

Acquired Pendular Nystagmus with Voluntary Inhibition

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This report documents a case of voluntary inhibition of acquired pendular nystagmus after head trauma. A 30-year-old male developed oscillopsia and decreased visual acuity, as well as findings of acquired pendular nystagmus with voluntary inhibition after head trauma. The EOG finding was horizontal 18-20Hz bilateral symmetrical pendular nystagmus in all directions of gaze at near and distant fixation. Nystagmus did not change with 14 Prism Diopter base-out prisms on both eyes, but it was possible to abolish it intentionally. Baclofen and Clonazepam had no effect in improving the patient's symptoms and EOG finding.

Key Words: Acquired pendular nystagmus, head trauma, voluntary inhibition

INTRODUCTION

Pendular nystagmus is a sinusoidal oscillation of the eyes which may be congenital or acquired in nervous diseases. Congenital pendular nystagmus is horizontal, binocular and conjugate, and can have a variable wave form and changes into a jerk nystagmus on lateral gaze. But, acquired pendular nystagmus is characterized by monocular or binocular, conjugate or disjunctive, independent eyeball motion with vertical, horizontal and torsional nystagmus. It is also associated with neurologic abnormalities.¹ This report documents a case of voluntary inhibition of acquired pendular nystagmus after head trauma suffered in an automobile accident.

CASE REPORT

A 30-year-old male visited our ophthalmologic

clinic due to the sudden onset of oscillopsia several times a day and lasting for more than 1 hour. The condition decreased visual acuity and remained regardless of eye movement or fixation, unless voluntarily inhibited. Neuro-ophthalmologic examination, electrooculography (EOG), and brain and cervical spine MRI were performed.

General physical examination showed signs of cervical radiculopathy involving the right fourth to sixth segments with shoulder pain. Monocular distance visual acuity was 20/70 in the right eye and 20/20 in the left eye. However, during oscillopsia, the patient's binocular visual acuity was finger counting at 30cm. Visual fields were full using kinetic Goldman perimetry and the pupils reacted well to light and accommodation. Visual Evoked Potential findings were normal. The brain MRI finding was normal except prominent cavum septum pellucidum and partial empty sella, and cervical spine MRI finding showed herniated cervical disc at C4-C5 and C5-C6. The EOG finding showed horizontal 18-20 Hz bilateral symmetric pendular nystagmus in all directions of gaze at near and distant fixation (Fig. 1). There were no associated head movements. However, the nystagmus could be intentionally inhibited, both at near and distant. The patient said that he could "intentionally reduce the oscillopsia." When we ordered, "stop the nystagmus!", it decreased in amplitude and frequency following voluntary inhibition (Fig. 2). The nystagmus did not change when 14 Prism Diopter base-out prisms were placed on both eyes to evoke near convergence. Saccadic and pursuit eye movements were normal when the nystagmus was intentionally inhibited. Horizontal pursuit was interrupted by conjugate, horizontal oscilla-

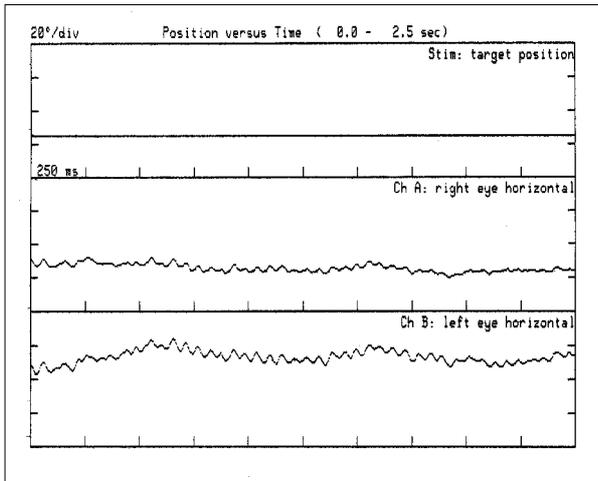


Fig. 1. The electrooculographic (EOG) finding showed horizontal 18-20 Hz bilateral symmetrical pendular nystagmus without voluntary inhibition.

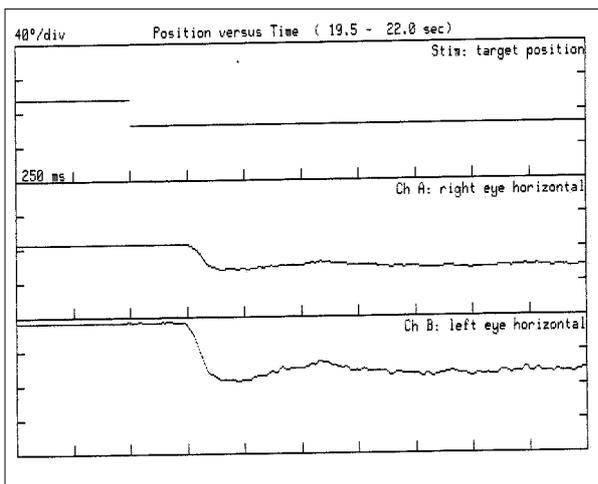


Fig. 2. The electrooculographic (EOG) finding showed horizontal pendular nystagmus after voluntary inhibition.

tion without intentional inhibition. We began treatment with baclofen, but his symptoms did not improve. The drug was tapered off and replaced by clonazepam for 3 months, but it had no beneficial effect on the symptoms on EOG findings. We stopped it and observed the patient regularly on an outpatient clinic basis.

DISCUSSION

The etiology of acquired pendular nystagmus includes multiple sclerosis, brain stem stroke,

toluene toxicity, spasmus nutans and myoclonus syndrome.¹ Their MRIs were characterized by multiple areas of abnormal signal in the red nucleus, the central tegmental tract, the medial vestibular nucleus and the inferior olive. Patients with horizontal pendular nystagmus predominantly show pontine lesions, whereas patients with torsional pendular nystagmus generally show medullary involvement.² Many hypotheses of its mechanism have been suggested, including abnormalities of internal feedback circuits, such as the reciprocal connections between brainstem nuclei and cerebellum; disruption of pathways proximal to the oculomotor nuclei;³ disturbance of cholinergic mechanisms; and multiple structural lesions, predominantly in the pons and the midbrain.⁴ Recently, gabapentin and baclofen is used.⁵ Recent experimental studies have indicated that the inhibitory neurotransmitter γ -aminobutyric acid (GABA) plays an important role in the normal mechanism by which gaze is held steady during visual fixation of an object of interest.⁵ The mechanism of action of gabapentin remains undetermined. Although it is structurally similar to GABA, gabapentin shows no direct activity on common GABAA or GABAB receptors or uptake carriers.⁶ Gabapentin enhances GABA synthesis by increasing activity of glutamic acid decarboxylase, decreases GABA degradation by inhibiting GABA transaminase, increases GABA turnover, and promotes release of GABA.⁵ Baclofen, a GABAB agonist, inhibits velocity storage, and this explains its effectiveness in treating periodic alternating nystagmus, which is viewed as being due to excessive velocity storage, secondary to loss of inhibition from the nodulus and uvula.^{5,7} The other treatment modalities included trihexyphenidyl, isoniazid, base-out converging prisms, scopolamine, benzotropine, botulinum toxin into the retrobulbar area or rectus muscles, and a glutamate antagonist.⁵

This case of acquired pendular nystagmus illustrated a rare condition that can be inhibited voluntarily after sudden head trauma. The mechanism of this phenomenon is not completely understood and further study will be needed.

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