The influence of head tilt on ocular torsion in patients with superior oblique muscle palsy

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INTRODUCTION Patients with superior oblique muscle palsy often assume a compensatory head tilt to the contralateral side of the affected eye. This tilt serves to decrease the hypertropia and, in theory, might decrease the excyclotropia.

METHODS A prospective investigation was undertaken of the effect of forced head tilt to the right and left on torsion and the hypertropia in patients with unilateral superior oblique muscle palsy.

RESULTS Twenty patients with unilateral superior oblique muscle palsy had a mean decrease in their hypertropia of $6.4^{\Delta} \pm 3.9^{\Delta}$ on forced head tilt from the head-erect position toward the contralateral side (95% CI, 4.7-8.1). This finding represented a decrease of 62% of the hypertropia found in the head-erect position. The difference was significant ($p < 0.001$, paired t-test). The mean decrease of the excyclotropia was only $0.25° \pm 0.6°$ on contralateral head tilt, which represented 3% of the excyclotropia in the head-erect position. This difference was not significant ($p < 0.06$). The percent by which the hypertropia decreased from the head-erect position to that found on contralateral head tilt was significantly greater than the amount by which the excyclotropia decreased ($p < 0.001$).

CONCLUSIONS With contralateral head tilt, patients with superior oblique muscle palsy demonstrate a significant decrease in their hypertropia but essentially no change in their excyclotropia. The compensatory head tilt they manifest appears to serve the purpose of decreasing the vertical but not the torsional misalignment. (J AAPOS 2009;13:132-135)

Patients with a unilateral superior oblique muscle palsy have a hypertropia of the paretic eye that increases on ipsilateral head tilt and decreases on contralateral head tilt.[1] In addition they also have an excyclotropia of the affected eye. They typically assume a compensatory head posture characterized by a tilt to the contralateral side, which has the effect of decreasing the hypertropia.[1,2] Many authorities speculate that the purpose for the compensatory head tilt is this decrease in the hypertropia.[3-6] However, in theory the compensatory head posture also may serve to decrease the excyclotropia, particularly if the compensatory head tilt is large. Classical teaching dictates that the incyclotorotary muscles (superior oblique and superior rectus muscles) are stimulated on ipsilateral head tilt and the excyclorotary muscles (inferior oblique and inferior rectus muscles) are stimulated on contralateral head tilt.[6] Thus it is theoretically possible that a compensatory head tilt to the opposite side of the affected eye would decrease the excyclotropia, because this would put the head in a position in which the paretic superior oblique muscle is inhibited and should thus not contribute to the torsional alignment. On the other hand, the position of an eye after head tilt is the result of a complex interplay of all of the cyclovertical muscles and it can be difficult to draw a conclusions based on the analysis of one particular muscle.[6-9] The purpose of this study is to determine whether the excyclotropia in patients with unilateral superior oblique muscle palsy changes on ipsilateral and contralateral head tilt, and to determine the possible influence of a change in torsion on the compensatory head tilt seen in this patient population.

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Subjects and Methods

This is a prospective study of patients diagnosed and treated by the author for unilateral superior oblique muscle palsy. The study protocol was written before patient enrollment and specified examination technique, data to be collected, inclusion and exclusion criteria, hypothesis tested, and statistical methods used. The study was approved by the University of Wisconsin Institutional Review Board and was compliant with the Health Insurance Portability and Accounting Act. Patients provided written informed consent to participate.

Patients were recruited consecutively from my practice with the only other exclusion criteria being the inability to perform reliably on the sensory tests needed for this study. There was no arbitrary age limit for inclusion. Diagnosis of unilateral superior oblique muscle palsy was based on the 3-step test[1] plus the presence of an excyclotropia (either subjective, as seen with the double Maddox rod test, or objective, as seen on fundoscopy),[10] the absence of a reversal of the hypertropia in any of the nine diagnostic fields of gaze or head tilt right and left, and the absence of dissociated vertical divergence. To rule out the possibility that any of these patients may have in fact had a bilateral masked superior oblique muscle palsy, the protocol prospectively included the criteria that any patient undergoing surgery had a satisfactory outcome with elimination of symptoms and no reversal of the hypertropia by 6 months after surgery.[11,12] Finally, a history compatible with the diagnosis of superior oblique muscle palsy had to be elicited. This meant that the hypertropia and diplopia began either after closed head trauma or neurologic insult or dated back to childhood, with a longstanding history of a slowly decompensating hypertropia. In the former, I categorized it as an acquired problem; in the latter, as being a presumed congenital problem. If presumed to be congenital, the history of a longstanding compensatory head posture was documented in all cases by a review of old photographs as well as the presence of facial asymmetry.[13] All patients who underwent surgery had either a recession of the ipsilateral inferior oblique muscle, or a recession of the ipsilateral inferior oblique muscle combined with a recession of the contralateral inferior rectus muscle using either an adjustable suture or semi-adjustable suture technique.[14]

