of the parietal lobe (Meek et al., 2013; Perenin & Vighetto, 1988). However, a closer look at single case studies (see Table 1), as well as at larger group studies

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**Table 1**
Summary of brain lesions reported in single case studies with OA patients.

| Patient | | Side | Lobes | Brodmann areas | Specified lesions |
|---|---|---|---|---|---|
| CAN | Blangero et al. (2007) | Left | Posterior-parietal | 7, 39, 40 | POJ |
| MH | Cavina-Pratesi et al. (2010, 2013), Rice et al. (2008) | Left | Posterior-parietal and frontal | | IPS, IPL |
| CF | Blangero et al. (2008), Khan et al. (2005, 2007), Striemer et al. (2009) | Bilateral | Parieto-occipital | 18, 19, 7, 5, 2 | |
| OK | Blangero et al. (2007), Khan et al. (2005) | Right | Posterior-parietal | 7, 39, 40, 5 | |
| FDL | Ferrari-Toniolo et al. (2014) | Right | Posterior-parietal | 5, 7, 31 | POJ, IPS, IPL, SPL, PC |
| JR | Khan, Pisella, Delporte, Rode, and Rossetti (2013) | Right | Posterior-parietal | | POJ, IPS, |
| MH | Kitadono and Humphreys (2007) | Bilateral | Posterior parietal | | POJ, IPS, SPL |
| IG | Granek, Pisella, Blangero, Rossetti, and Sergio (2012), Gaveau et al. (2008), Himmelbach et al. (2009), McIntosh et al. (2010), Milner et al. (2001, 2003), Pisella et al. (2000, 2004), Schindler et al. (2004) | Bilateral | Parietal | 18, 19, 7, 39 | |
| AT | Gaveau et al. (2008), Milner et al. (2003), Rossetti et al. (2003), Schindler et al. (2004) | Bilateral | Parietal | 18, 19, 7, 39 | |
| US | Himmelbach and Karnath (2005) | Left | Occipito-temporal, inferior frontal gyrus | | |
| GH | Himmelbach and Karnath (2005) | Left | Medial parietal cortex | | POJ, precuneus |
| JJ | Jackson et al. (2005, 2009) | Bilateral | Posterior parietal _AG | | IPS, SPL |
| KE | Jax et al. (2009) | Left | Posterior parietal | | |

POJ (parieto-occipital junction); IPS (intraparietal sulcus); PC (precuneus); SPL (superior parietal lobe); IPL (inferior parietal lobule).

(Blangero et al., 2010; Karnath & Perenin, 2005; Perenin & Vighetto, 1988) highlighted other possible neural underpinnings of OA, in particular the parieto-occipital junction (POJ), the intraparietal sulcus (IPS) and the precuneus. Note that Karnath and Perenin (2005) did not confirm the association between OA and damage to the superior parietal lobule; instead, he showed that in a sample of 18 patients with OA (10 left brain-damaged; 8 right brain-damaged), the superior parietal lobule was damaged only in about half of the sample (69% of left brain-damaged patients and 50% of right brain-damaged patients). On the contrary, lesions in the inferior parietal lobe were observed in all right brain-damaged patients and in a large portion of left brain-damaged (70%). In this study (Karnath & Perenin, 2005), a further subtraction analysis confirmed the association between these regions and OA, with the specific involvement of parietal occipital junction (POJ). This region, in addition to the intraparietal sulcus (IPS), has been shown to be specifically involved in reaching (Prado et al., 2005). In particular, fMRI evidence demonstrated that while IPS is recruited during reaching independently of the target position in the visual field, POJ is activated during movements towards peripherally presented targets (Prado et al., 2005), reinforcing the hypothesis that this region plays a critical role in the emergence of OA.

For instance, despite rare examples of OA involving central vision (i.e., *foveal optic ataxia*, Buxbaum & Coslett, 1997; Jeannerod, Decety, & Michel, 1994; Perenin & Vighetto, 1988), OA is characterized by evident misreaches towards peripherally presented targets (Rossetti, Pisella, & Vighetto, 2003), with spared basic perceptual and motor abilities (Balint, 1909; McIntosh, 2010). In some patients, OA can affect reaching movements towards targets presented in the controlesional visual field (the so-called *field effect*) and/or using the controlesional hand (the so-called *hand effect*) (Blangero et al., 2008; Khan et al., 2007; Rice et al., 2008; Striemer et al., 2009). This symptom is modality-specific and does not seem to emerge with auditory or tactile stimuli (Rossetti et al., 2003), suggesting that OA may derive from a specific deficit in coupling vision and action.

Although alterations of the grasping component have also been noted when grasping objects placed at different distances from the body and/or hemispaces of action (Cavina-Pratesi, Ietswaart, Humphreys, Lestou, & Milner, 2010; Jakobson, Archibald, Carey, & Goodale, 1991; Perenin & Vighetto, 1988) and in scaling the grip aperture according to the size of the object in OA (Cavina-Pratesi et al., 2010; Milner et al., 2001), most studies focused on the assessment of patients' reaching and transport phase of the movement. This literature reports a systematic deviation of both end points and movement trajectories in reaching for peripheral targets (Blangero et al., 2010; Dijkerman et al., 2006; Jackson, Newport, Mort, & Husain, 2005; Khan et al., 2005, 2007; Milner et al., 2003). Other alterations of the reaching component pertain the lack of movement modulation to avoid possible collisions with no-target stimuli (Schindler et al., 2004) or the automatic correction of the reaching movements in relation to rapid changes of the target location (Blangero et al., 2008; Pisella et al., 2000), a phenomenon commonly observed in normal adults and known as automatic pilot (McIntosh, Mulroue, & Brockmole, 2010). Interestingly, despite these deficits, the performance of patients with OA seems to improve when (i) the movement onset is delayed (~5 s) (Himmelbach & Karnath, 2005; Milner et al., 2001, 2003) or when (ii) the visuo-motor coordination demand is reduced like, for instance, when the target of the action is not physically present and patients are asked to pantomime the reaching and/or grasping action (Milner et al., 2003) or when on-line vision is removed (Jackson et al., 2005; Milner et al., 2003). In these conditions, participants rely on previous knowledge of the target location and proprioceptive feedbacks (see also Lingnau et al., 2014), rather than on online integration between vision of the target or of the hand and the actual movement. Taken together these observations led to one of the most acknowledged interpretations of OA as the impairment of online visuo-motor control (Rossetti et al., 2003). According to this view, the posterior parietal lobe is responsible for the conversion and integration of perception into action and for online motor control, and is involved in more automatic rather than voluntary corrections (Blangero et al., 2008; Pisella et al., 2000).

On the other hand, limb apraxia is a high-order impairment of goal-directed movements in which patients' difficulties cannot be ascribed to simple perceptual or motor deficits (Rumiati, Papeo, & Corradi-Dell'Acqua, 2010). Although it is commonly observed as a consequence of damage to parietal and premotor cortices (Haaland, Harrington, & Knight, 2000; Kertesz & Ferro, 1984), limb apraxia has also been associated with left-brain damage, affecting the frontal lobes (Haaland et al., 2000) or subcortical structures, such as basal ganglia or periventricular and internal capsule (Hanna-Pladdy, Heilman, & Foundas, 2001). Limb apraxia has most frequently been observed following stroke in the left hemisphere (Buxbaum, 2001; for a recent review, see Rumiati, Papeo, & Corradi-Dell'Acqua, 2010), but it is not limited to stroke. It can be observed in patients with different conditions including Alzheimer's and Parkinson's disease (Leiguarda et al., 1997; Wheaton & Hallett, 2007).