Hyperdeviation and Static Ocular Counterroll in Unilateral Abducens Nerve Palsy

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INTRODUCTION

When a vertical strabismus accompanies sixth nerve palsy, multiple cranial nerve palsy or skew deviation should be considered. The purpose of this study is to detect and determine the magnitude of vertical deviation in patients with unilateral sixth nerve palsy.

METHODS

Twenty-seven patients with unilateral sixth nerve palsy and ten normal subjects were studied. The range of ductions was examined and the degree of abduction defect was recorded. The amount of horizontal and vertical deviations was measured in nine diagnostic positions by the prism and cover test, Maddox rod and prism test, and magnetic scleral search coils.

MRI with enhancement was performed for patients under 50 years of age and those with neurologic signs. CT with contrast was obtained in patients with ischemic risk factors and those over 50 years of age. If CT was normal, patients were followed at about 3 months. Those without improvement at 3 months and those with an abnormal CT were further investigated with MRI. We report here the primary deviations in our subjects; the results for secondary deviations were similar. ANOVA was used for statistical analysis.

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RESULTS

Vertical Deviations in Nine Diagnostic Positions

Mean vertical deviations, for all positions of gaze, in peripheral palsy (n = 20) were: 0.3 ± 0.8 prism diopters (PD) by prism-cover test, 1.3 ± 1.6 PD by Maddox test, and 2.0 ± 1.4 PD by coil recordings. Mean vertical deviations in normal subjects were: 0.0 ± 0.0 PD by prism-cover test, 1.0 ± 0.9 PD by Maddox test, and 1.9 ± 2.1 PD by coil recordings. Therefore, peripheral palsy did not cause abnormal vertical deviation. In central palsy (n = 7), mean vertical deviations were: 0.9 ± 1.3 PD by prism-cover test, 1.4 ± 1.6 PD by Maddox test, and 2.5 ± 1.6 PD by coil recordings; they were not different from normal values. There was no difference in mean or maximum vertical deviations between patients and normal controls (FIG. 1). The vertical deviations were comitant in individual patients and in normal subjects, as were the group means. The maximum difference between hyperdeviation on up- and down-gaze in any individual patients was 3 PD.

Vertical Deviations during Static Head Roll

Eighteen of 20 patients (90%) with peripheral palsy exhibited a right hyperdeviation (mean = 4.3 PD) on static lateral head tilt to the right shoulder (right head tilt), and a left hyperdeviation (mean = 2.9 PD) on lateral tilt toward the left shoulder (left head tilt), regardless of the side of palsy (FIG. 2) (p < 0.001). In contrast, four of seven patients (57%) with central palsy had hyperdeviation that remained on the same side during static lateral head tilt to either side (FIG. 2). Mean deviations were 2.1 PD right hyperdeviation on right head tilt, and 1.3 PD right hyperdeviation on left head tilt.

DISCUSSION

A small hypertropia can be detected in patients with peripheral and central sixth nerve palsy. This hypertropia falls within the normal range of hyperphoria seen in
healthy subjects, indicating that it is a normal hyperphoria that becomes manifest in
the presence of esotropia. In normal subjects, the mean vertical deviation in the
straight-ahead position is 1.52 ± 1.49 PD. Thus, in patients with sixth nerve palsy, if
a hypertropia is detected in the straight-ahead position, which is less than or equal
to 5 PD (normal mean + 2 SD), investigation for multiple cranial nerve palsy or skew
deveiation is not indicated.

Static head tilt stimulates otolith receptors, leading to ocular counterroll and a
small change in vertical alignment in normal subjects. However, when the otolith–
ocular reflex pathway is disrupted, ocular torsion and skew deviation are observed.
This indicates that under normal circumstances, the otolith–ocular reflex is sym-
metrical and balanced; it is also suppressed during static head roll. Disruption of
binocular vision may remove the suppression on the otolith–ocular reflex, and lead
to the pattern of right hyperdeviation on right head tilt and left hyperdeviation on
left head tilt observed in peripheral palsy. In contrast, in central fascicular sixth
nerve palsy, unilateral brainstem lesions can also disrupt the balance of the otolith–
ocular reflex and lead to the pattern of vertical deviation that remains on the same
side regardless of the direction of head roll. This pattern of hyperdeviation induced
by lateral head tilt may warrant investigation for a brainstem lesion as the cause of
paretic abduction.

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