The Cervical Range of Motion (CROM) device (Performance Attainment Associates, Roseville, MN)[15] was used to measure the spontaneous compensatory head tilt in all patients while they read Snellen optotypes at threshold acuity at a distance of 6 meters while wearing appropriate optical correction. In brief, the CROM device is a head-mounted unit with a gravity metered scale, which has been shown to measure head tilts with a reproducibility and accuracy of 1°.[15] The angle of misalignment was measured in the nine diagnostic fields of gaze at 6 meters and on head tilt right and left at 6 meters with the appropriate optical correction in place.

Subjective torsion was measured with the double Maddox rod test using white and red lenses that were etched to accurately denote the 90° and 180° positions. First, the paretic eye was occluded and the white Maddox rod was placed over the nonparetic eye. The Maddox rod was then rotated until the patient perceived the white line as being vertical while the head was in the straight upright position. Next the paretic eye was uncovered and the red Maddox rod was placed in front of it, initially oriented so that the line was approximately 30° counterclockwise from the vertical position. It was then slowly rotated clockwise until the patient perceived the red and white lines as being parallel or superimposed, and the amount of torsion was recorded to the nearest 1°. The red Maddox rod was then rotated approximately 30° clockwise from the vertical position and slowly rotated back toward the 90° orientation until the subject saw the lines as parallel or superimposed, and this reading of torsion also was recorded. These were repeated in sequence for a total of 6 readings, 3 from each direction to assess accuracy and reproducibility for each subject. The 6 readings were then averaged, rounded to the nearest 1°, and used for the data analysis. If any of the readings varied by more than 2° for any subject, the patient was considered unreliable and excluded from the study. This exclusion criterion was in place before data review. The patient's head was then tilted approximately 30° to the contralateral side. The patient was first asked to orient the line perceived from the white Maddox rod vertically from floor to ceiling while the paretic eye was again occluded. This reading should have approximated the magnitude of the head tilt, with any difference between the size of the head tilt and the reading probably representing partial compensatory countertorsion.[6] The red Maddox rod was then placed over the paretic eye, initially at an oblique angle to the orientation of the white Maddox rod, as was done with the head erect, and rotated until the line it created was perceived as being parallel with or superimposed on the line created by the white Maddox rod. These readings were repeated 6 times in the aforementioned manner. The difference between the two readings of the white and red Maddox rods would represent the total torsional disparity, irrespective of which eye was abnormally torted (Figure 1). A set of 6 measurements was obtained in a similar manner after ipsilateral head tilt, and the same exclusion criteria of accuracy within 2° for all measurements applied. In an earlier unpublished pilot study, I tested subjective torsion in 10 cooperative patients in the manner used in this study. They each had 6 measurements of torsion in the head-erect position as well as 6 with the head tilted to right and left, respectively. In each head position, the mean value for the 6 measurements was calculated. In all 10 patients, each of the 6 measurements in each head position was within 2° of the mean; in 4 patients each measurement was within 1° of the mean. I thus concluded that using the scored Maddox rods as seen in Figure 1 in the manner employed in this study has an accuracy of 2° with respect to reproducibility. Classically, the double Maddox rod test is performed with the lenses oriented so the subject perceives them as being horizontal, because of a minor artifact that occurs when trying to assess the vertical subjective meridian.[16,17] However, I have determined when their head is tilted, subjects have an easier time orienting the Maddox rod vertically (eg, from floor to ceiling), than trying to assess the true horizontal plane as is classically taught for double Maddox rod testing (unpublished pilot study). Because the same orientation of the lenses was used in this study for all measurements, any error induced by this artifact would not affect the results as it would cancel itself.

