Vergence-Mediated Changes in Listing's Plane Do Not Occur in an Eye with Superior Oblique Palsy

Americo A. Migliaccio, Phillip D. Cremer, Swee T. Aw, and G. Michael Halmagyi

Purpose. As a normal subject looks from far to near, Listing's plane rotates temporally in each eye. Since Listing's plane relates to the control of torsional eye position, mostly by the oblique eye muscles, the current study was conducted to test the hypothesis that a patient with isolated superior oblique palsy would have a problem controlling Listing's plane.

Method. Using the three-dimensional scleral search coil technique, binocular Listing's plane was measured in four patients with congenital and in four patients with acquired unilateral superior oblique palsy during far- (94 cm) and near- (15 cm) viewing. The results were compared to previously published Listing's plane data collected under exactly the same conditions from 10 normal subjects.

Results. In patients with unilateral superior oblique palsy, either congenital or acquired, Listing's plane in the normal eye rotated temporally on near-viewing, as in normal subjects, while in the paretic eye it failed to do so. In patients with acquired superior oblique palsy, Listing's plane was already rotated temporally during far-viewing and failed to rotate any farther on near-viewing, whereas in patients with congenital superior oblique palsy Listing's plane in the paretic eye was oriented normally during far-viewing and failed to rotate any farther on near-viewing.

Conclusions. These results suggest that the superior oblique muscle, at least in part, is responsible for the temporal rotation of Listing's plane that occurs in normal subjects on convergence. (*Invest Ophthalmol Vis Sci.* 2004;45:3043–3047) DOI:10.1167/iovs.04-0014

Although the eye can rotate with three degrees of freedom, during visual fixation, smooth pursuit, and saccades, it exercises only two: horizontal and vertical. Furthermore, when the head is not moving and there is no vestibular input, horizontal and vertical eye-in-head position (gaze position) determines how much the eye has rotated about its line of sight (i.e., the amount of torsion). This relationship between torsional eye position and gaze position is described by Listing's law. During visual fixation, smooth pursuit,[1] and saccades,[2] Listing's law correctly predicts that the tips of the rotation vectors used to describe eye positions all lie in a plane called the displacement plane.[3] The displacement plane is determined by Listing's plane (LP), which is head fixed and changes orientation under few conditions. For example, LP changes orientation during prolonged fusion of an imposed vertical disparity[4] and during prismatically induced horizontal and vertical vergence.[5] In this study, however, we examined changes in LP that occur during near-viewing (i.e., during vergence). As a fixation target is brought from far to near, the vergence angle (the angle between the lines of sight of the two eyes) increases, and LP rotates temporally, equally in each eye, by an amount proportional to the increase in vergence angle. This occurs even if the target is directly in front of one eye, so that the position of one eye does not change as the target nears the subject (asymmetrical vergence). The resultant increase in vergence angle nonetheless rotates LP in each eye by an equal amount.[6–9] LP rotates in each eye around a point that is not at the origin of the coordinate system describing eye position. Consequently, it is only during downward gaze that torsional eye position changes significantly on near-viewing.

Temporal rotation of LP on near-viewing approximately aligns the three-dimensional eye rotation axes during saccades and, as a consequence, eye eccentricity is minimized.[10] The mathematical complexity of this task suggests that LP orientation is centrally optimized and implemented peripherally using all six extraocular muscles. In support of this hypothesis is the apparent plasticity of LP after strabismus surgery.[11] However, another line of evidence suggests that the vergence-mediated change in LP may be due to relaxation of one extraocular muscle, the superior oblique.

Eye torsion is produced mainly by the oblique eye muscles.[12] The superior oblique produces intorsion and the inferior oblique produces extorsion. The oblique muscles are most likely responsible for the torsion reflected by the temporal rotation of LP that occurs on near-viewing. Because the most obvious change in LP is in downward gaze, the superior oblique muscle could be particularly crucial in the control of LP.[13] A recent study in patients with acquired superior oblique palsy (SOP) showed that LP was rotated temporally in the paretic eye during far-viewing[14] by an amount close to that measured in normal subjects during near-viewing. LP normally rotates 6° to 12° when the vergence angle changes by 25°.[6,8,9] If the superior oblique were responsible for the change in LP then one could predict that in an eye with a SOP, LP would not change between far and near-viewing.

There could be some structural differences between congenital and acquired SOPs. One study reported imaging of abnormalities of the superior oblique tendon in congenital SOP in contrast to atrophy of the superior oblique muscle in acquired SOP,[15] but this result was not replicated.[16] In ~5% of patients with congenital SOP the superior oblique muscle is missing.[17] In general, SOP does not cause the inferior oblique muscle to atrophy or lose contractility.[18] However, irrespective of these observations, one could predict that patients with congenital SOP would show better adaptation than those with acquired SOP. LP in patients with congenital SOP could be closer to normal during far-viewing, which is the usual viewing condition.

