Published in final edited form as:

J AAPOS. 2012 April ; 16(2): 141–144. doi:10.1016/j.jaapos.2011.11.009.

Torsional augmentation for the treatment of lateropulsion and torticollis in partial ocular tilt reaction

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Abstract

Purpose—We describe a new surgical treatment for lateropulsion and torticollis arising from an ocular tilt reaction.

Methods—Visual sensory parameters and postural abnormalities were retrospectively reviewed in a patient with a partial ocular tilt reaction who was treated with horizontal transposition of the vertical rectus muscles to surgically augment preexisting binocular torsion.

Results—Torsional augmentation eliminated the sensation of lateropulsion and produced almost complete resolution of her torticollis and associated body tilt.

Conclusions—Binocular torsional augmentation can be used to reduce both lateropulsion and torticollis in the setting of an ocular tilt reaction.

The ocular tilt reaction is usually caused by unilateral injury to the brainstem or utricular pathways subserving central vestibular function in the roll plane.^{1–3} Mechanistically, a lesion of the right utricular pathways is equivalent to activation of the left utricle, creating a tilt of the subjective visual vertical to the right (as if the head were being tilted to the right to stimulate the left utricle).^{1–3} This imbalance evokes a compensatory ocular tilt reaction, comprising skew deviation, binocular torsion, and a head tilt toward the side that the patient erroneously perceives as vertical (Figure 1A).^{1–3} Because the head tilt is not compensatory for diplopia (unlike in superior oblique palsy), surgical treatment of the vertical misalignment will not improve the associated torticollis.⁴

As the natural history of the ocular tilt reaction is often one of spontaneous resolution,⁵ it is considered advisable to observe patients initially and to treat any associated diplopia conservatively with prisms. The associated torsion (consisting of an intorsion in the hypertropic eye and an extorsion in the hypotropic eye) produces a fairly conjugate torsional deviation that does not create an impediment to fusion once any vertical deviation resolves. If a vertical deviation fails to resolve, small recessions of the superior rectus muscle (in the higher eye) or inferior rectus muscle (in the lower eye) will often restore single binocular vision, but such an approach does not address the head tilt.^{6,7}

Head tilt is usually a minor component of the ocular tilt reaction and rarely requires treatment. In rare instances, however, the patient may report a compelling sensation of being pulled to one side (lateropulsion) or a large persistent head tilt.⁸ In this situation, we reasoned that paradoxically increasing the torsional position of the eyes to further rotate the eyes in the direction of the tilt would realign the vertical poles of eyes more closely with the tilted subjective visual vertical, thereby obviating the compensatory function of the head tilt (Figure 1B). Since torsional rotation of both eyes counter-rotates the visual image, this procedure should counter-rotate the tilted subjective visual vertical back toward the gravitational vertical, thereby reducing the error signal that generates the head tilt. To test this hypothesis, we performed horizontal transposition of the vertical rectus muscles to augment the preexisting binocular torsion as a means of treating lateropulsion and persistent torticollis in a patient with a partial ocular tilt reaction.

Case Report

Institutional review board approval was obtained for retrospective chart review and the study conformed to the requirements of the United States Health Insurance Portability and Accountability Act. A 59-year-old woman presented with a 2-year history of lateropulsion with perpetual tilting of her whole body to the right. The onset of her symptoms had been accompanied by acute vertigo and nausea, which resolved gradually over a two-week period. When placed upright, she reported that it felt as though other people were tilted to the right. Family members reported that pictures she hung on the wall were tilted to the right despite the fact that they appeared straight to her. Her lateropulsion did not become noticeably worse in the dark. Magnetic resonance imaging of the brain showed a few nonspecific foci of T2-hyperintensities in the periventricular and subcortical white matter of both cerebral hemispheres, most likely representing ischemic small-vessel disease without an infarction. Vestibular evaluation showed no objective findings of peripheral vestibular disease. Testing of the subjective visual vertical in the dark with her head held in the neutral position demonstrated an average of 12.3° bias to the right, which was well beyond two standard deviations of normal, that is, 4° in either direction.

