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# Optic ataxia errors depend on remapped, not viewed, target location

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**Optic ataxia is a disorder associated with posterior parietal lobe lesions, in which visually guided reaching errors typically occur for peripheral targets. It has been assumed that these errors are related to a faulty sensorimotor transformation of inputs from the 'ataxic visual field'. However, we show here that the errors observed in the contralesional field in optic ataxia depend on a dynamic gaze-centered internal representation of reach space.**

Optic ataxia is characterized by reaching errors that cannot be attributed to a solely visual or motor pertubation[1,2]. Almost all optic ataxia studies emphasize that central foveal vision is relatively unimpaired but that reaching errors increase with retinal eccentricity[1–9]. Indeed, the damaged posterior parietal cortex (PPC) is part of the dorsal visuomotor route fed by magnocellular inputs from the retinal periphery[3–6]. Classically, optic ataxia has been interpreted as a perturbation in the static transformation of information from the retinal image to the representation of contralateral visual space in the damaged PPC. In contrast, recent functional imaging and single-unit recording investigations have shown that the PPC contains a dynamic internal spatial representation in which each hemisphere represents contralateral space for reaching in gaze-centered coordinates[10–12]. According to this scheme, the location of the remembered target has to be recomputed or updated during each eye movement. Along this line, optic ataxia could instead result from a degradation of a dynamic representation of reaching space that is independent of the original retinal stimulus. Previous studies did not test these alternatives because the original retinal location and the internal representation of the target were not dissociated. Here, we achieved this dissociation by asking

