AB014. Impaired anti-saccade production in posterior parietal cortex damaged patients

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Background: Performing an anti-saccade relies on two mechanisms: (I) inhibiting an automatic saccade to a target, and (II) generating, instead, a voluntary saccade to a location other than this visual target. It remains unclear where and how these two processes are implemented to ensure the production of correct anti-saccades. Previous research in optic ataxia has implicated the posterior parietal cortex (PPC) in anti-pointing, implying a possible role of the PPC in anti-saccade production.

Methods: Here, we tested how three patients with unilateral or bilateral damage to the PPC, as well as six neurologically intact controls, perform different types of anti-saccade: classic anti-saccades (180° rotation) or mirror saccades (90° rotation) across and within hemi-fields.

Results: We showed that PPC damaged patients were impaired in anti-saccade production for their contralesional visual fields. This was reflected in a longer period of erroneous pro-saccades, longer latencies associated with correct anti-saccades to the contralesional visual field and more imprecise anti-saccades.

Conclusions: Our results thus suggest that PPC damage results in delayed and prolonged competing saccade planning processes between two locations (i.e., visual target and saccade goal location). Taken together, our results provide evidence for a crucial role of the PPC in parallel mechanisms underlying anti-saccade performance.

Keywords: Saccade planning; anti-saccade; inhibition; posterior parietal cortex (PPC)

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