Results

Initially, 23 patients were enrolled in the study; however, 3 were excluded as the result of unreliable measurements. No patients were excluded as a result of being found to have a bilateral masked superior oblique muscle palsy after surgery for what was thought to have been a unilateral problem. Thus the series consists of twenty patients, the demographics of whom are presented in e-supplement 1 (available at jaapos.org). Eleven subjects were male and 9 were female. The ratio of right to left eyes was 8 to 12, respectively, and the ratio of acquired to presumed congenital causes was also 8 to 12, respectively. Of the 20 patients, 16 underwent surgery and 4 were treated conservatively (Cases 8, 10, 14, and 16) There was a mean decrease in the hypertropia from the head-erect measurement to

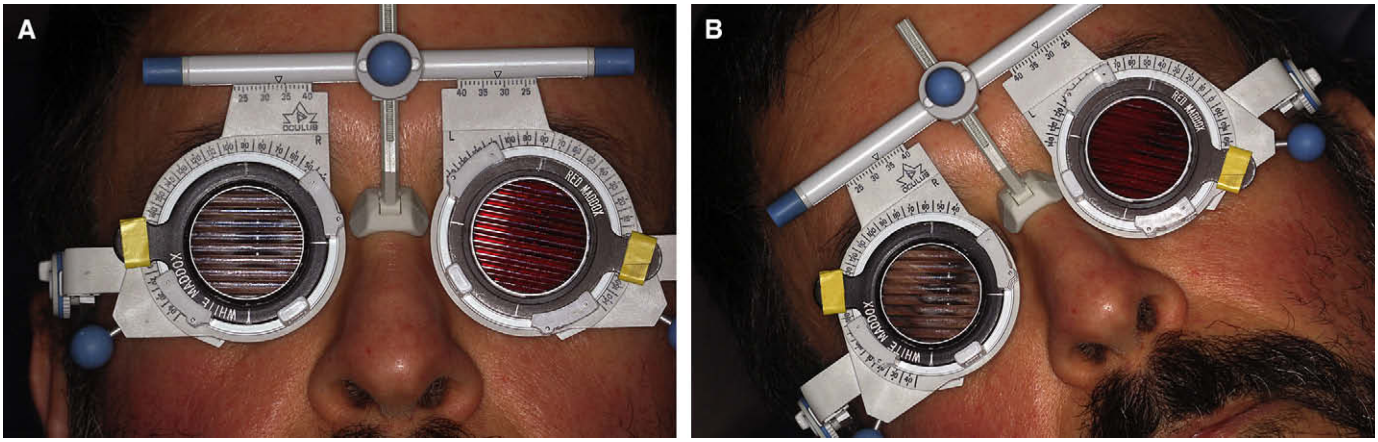

FIG 1. This patient (Case 10) has a left superior oblique muscle palsy associated with a 10° left excyclotropia in the head-erect position (A). With a head tilt of approximately 30° to the right, he orients the white Maddox rod over the nonparetic right eye as being vertical when it is positioned at 55° in the trial frame (B). The paretic left eye perceives the lines as parallel when the red Maddox rod is oriented at 45°, indicating 10° of excyclotropia. Note that there is a difference of 5° between the 30° of head tilt and the 35° of rotation needed to perceive the white Maddox rod as vertical. This probably represents partial compensatory countertorsion.

that found on contralateral head tilt of $6.4^{\Delta} \pm 3.9^{\Delta}$ (95% CI, 4.7-8.1). This finding represented a mean decrease of 62% ± 34% of the head-erect hypertropia and was significant ($p < 0.001$, paired *t*-test). The mean decrease in the excyclotropia from the head-erect measurement to that found on contralateral head tilt was only 0.25° ± 0.6°. This represented a mean decrease of 3% ± 7% of the head-erect excyclotropia and was not significant ($p < 0.06$, paired *t*-test). The percent by which the hypertropia decreased from the head-erect position to that found on contralateral head tilt was 62% ± 34%, which was significantly greater than the 3% ± 7%—the amount by which the excyclotropia decreased ($p < 0.001$, paired *t*-test). Although most patients had a difference in their excyclotropia of 1° or 2° between the head-erect or forced tilt readings, for at least one of the 6 readings taken only 2 patients showed any difference at all in their torsion on head tilt when the results of their 6 readings were averaged.