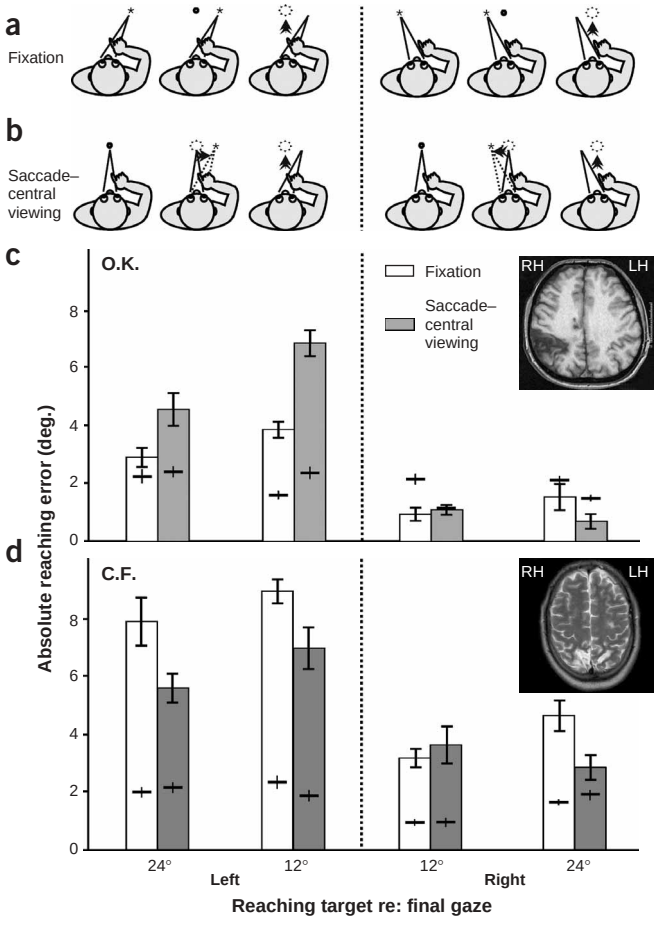


**Figure 1** Fixation task and saccade–central viewing task. (**a**) Fixation task. Subjects foveated on a fixation light (*), while the target was illuminated (small circle). After both lights were extinguished, subjects reached toward the target location while fixating at the fixation position location. Left, reaching target in the left visual field. Right, reaching target in the right visual field. (**b**) Saccade–central viewing task. Subjects first fixated on the target (small circle), then saccaded to a fixation light (*). After the fixation position was extinguished, they reached toward the target location while maintaining gaze at the fixation position location. (**c**) Absolute reaching errors from the target for the fixation (white bars) and the saccade–central viewing tasks (gray bars) for the four fixation positions (24° and 12° left and 12° and 24° right) shown for subject O.K. The *x*-axis shows target position relative to gaze; left and right correspond with the target being in the left or right visual field. The small crosses are mean errors (horizontal bar) for five controls with s.e.m. (vertical bar). Means and s.e.m. are shown for each final fixation across approximately 12 trials. The T1 magnetic resonance imaging (MRI) scan shows damage to the right posterior parietal lobe (darker area). (**d**) Absolute reaching errors for subject C.F., depicted in the same manner as in **c**. The T2 MRI scan shows asymmetrical bilateral damage to the posterior parietal lobe (white areas at bottom). The small crosses are mean and s.e.m. for three controls that performed the tasks in the same time period as the subject.

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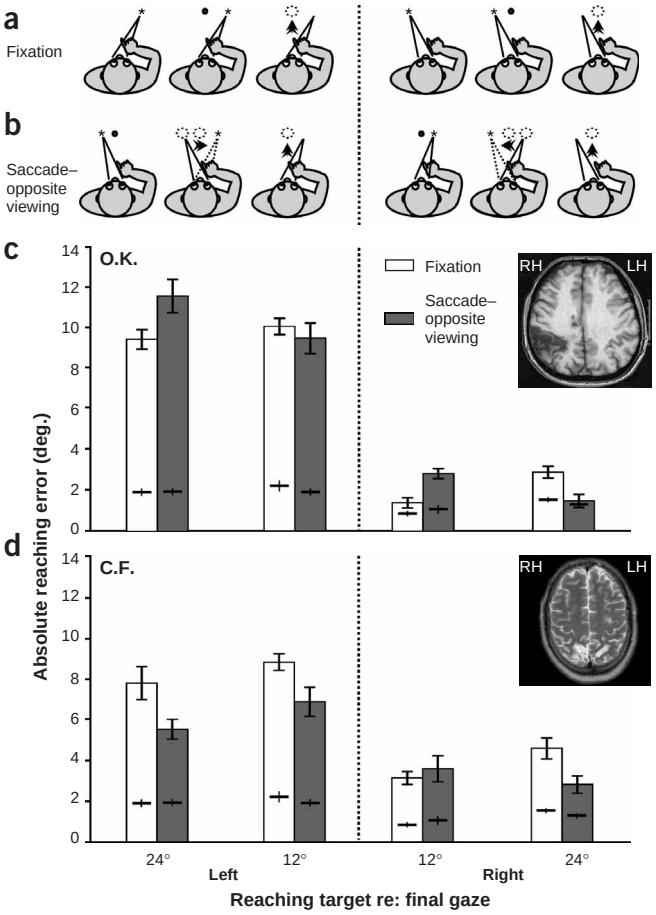


**Figure 2** Saccade–opposite field viewing task. (**a**) Fixation task repeated for comparison purposes. (**b**) Saccade–opposite field viewing task. Subjects first fixated on a fixation light (*) while the central target light was illuminated (small circle). After both lights were extinguished, subjects made a saccade to a second fixation position (*) in the opposite visual field (left panel, second fixation toward the right, target in LVF relative to final gaze; right panel, second fixation toward the left, target in RVF). After this light was also extinguished, subjects reached toward the target location while holding fixation at the final fixation position. (**c**) Absolute reaching errors for subject O.K. for the fixation task (white bars) and the saccade–opposite field viewing task (gray bars). Average errors are plotted as a function of target location relative to final gaze; for example, the average error shown for 24° right means that subjects first viewed the target in their LVF and then made a saccade, and when they reached to it, the target was in their RVF. In addition to performing the saccade–opposite field viewing task, subject O.K. repeated the fixation task as well. Control group data (small crosses) are the same for both subjects in this task (three controls), performed in the same time period as the two subjects. (**d**) Absolute reaching errors for subject C.F. depicted in the same manner as **c**. Errors for the fixation task for subject C.F. are the same as in **Figure 1**.



subjects to make a saccade between viewing and reaching toward remembered visual targets.

