

## Nonvisual Eye Position Control in a Patient with Ocular Lateropulsion

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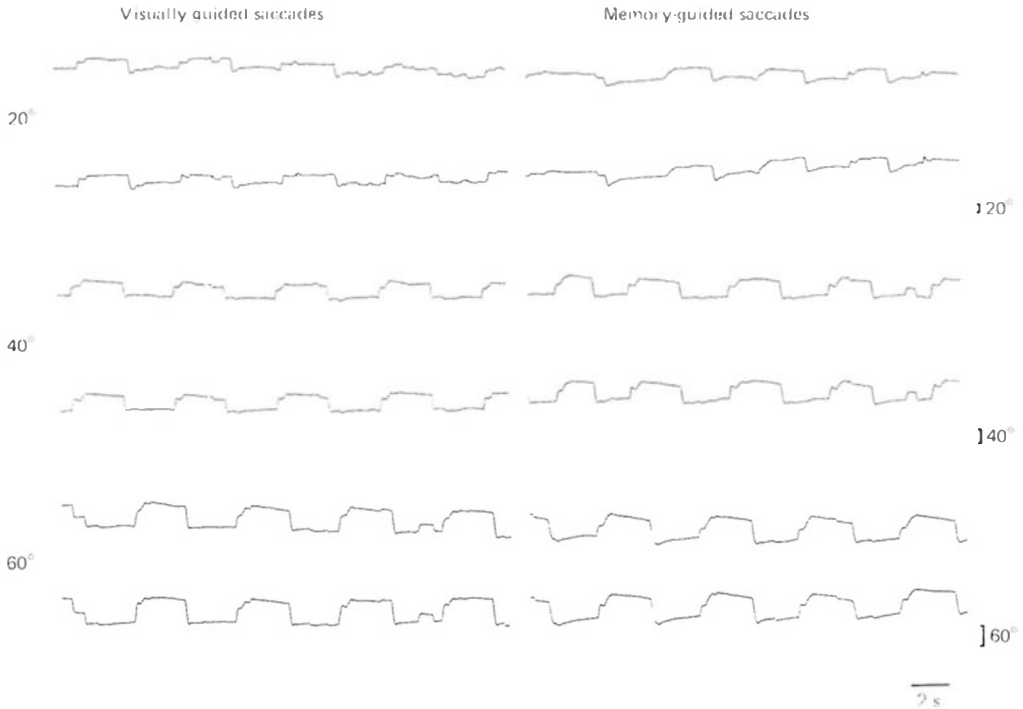
**Abstract.** For elucidating the nonvisual eye position control system, we studied the accuracy of saccades toward the remembered position (memory-guided saccades) in a patient with ocular lateropulsion having tonic bias of the eye position. Although the saccadic dysmetria caused by ocular lateropulsion was recorded during memory-guided saccades in the same manner as visually guided saccades, the eyes reached precisely the intended position by corrective saccades. The mean latency of corrective saccades was significantly longer than that of corrective saccades observed for visually guided saccades. This long latency of corrective saccades supports the assumption that feedback information of the eye position is a signal generated by orbital afferents rather than a copy of the motor command.

It is known that the eye position is accurately controlled without a visual fixation target [1-3]. Efference copy [4] or the afferent information from the orbit [5] relates to nonvisual eye position control. Currently, the efference copy theory is widely accepted, and the role of the afferent is still unknown in human subjects.

A patient with ocular lateropulsion has tonic bias of the eye position toward the side of the lesion [6-8]. The tonic bias is most prominent when fixational effort is inhibited. The eyes deviate tonically toward the side of the lesion when the patient closes his eyes.

Furthermore, it interferes with saccadic eye movements. Horizontal saccades directed to the side of the lesion overshoot the target, and contralateral-directed saccades undershoot the target. It is assumed that the tonic bias is generated after the level where the efference copy is evaluated [6]. Therefore, the patient with ocular lateropulsion will not be able to correct the tonic bias without the nonvisual afferent conveying the eye position information.

To evaluate the functional role of the nonvisual afferent in the control of eye position, we investigated the accuracy of saccades



**Fig. 1.** Examples of visually and memory-guided saccades of 20, 40 and 60°. Upper traces indicate right-eye movements, and lower traces indicate left-eye movements in each recording. Upward deflections denote rightward movements in each trace.

toward the remembered position in a patient with ocular lateropulsion in Wallenberg's syndrome. Our data indicate that the patient can move the eye accurately without visual information, despite the tonic bias of the eye position.

