Sir,

We read with great interest the article entitled ‘Visual exploration in Parkinson’s disease and Parkinson’s disease dementia’ recently published in *Brain* by Archibald *et al.* (2013), because the pattern of results shows a remarkable coincidence with the results of our study on visual exploration in Parkinson’s disease (Matsumoto *et al.*, 2011a, 2012).

Archibald *et al.* (2013) investigated how cognitive function affects saccade amplitude and fixation duration during visual exploration in Parkinson’s disease. In an observation similar to our own (Matsumoto *et al.*, 2011a), they demonstrated that the visual exploration of complex figures in patients with Parkinson’s disease was characterized by a scanning pattern consisting of hypometric saccades with fixation durations longer than those seen in normal healthy subjects. Hallett (2011) pointed out that these ocular features in Parkinson’s disease could represent a type of bradykinesia called ‘ocular bradykinesia’, and that they might be caused by basal ganglia dysfunction. We also presented data supporting the contribution of basal ganglia dysfunction to the ocular bradykinesia and ocular paradoxical movement in Parkinson’s disease based on our experiments with a visual exploration task as well as oculomotor tasks (Matsumoto *et al.*, 2012).

An important implication of the Archibald *et al.* (2013) results is the influence of cognitive function on visual exploration. In experiments consisting of five kinds of tasks that demand higher brain function in which subjects matched comparator images against a central stimulus using four different images, Archibald *et al.* (2013) found that patients with Parkinson’s disease with dementia made saccades with more severe motor dysfunction as well as cognitive decline compared with those made by patients with Parkinson’s disease without cognitive impairment. They also showed that saccade amplitude and fixation duration did not largely differ between patients with Parkinson’s disease without cognitive impairment and normal healthy subjects. They propose that the observed changes in scanning behaviour reveal the efficiency with which fixations and saccades are deployed in the build-up to a cognitive response, and that the fixation duration in particular may be used as a predictor of cognitive decline. This is a very important point since the visual fixation pattern and duration of a visual scene is known to affect the perception and memory of that scene (Chapman *et al.*, 2003), and since changes in scanning behaviour may hamper the cognition of the scene explored.

It should be noted, however, that scanning parameters such as saccade amplitude and fixation duration can vary greatly depending on the task in use or even on the characteristics of the presented images. In our eye-tracking study, which excluded cognitively impaired patients with Parkinson’s disease (Matsumoto *et al.*, 2011a), patients with Parkinson’s disease memorizing images of varying complexity (a cube, two overlapping pentagons, a house, and the Rey-Osterrieth complex figure) made saccades with smaller amplitudes and longer fixation durations compared with healthy control subjects. Importantly, although patients with Parkinson’s disease made saccades with long fixation durations when scanning simple figures, they reduced their fixation durations when scanning more complex figures. Interestingly, the differences in scanning parameters between patients with Parkinson’s disease and normal healthy subjects did not correspond to differences in their rates of success at accurately reproducing the figures. Therefore, the differences in exploration strategy in patients with Parkinson’s disease did not directly translate into disruption of their visual memory of the images.

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Ocular paradoxical movement and severity of Parkinson’s disease

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These changes in scanning parameters emerged even though we did not explicitly instruct the subjects to scan the images with saccades. To explain this phenomenon, we proposed the notion of ‘ocular kinesie paradoxale’ or ‘ocular paradoxical movement’. The number of visual cues is greater in complex figures, and it seems that patients with Parkinson’s disease can use these visual cues to generate saccades. Similarly, akinetic patients with Parkinson’s disease can move easily if sensory guidance is present, such as when they are asked to walk along regularly-spaced lines drawn on the floor; this phenomenon is called somatomotor paradoxical movement (Glickstein and Stein, 1991; Okuma, 2006). Similarly, patients with Parkinson’s disease may be able to initiate saccades more efficiently, without ocular freezing, with the help of the many visual cues found in complex images.

The purposes, subjects, devices, tasks, etc. differ between our study and that of Archibald et al. (2013), but some aspects of their study merit comment based on our experience in a similar analysis, with particular reference to ocular paradoxical movement.

First, the tasks in their study used complex figures with many visual cues. When subjects visually explore such images, ocular paradoxical movement is expected to play a more important role than it does in the exploration of simpler images. It should have been especially important in viewing the overlapping figures, which contained abundant visual cues. However, Archibald et al. (2013) did not point out the possibility that ocular paradoxical movement could contribute to impairment of visual exploration in patients with Parkinson’s disease or Parkinson’s disease with dementia. Because all of their tasks contain relatively abundant visual cues, we assume that the similarity between saccade amplitudes and fixation durations in patients with Parkinson’s disease without cognitive impairment and those in healthy control subjects could be explained by ocular paradoxical movement.