We examined the reaching performance of two subjects diagnosed with optic ataxia. Subject O.K. is a right-handed 39-year-old male with right posterior parietal lobe damage (Brodmann's area 7 laterally and medially, with a slight extension into areas 39 and 40 and into the right posterior corpus callosum) caused by an ischemic stroke involving the posterior branch of the right sylvian artery[8]. Subject C.F. is a right-handed 27-year-old male who suffered from a watershed posterior infarct, resulting in distributed and asymmetrical bilateral lesions of the occipitoparietal region (Brodmann's area 18, 19, 7, 5 and 2) with a minute extension into the semiovale centers and a parietofrontal disconnection from intrahemispheric fiber lesions. Subjects provided informed written consent to participate in the experiment, which was pre-approved by the York University Human Participants Sub-Committee, Toronto, Canada, and authorized by the French Ministry of Research (22260S). At the time of testing, both subjects showed optic ataxia predominantly with the left hand in the left visual field, thought to be the consequence of damage to Brodmann's area 7 in the right hemisphere (larger than in the left hemisphere for C.F.), with no ataxia for targets in central vision and without any purely motor, somatosensory or visual deficits or any sign of neglect. Across all tasks, eight neurologically intact controls were also tested (age range = 24–40, mean ($M$) = 28.3).

In the first (fixation) task, horizontal directional errors were evaluated in different areas of the eye-centered visual field by varying eye position during reaches to a common central reaching target (**Fig. 1a** and **Supplementary Note** online). Having a constant target and different eye positions (as opposed to a constant fixation and different reaching targets) allowed us to minimize biomechanical and motor effects. We also isolated visuospatial effects by having subjects use their right (ipsilesional) hand: that is, we investigated only the 'visual field' effect[1]. Subjects were unaware that the location of the target was constant.

In this fixation task, both subjects show greater errors when the target was in their left visual field compared to when it was in their right visual field (**Fig. 1c,d**, white bars). For statistical purposes, absolute reaching errors were compared by grouping errors into two groups depending on where the target was relative to gaze (i) in the left visual field (LVF; rightward fixations) and (ii) in the right visual field (RVF; leftward fixations). Reaching errors for subject O.K. for leftward fixations (target in RVF, M = 1.24°) were significantly smaller than errors for rightward fixations (target in LVF, M = 3.35°; $t_{19} = 4.968$, $P < 0.001$). The same was true for subject C.F. (target in RVF: M = 3.99°, target in LVF: M = 8.29°, $t_{15} = 10.286$, $P < 0.001$). To summarize, although both subjects always reached toward the central target, they reached differently depending on the location of the target relative to gaze. These findings suggest that reaching errors vary based on target location in the visual field in gaze coordinates, not in arm or head coordinates (**Supplementary Note**).

In the second task (saccade–central viewing task), we investigated how the subjects (and controls) reached when first fixating on the target itself and then saccading to a right or left fixation position before reaching to the remembered target location (**Fig. 1b**). Unlike in the fixation task, the target was always viewed foveally. However, at the time of reaching, the eccentricity of the extinguished target relative to the final gaze, head, body and hand positions was identical in both conditions.

If the deficit in optic ataxia is determined by a static sensorimotor transformation based on initial encoding (that is, retinal input), which was always from the fovea (central viewing), the eye movement should have no effect on reaching. However, if optic ataxia involves a dynamic updated gaze-centered representation, then the errors should be based on final eye position and should be similar to errors made when the target was viewed peripherally.

