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Cerebellar Role in Eye Movement

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Although the cerebellum plays a key role in motor and non-motor behavior control, it remains controversial whether the cerebellar neurons are involved in the control of eye movement. Here Ohmae [1] and his colleagues have recently discovered neural correlation with saccadic eye movement in the cerebellar Dentate Nucleus (DN). Further Lu [2] and his research team have defined a new neural channel where the ocular cortical area, such as Supplementary Eye Field (SEF), remarkably receives inputs from cerebellar nucleus neurons. Given the above, it turns out to be the ironclad fact that the cerebellum plays a key role in performance of eye movements.

Impairment of eye movement following cerebellar inactivation during the cognitive behavior has long been reported [2-4]. Moreover, evidence that the cerebellum may link visual perception and eye movement comes from studies in subjects with autism: these individuals have reduced eye contact and poor activation in the SEF and cerebellum during eye movement task [5]. Furthermore, a consistent brain abnormality is a sharp reduction in the number of Purkinje cells in the cerebellum of the individuals with autism [6]. However, virtually nothing is known regarding the neural correlate to eye movement in the cerebellum, and whether the cerebellar projection to the cortical ocular motor area exits.

Here, Ohmane and his colleagues have historically performed single unit recording in the non-human primate DN during eye movements and analysis of the neural correlation with saccadic eye movement. They found that the DN neurons involve temporal detection of the external stimulus omission in order to produce saccades [1]. Furthermore, Lu and his co-researchers have recently performed rabies virus injections into the macaque SEF to determine whether the cerebellum and the basal ganglia neurons project to the SEF via the thalamus. They find that many neurons located in the DN and posterior interpositus nucleus of the cerebellum, and the internal globus pallidus of the basal ganglia were infected disynaptically via the thalamus (Lu et al., unpublished observation). Thus, these clearly define a new cerebllo-SEF circuit and a new anatomical substrate for the contribution of cerebellar and basal ganglia output to cognitive functions. Such progress has filled a critical void in our understanding of physiology and pathology of the cerebellum and cerebello-cortical channel. More aggressive progress determining both motor and non-motor roles of the cerebellum and the new neural pathway of the cerebello-SEF is desired. If so, difficult disease like Autism may have more hope for solution.

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