Secondly, the severity of cognitive and motor dysfunction in Parkinson’s disease can influence not only the scanning parameters used during visual exploration, such as saccade amplitude and fixation duration, but also ocular paradoxical movement. Although the relationship between somatomotor paradoxical movement and the severity of motor symptoms in Parkinson’s disease remains to be elucidated, patients with Parkinson’s disease in the advanced stage are known to compensate for their motor deficits by relying on sensory guidance (Brown and Marsden, 1988). Neuroimaging studies have shown that, whereas the dysfunction of the supplementary motor area plays a substantial role in the pathophysiology of Parkinson’s disease (Playford et al., 1992; Samuel et al., 1997), enhanced lateral premotor cortex activity can compensate for impaired supplementary motor area function in the presence of visual input (Hanakawa et al., 1999). Although it is not clear whether this holds true in the oculomotor domain (Alexander et al., 1986), our study on saccade abnormalities in patients with Parkinson’s disease found that, as patients with Parkinson’s disease become progressively impaired in making voluntary saccades in the absence of visual signals, they tend to inadvertently make ‘reflexive’ saccades towards suddenly presented visual signals (Terao et al., 2011). Terao et al. (2013) suggested that these inadvertent saccades may emerge as a functional compensation for basal ganglia dysfunction in Parkinson’s disease. In patients with Parkinson’s disease with normal cognition, mild cognitive impairment, or Parkinson’s disease with dementia, these changes can arise from different extents of pathological burden in the oculomotor system, not only in the basal ganglia but also in the frontal cortex as well as the parieto-occipital cortex.

Finally, shifts in the strategy of visual exploration could also contribute to the differences between patients with Parkinson’s disease and Parkinson’s disease with dementia. In visual exploration, both the mechanism of top–down instruction (in which attention is allocated to an object in a goal-oriented manner) and that of bottom–up salience (in which attention is captured by a visually conspicuous object irrespective of the subject’s intention) are most likely at work. A similar shift from one exploratory strategy to the other is also noted in healthy control subjects depending on the context (Ludwig and Gilchrist, 2002; Terao et al., 2002; Matsumoto et al., 2011b). Some of the cognitively demanding tasks in Archibald et al. (2013) experiments would have predominantly engaged the top–down instruction mechanism, as the subjects had to actively search for a target (e.g. the overlapping figures task), whereas other easier tasks (e.g. the clock-matching task) may have involved the bottom–up salience mechanism. The interaction between the scanning strategy used in a particular task and oculomotor paradoxical movement must be considered, and can be assessed using images with minimal and maximal numbers of visual cues. To clarify this interaction, the authors could compare saccade amplitudes and fixation durations in tasks with varying levels of cognitive demand (as reflected in the varying error rates), and also varying numbers of visual cues, between patients with Parkinson’s disease and Parkinson’s disease with dementia.

The most difficult task, defined as that with the largest number of visual cues, would be the overlapping figures task. To reveal the effect of greater Parkinson’s disease severity on ocular paradoxical movement, a sub-analysis might be useful.

Taking all the data together, we consider that ocular paradoxical movement and visual exploration strategy both contributed to the results of the eye-tracking analysis reported by Archibald et al. (2013). We assume that an analysis comparing the scanning parameters used in simple and complex tasks by patients with Parkinson’s disease and Parkinson’s disease with dementia would yield information about how Parkinson’s disease severity relates to ocular paradoxical movement. Resolving the relationship between ocular paradoxical movement, scanning strategy and the severity of Parkinson’s disease through studies such as ours and that of Archibald et al. (2013) will certainly lead to a breakthrough regarding the pathophysiology of Parkinson’s disease and related cognitive impairments.

**Funding**

Dr Terao is supported by a Research Project Grant-in-Aid for Scientific Research from the Ministry of Education, Culture, Sports, Science, and Technology of Japan (22590954), and research funds from THE KATO MEMORIAL TRUST FOR NAMBYO RESEARCH, Japan. Dr Ugawa is supported by a Research Project Grant-in-Aid for Scientific Research from the Ministry of Education, Culture, Sports, Science, and Technology of Japan (22390181, 25293206), and grants from the Research...
Committee for rTMS Treatment of Parkinson’s Disease, the Research Committee for Intractable Pain, the Research Committee for Dystonia, and the Research Committee for Ataxic Disease, the Ministry of Health, Labour, and Welfare of Japan, and by grants from the Magnetic Health Science Foundation, NOVARTIS Foundation (Japan) for the Promotion of Science, and from the Uehara Memorial Foundation.

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