### Patient and Methods

A 51-year-old man was admitted to the hospital after the sudden onset of numbness of the left side of the face, nausea and vertigo. Neurologic examinations demonstrated: (1) dissociated analgesia (thermoanesthesia with preserved tactile sensation) on the left side of his face and the right side of his body; (2) Horner's syndrome of the left; (3) mild weakness and hyperre-

flexia in his left limbs; (4) tendency to stumble to the left, and (5) cerebellar signs in the left arm. Visual acuity was 20/20 in each eye. Visual fields and ocular fundi were normal. The patient's neurologic signs and symptoms were typical of Wallenberg's syndrome. Low density of the left lateral medulla oblongata was observed by computed tomography (CT) examination.

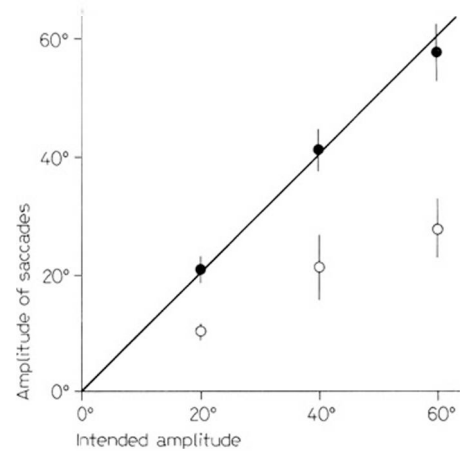
The patient was tested 12 days after the onset of the symptoms. Eye movements were recorded with direct-current electro-oculography. To evaluate the tonic bias of the eye position, the patient was instructed to fixate on a target, and then close the eye. A light-emitting diode was used as the target. To evaluate horizontal or vertical saccades and smooth pursuit, a laser dot projected onto a screen was used as the target. For saccades, the target moved stepwise with amplitudes of 20, 40, 60°, centering on the primary position in total darkness. Initially, saccades were performed with the

visual fixation point (visually guided saccades) for 30 s, and then the target was switched off. The patient was instructed to continue saccadic eye movements with the given amplitude (memory-guided saccades). For smooth pursuit, the target moved with a constant velocity (frequency 0.2 Hz, velocity 23°/s). Throughout the experiment, the patient was seated on a chair, and the chin, brow and head were fixed on a frame.

## Results

The ocular lateropulsion was observed by examination of the eye movements. On attempting visually guided saccades, horizontal saccades directed toward the side of the lesion overshot the target. The overshoot was marked for saccades of 20°, and indistinct for saccades of 40 and 60°. Contralaterally directed saccades undershot the target. The undershoot was recorded for all saccades of 20, 40 and 60°. The eye reached on the target with multiple corrective saccades (fig. 1). The intersaccadic interval between the initial saccade and the first corrective saccade was  $203 \pm 45$  ms (mean  $\pm$  SD,  $n=20$ ). Vertical saccades were deflected toward the side of the lesion by an aberrant horizontal component. Thus, all vertical saccades terminated to the left of the goal, but the eyes were immediately brought back on target by multiple corrective saccades. Horizontal pursuit was asymmetric. The velocity of horizontal pursuit directed to the intact side was decreased and pursuit movements were saccadic. When the fixation was suddenly inhibited by closing the eye, the eye position deviated by about 30° toward the side of the lesion. The deviation was achieved by a slow or fast eye movement, or by a combination of them.

On attempting memory-guided saccades, dysmetria and its corrective movements were recorded in the same manner as visually



**Fig. 2.** Initial and final amplitudes of memory-guided saccades directed to the right. The mean amplitude of initial saccades (○) and the mean of final amplitudes (●) are plotted against the intended amplitude. Bars represent the standard deviation.

guided saccades (fig. 1). However, the intersaccadic interval between the initial saccade and the first corrective saccade was  $250 \pm 47$  ms (mean  $\pm$  SD,  $n=20$ ), and was significantly longer than that of visually guided saccades (two-tailed *t* test,  $p<0.005$ ). In spite of the dysmetria, the eyes reached the area within  $\pm 5^\circ$  from the intended position by corrective saccades (fig. 2).

## Discussion

The present study shows that the saccadic dysmetria caused by ocular lateropulsion can be corrected without visual information. The final position error of memory-guided saccades was within  $\pm 5^\circ$ . In normal subjects, this error ranges from  $-3$  to  $11^\circ$  for 40° saccades [1]. Thus, these findings indicate that the patient has normal accuracy for nonvisual

eye position control. During visually guided saccades, corrective saccades occur depending upon the differences in space between the target and the target image on the retina (retinal error signal). On the other hand, during memory-guided saccades, the retinal error signal is not available to control the eye position.