Discussion

The data show that the effect of a compensatory head posture in patients with unilateral superior oblique muscle palsy is to minimize the vertical deviation rather than to decrease ocular torsion. Although theoretically one might assume that extorsion would decrease with contralateral head tilt in patients with superior oblique muscle palsy, many factors come into play.[18] In patients with unilateral superior oblique muscle palsy the ipsilateral inferior oblique muscle is unopposed and becomes overacting. This could result in a relative increase in the excyclotropia on contralateral head tilt as compared with what occurs in normal individuals. Although this is speculation, it is a possible mechanism that is consistent with the observations made herein.

There are some practical implications from this study. If the presence of a large compensatory head tilt indicated that a patient had a greater torsional problem than in a patient with a smaller compensatory head tilt, surgery on the oblique muscles might be necessary in such a patient to correct the greater amount of torsion. Although surgery to treat unilateral superior oblique muscle palsy frequently does involve oblique muscle surgery, there are clinical situations in which the motility pattern may indicate that rectus muscle surgery alone may be preferable, particularly if previous oblique muscle surgery already has been performed. This study does not imply that torsion does not need to be addressed in patients with superior oblique muscle palsy. It merely indicates that the magnitude of the head tilt is not the driving force in making the decision as to which muscles should be operated upon. Instead, the decision should be made based on the size of the hypertropia and excyclotropia found. One limitation of this study is that measurements were all made by the author and were not masked.

It appears that the compensatory head posture in patients with unilateral superior oblique muscle palsy is not associated with a decrease in the excyclotropia and may serve the purpose of decreasing the hypertropia rather than the excyclotropia.

References

1. Parks M. Isolated cyclovertical palsy. Arch Ophthalmol 1958;60:1027-35.
2. Kushner B. Ocular causes of abnormal head postures. Ophthalmology 1979;86:2115-25.
3. Knapp P, Moore S. Diagnosis and surgical options in superior oblique surgery. Int Ophthalmol Clin 1976;16:137-49.
4. Jampolsky A. Management of vertical strabismus. Symposium on Pediatric Ophthalmology: Transactions of the New Orleans Acad Ophthalmol. New York: Raven Press; 1986. p. 141–71.

5. Prieto-Diaz J, Souza-Dias C. Oculomotor paresis and paralysis. Strabismus. Oxford: Butterworth Heinemann; 2000. p. 289–358.
6. Kushner BJ. Ocular torsion: rotations around the “WHY” axis. J AAPOS 2004;8:1-12.
7. Kushner BJ, Kraft S. Ocular torsional movements in normal humans. Am J Ophthalmol 1983;95:752-62.
8. Kushner BJ, Kraft SE, Vrabec M. Ocular torsional movements in humans with normal and abnormal ocular motility—Part I: Objective measurements. J Pediatr Ophthalmol Strabismus 1984;21:172-7.
9. Kushner BJ. Ocular torsional movements in humans with normal and abnormal ocular motility: Part II—subjective observations. J Pediatr Ophthalmol Strabismus 1986;23:4-11.
10. Morton G, Lucchese N, Kushner B. The role of fundoscopy and fundus photography in strabismus diagnosis. Ophthalmology 1983;90: 1186-91.
11. Kushner BJ. The diagnosis and treatment of bilateral masked superior oblique palsy. Am J Ophthalmol 1988;105:186-94.
12. Kushner B. Errors in the three-step test. Ophthalmology 1989;96: 127-32.
13. Wilson M, Hoxie J. Facial asymmetry in superior oblique palsy. J Pediatr Ophthalmol Strabismus 1993;30:315-18.
14. Kushner BJ. An evaluation of the semiadjustable suture strabismus surgical procedure. J AAPOS 2004;8:481-7.
15. Kushner B. The usefulness of the cervical range of motion device in the ocular motility examination. Arch Ophthalmol 2000;118: 946-50.
16. von Noorden GK, Campos EC. Examination of the patient—II. Binocular Vision and Ocular Motility. St. Louis: Mosby, 2002:168–210.
17. von Noorden GK, Campos EC. Binocular vision and space perception. Binocular Vision and Ocular Motility. St. Louis: Mosby, 2002. p. 7–37.
18. Kushner BJ. Torsion as a contributing cause of the anti-elevation syndrome. J AAPOS 2001;5:172-7.

An Eye on the Arts — The Arts on the Eye

My world is built of touch-sensations, devoid of physical color and sound; but without color and sound it breathes and throbs with life. Every object is associated in my mind with tactile qualities which, combined in countless ways, give me a sense of power, of beauty, or of incongruity.

—Helen Keller, *The World I Live In*. (Quoted from *The Color of Angels by Constance Glassen*)