The results showed that O.K.'s reaching errors toward the target in the LVF (M = 5.49°) were significantly greater than his reaching errors toward the target in the RVF (M = 0.98°, $t_{13} = 14.197$, $P < 0.001$), even though in both cases he viewed the target foveally (**Fig. 1c**, gray

bars). C.F. showed a similar pattern of reaching (**Fig. 1d**, gray bars) where his errors in reaching toward the LVF (M = 6.31°) were significantly greater than his errors in reaching toward the RVF (M = 3.34°, $t_{19} = 4.59$, $P < 0.001$). Although, relative to fixation errors, errors increased for subject O.K. and decreased for subject C.F., both subjects nevertheless showed greater reaching errors when an eye movement projected the target location toward the left visual field. This means that reaching errors did not depend on the initial retinal stimulus location, but rather on the final (remapped) coordinates of the central target relative to gaze.

In the latter task, subjects viewed the target foveally, which normally gives rise to a bilateral cortical representation[12]. It seems that the representation of the target location could have been internally remapped[10–14] either into the lesioned part of the cortex itself or into the damaged hemisphere at stages of processing earlier than those affected by the lesion. In order to distinguish between these alternatives, we created a saccade–opposite field viewing task. In this task, subjects viewed the target in their periphery as in the fixation task (**Fig. 2a**). However, before reaching to the target, they made a saccade to a second fixation position located on the opposite side of the target (**Fig. 2b**). This task was designed to test what happens when the subjects viewed the target in either the 'good' (**Fig. 2b**, left) or 'impaired' (**Fig. 2b**, right) visual hemifield, and then reached with the target positioned relative to the opposite hemifield.

**Figure 2c,d** shows the results from both subjects as well as their errors from the fixation task for comparison. Both subjects' errors for the saccade–opposite field task (gray bars) follow the same pattern as the fixation task (white bars). Other control tests (data not shown) in which subjects viewed and reached toward the target within the same hemifield did not show any significant saccade-dependent effects. Statistical analyses showed significant differences between errors made in the two visual fields for subject O.K. (**Fig. 2c**, target in LVF: 10.34°, target in RVF: 1.98°, $t_{19} = 15.644$, $P < 0.001$) and subject C.F. (**Fig. 2d**, target in LVF: M = 6.09°; target in RVF: M = 2.97°, $t_{17} = 5.653$, $P < 0.001$). This confirms that if subjects view the target in the good hemifield but reach with the extinguished target located relative to the opposite hemifield, they behave as if they had remapped the target into the impaired hemifield. Conversely, when the subjects viewed the targets in their impaired visual field, but reached—after a saccade—to the location of the target in their good visual field, they reached just as well as if they had viewed the target peripherally in their good visual field. This task showed that the initial visual information was still intact in that case; it was simply not being properly transformed.

Thus, it did not matter where on the retina the original visual information was; what mattered was the relative location of the extinguished target in gaze coordinates at the time of reaching, which in the second and third tasks could be calculated only by using an internal sense of eye movement to update the representation. This is inconsistent with damage to a static visuomotor transformation based on initial retinal encoding, but instead is consistent with a transformation based on dynamic gaze-centered internal representation of reach space[10–14]. The overall pattern of errors that we have observed suggests that the updating and storage processes are relatively intact; that is, for a given final eye position, reaching errors were similar regardless of the task. This is consistent with hypothetical remapping processes in the inferior parietal cortex[14] and observations of gaze-centered updating as early as the occipital cortex[15]. Therefore, we surmise that the deficit in optic ataxia rises from the visuomotor transformation that takes place after the updating stage (in the lesioned hemisphere), in the subsequent visuomotor transformation from this gaze-centered representation to the arm-centered representation for reaching.

Notably, this new procedure can be used clinically to test for optic ataxia in the presence of hemianopia, as it allows the examiner to present the target in the healthy visual field and test for optic ataxia within the hemianopic field.

In summary, our subjects showed greater errors for reaching in their left visual field regardless of whether the targets were extrinsically viewed peripheral targets or intrinsically remapped representations. We propose that the deficits that characterize optic ataxia result from faulty visuomotor transformation processes that depend on a dynamic gaze-centered spatial representation, rather than on a static early stage of spatial representation of the retinal image.

*Note: Supplementary information is available on the Nature Neuroscience website.*

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**COMPETING INTERESTS STATEMENT**

The authors declare that they have no competing financial interests.

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