Ocular lateropulsion with Wallenberg's syndrome may reflect vestibular imbalance [9]. The output of the vestibular nucleus projects directly to motoneurons. Thus, voluntary eye movements are perturbed by the tonic bias generated by vestibular imbalance at the motoneuron level. If tonic bias with lateropulsion can be corrected by the efference copy, the efference copy must originate in the motoneurons. However, that possibility seems very improbable, because motoneurons never project their axons to places other than extraocular muscles. The mean latency of corrective saccades during memory-guided saccades was significantly longer than that of corrective saccades during visually guided saccades. This fact indicates that the pattern of memory-guided saccades was not a repetition of visually guided saccades, and that another correction mechanism exists for memory-guided saccades. The long latency of corrective saccades observed for memory-guided saccades supports the assumption that feedback information of the eye position is a signal generated by orbital afferents, rather than a copy of the motor command.

It is assumed that extraocular muscle proprioception is a main signal in orbital afferents. The most proprioceptive fibers from the eye muscles run in the ophthalmic branch of the Vth nerve [10]. Fiorentini and Maffei [11] reported that eye oscillations are seen in the dark after section of the ophthalmic branch of the Vth nerve in the cat. The extraocular

muscle afferent mainly projects to the cerebellar vermis, lobules VI–VII [12, 13]. It is well known that this area plays an important role in the control of eye movements [14–16]. Recently, it has been reported that nonvisual eye position control is impaired in patients with cerebellar degeneration [17]. Kimura and Mackawa [18] suggested that the extraocular muscle afferent conveys the information of the eye position to the cerebellum. Thus, it is probable that the extraocular muscle afferent may be related to the nonvisual eye position control system through the cerebellum.

## References

- 1 Becker, W.; Fuchs, A. F.: Further properties of the human saccadic system: eye movements and correction saccades with and without visual fixation points. *Vision Res.* 9: 1247–1258 (1969).
- 2 Skavenski, A. A.; Steinman, R. M.: Control of eye position in the dark. *Vision Res.* 10: 193–203 (1970).
- 3 Ohtsuka, K.; Mukuno, K.; Ukai, K.; Ishikawa, S.: The origin of square wave jerks: conditions of fixation and microsaccades. *Jap. J. Ophthal.* 30: 209–215 (1986).
- 4 Helmholtz, H. von.: A treatise on physiological optics, vol. 3 (Dover, New York 1963).
- 5 Skavenski, A. A.: Inflow as a source of extraretinal eye position information. *Vision Res.* 12: 221–229 (1972).
- 6 Kommerell, G.; Hoyt, W. F.: Lateropulsion of saccadic eye movements. *Archs Neurol.* 28: 313–319 (1973).
- 7 Meyer, K. T.; Baloh, R. W.; Krohel, G. B.; Dow, B. M.: Ocular lateropulsion: a sign of lateral medullary disease. *Archs Ophthal.* 98: 1614–1616 (1980).
- 8 Baloh, R. W.; Yee, R. D.; Honrubia, V.: Eye movements in patients with Wallenberg's syndrome. *Ann. N.Y. Acad. Sci.* 374: 600–613 (1981).
- 9 Leigh, R. J.; Zee, D. S.: The neurology of eye movement (Davis, Philadelphia 1983).

- 10 Manni, E.; Bortolami, R.; Desole, C.: Eye muscle proprioception and the semilunar ganglion. *Expl Neurol.* 16: 226–236 (1966).
- 11 Fiorentini, A.; Maffei, L.: Instability of the eye in the dark and proprioception. *Nature* 269: 330–331 (1977).
- 12 Fuchs, A.F.; Kornhuber, H.H.: Extraocular muscle afferents to the cerebellum of the cat. *J. Physiol.* 200: 713–722 (1969).
- 13 Schwarz, D.W.F.; Tomlinson, R.D.: Neuronal responses to eye muscle stretch in cerebellar lobule VI of the cat. *Exp. Brain Res.* 27: 101–111 (1977).
- 14 Ritchie, L.: Effects of cerebellar lesions on saccadic eye movements. *J. Neurophysiol.* 39: 1246–1256 (1975).
- 15 Kase, M.; Miller, D.C.; Noda, H.: Discharges of Purkinje cells and mossy fibres in the cerebellar vermis of the monkey during saccadic eye movements and fixation. *J. Physiol.* 300: 539–555 (1980).
- 16 Ohtsuka, K.; Edamura, M.; Kawahara, K.; Aoki, M.: The properties of goal-directed eye movements evoked by microstimulation of the cerebellar vermis in the cat. *Neurosci. Lett.* 76: 173–178 (1987).
- 17 Ohtsuka, K.; Mukuno, K.; Sakai, H.; Ishikawa, S.: Eye position control in the dark in cerebellar degeneration. *Ophthalmologica* 196: 35–39 (1988).
- 18 Kimura, M.; Maekawa, K.: Activity of flocculus Purkinje cells during passive eye movements. *J. Neurophysiol.* 46: 1004–1017 (1981).

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