On presenting to the Strabismus Service at Mayo Clinic, Rochester, Minnesota, she was unable to properly judge vertical orientation of objects in her environment. On our examination, she had right head tilt that varied from 25° to 45° and a constant tendency to lean her body to the right (Figure 2A), resulting in difficulty sitting in a chair without falling out of the chair and walking down a corridor without bumping into the wall. A compelling sense of being pulled to the right (lateropulsion) caused her to lean rightward until gravitational constraints (preventing a fall) or physical constraints (walking into walls or pushing against the arm of her chair) caused her to stop.

Her ocular motility showed full ductions and versions, and she was orthotropic at distance straight ahead and at near fixation. She had no gaze-evoked or torsional nystagmus. Prism and alternate cover test revealed 6Δ exophoria and 1Δ left hyperphoria at distance straight ahead and 12Δ of exophoria at near. The left hyperphoria did not increase on lateral gaze, up or down, and there was no measurable hyperphoria on either right or left head tilt. Testing with double Maddox rods with her head and body in the neutral position, over several examinations, revealed consistent right extorsion (5°–9°) and consistent left intorsion (5°–10°). Retinal examination consistently showed moderate intorsion of the left eye and marked extorsion of the right eye. When her head was held straight, she tilted objects to the right when attempting to orient them vertically. When she tilted her head to the right, however, she was able to position them correctly.

We designed our surgical approach to rotate the eyes in the direction of the tilt. Under general anesthesia, the 12 o'clock and 6 o'clock positions were marked at the limbus of each eye with a skin marking pen.^{9,10} The right superior rectus muscle was then transposed nasally a full tendon width (10mm), the right inferior rectus muscle was transposed temporally a full tendon width (inducing further extorsion of the right eye). In addition, the left superior rectus muscle was transposed temporally a full tendon width and the left inferior rectus muscle was transposed nasally a full tendon width (inducing further intorsion of the left eye). Prior to reversing general anesthesia, the positions of the limbal markings were noted to be extorted in the right eye (approximately 15°) and intorted in the left eye (approximately 15°), confirming that the transposition of the vertical rectus muscles had achieved the planned torsional augmentation. The following day, the patient had 3^Δ of right hyperphoria. She was no longer leaning to the right and no longer had a head tilt. Seven weeks postoperatively, she maintained her improved body position and head tilt, commenting that she "didn't feel pulled anymore." She had full ductions and versions, 5^Δ exophoria in distance straight ahead, 8^Δ exophoria at near, with no measurable vertical phoria in any position of gaze. Double Maddox rod testing revealed 6° of subjective extorsion in the right eye and 6° of subjective intorsion in the left eye.

One year following surgery the patient had maintained her improvement and was able to sit straight in a chair (Figure 2B), and only has a slight body tilt when walking, she specifically commented that she did not "run into walls" as she had done before surgery. Her head tilt had improved from 45° to 5° (Figure 2).

Discussion

When the superior and inferior rectus muscles are horizontally transposed in opposite directions from each other, orbital elastic forces allow them to resume their normal positions within the orbit. As they do so, they torsionally rotate the globes in the direction opposite to the original transposition, for example, nasally transposing the superior rectus and temporally transposing the inferior rectus induces an extorsion of the globe (Figure 1).

Vertical rectus muscle transposition has been used to treat other forms of torticollis, such as idiopathic head tilt and the rare head tilt associated with congenital nystagmus.¹¹ Proponents of this procedure,^{11,12} believe that torsional rotation of the eyes in the direction of the head tilt serves to induce a contraversive tilt of the visual world, which stimulates the patient to straighten the head to move it into closer alignment with the tilted visual world. Because previous patients with torticollis have not reported induced visual tilt after successful treatment with vertical rectus transpositions, we suggest an alternative mechanism that we now propose for our case of lateropulsion. We suggest that procedures to induce or augment binocular torsion may serve to realign the vertical meridians of the eyes with a tilted subjective visual vertical, which counter-rotates the tilted subjective vertically back toward the gravitational vertical, thereby obviating the need for the head tilt.

Our patient had an incomplete ocular tilt reaction associated with lateropulsion, a tilted subjective vertical, disconjugate torsion (greater in the extorted eye), and a postural tilt that was directed toward the subjective visual vertical. Although patients with this condition may not perceive the visual world as tilted, they consistently assign a tilted orientation when trying to orient objects vertically. Brandt and Dieterich¹³ have found uniocular torsion with subjective visual tilt to be diagnostic of unilateral brainstem injury. Binocular torsion has not been found to accompany hemispheric lesions that cause lateropulsion.¹⁴

In our patient, torsional augmentation of the eyes produced resolution of lateropulsion and almost complete normalization of head and body position, restoring her ability to correctly

assign vertical orientation to objects in the visual world. When planning this procedure, it is important to rule out coexisting oblique muscle palsies, which can simulate the ocular tilt reaction. The diagnosis of an incomplete ocular tilt reaction was confirmed by demonstrating intorsion of the higher eye (in contradistinction to the extorsion of the higher eye that is seen in patients with superior oblique palsy).¹⁵

While it could be argued that all components of the ocular tilt reaction (including the subjective visual tilt) are direct effects of neurological defects, Dieterich and Brandt⁸ postulated that the motor abnormalities are not neurological defects but compensatory rotations for a tilt in the subjective visual vertical caused by a unilateral lesion of central otolithic pathways. In our case, if the binocular torsion had caused the subjective visual tilt, one would expect the subjective visual tilt to have increased when the objective binocular torsion was surgically increased. Our observation that the subjective visual tilt and postural tilt resolved following surgery to increase the binocular torsion thus reinforces the notion that the postural tilt is compensatory for an underlying subjective visual tilt, providing the driving force for each component of the ocular tilt reaction.

Based on the dramatic improvement in our patient when the objective binocular torsion was surgically increased, we postulate that surgery to reduce the objective preoperative binocular torsion would have the potential to make the head tilt worse. This paradoxical response to generating ocular torsion provides a cautionary note that strabismus surgery cannot always be designed to “fix the deviation,” particularly when its cause remains ill defined. It is therefore essential to understand the driving force behind a deviation when designing optimal treatment.

We conclude that horizontal transposition of the vertical rectus muscles to increase preexisting binocular torsion may provide a simple treatment for the torticollis, lateropulsion, and other disabling symptoms of tilt that may accompany the ocular tilt reaction.

Acknowledgments

Supported by National Institutes of Health Grant EY018810 (JMH), Research to Prevent Blindness, New York, NY (JMH as Olga Keith Weiss Scholar and an unrestricted grant to the Department of Ophthalmology, Mayo Clinic), and Mayo Foundation, Rochester, MN.

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FIG 1.

Therapeutic effect of torsional augmentation. In the ocular tilt reaction, lesioning the right utricular pathways causes the subjective visual vertical to be tilted to the right. A, A compensatory oculocephalic synkinesis rotates the eyes and head to a position that the central nervous system erroneously interprets as is tilted to the right, resulting in torsional rotation of the superior aspect of both globes to the right and skew deviation. *GV*, gravitational vertical; *HV*, head vertical; *LP*, Lower pole of eyes; *SVV*, subjective visual vertical; *UP*, upper pole of eyes. B, Following horizontal transposition of the vertical rectus muscles, the transposed vertical rectus muscles rotate the eyes torsionally as connective tissue attachments within the orbit restore them to their previous positions. This torsional augmentation serves to more closely realign their vertical poles with the tilted subjective visual vertical. In so doing, it counterrotates the tilted subjective visual vertical back toward the gravitational vertical (curved arrow), thereby reducing the need for a compensatory head tilt.

FIG 2.
A, Preoperative head tilt and body tilt characteristic of lateropulsion. B, Postoperative head and body position, showing improvement of from approximately 45° to 5°.