From the Neurology Department, Royal Prince Alfred Hospital, Sydney, Australia.

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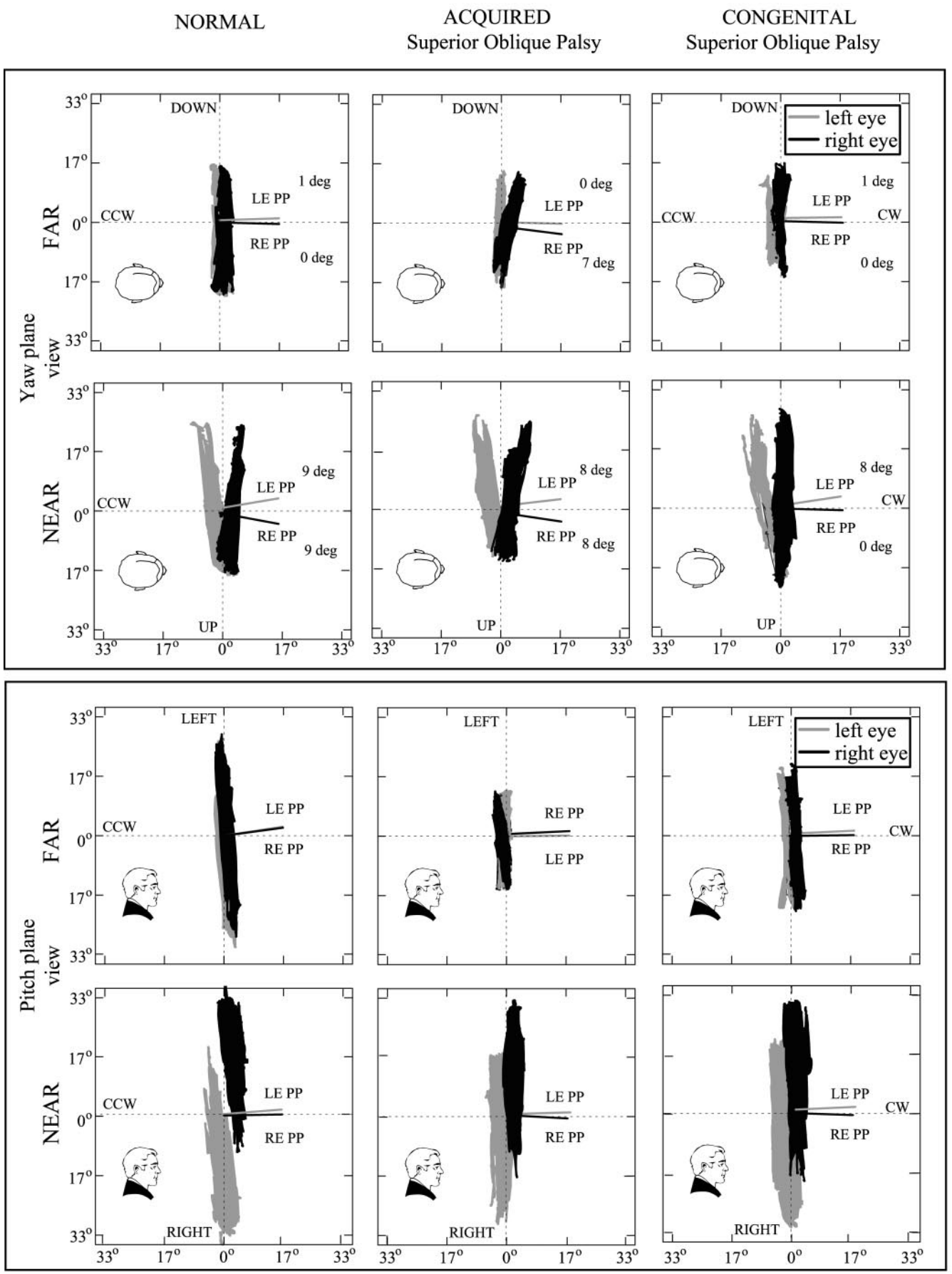
Corresponding author: G. Michael Halmagyi, RPA Hospital, Camperdown, NSW 2050, Sydney, Australia; michael@icn.usyd.edu.au.



METHODS

Patients

We studied eight patients with isolated unilateral SOP diagnosed at the Neuro-ophthalmology Clinic of Royal Prince Alfred Hospital or the Sydney Eye Hospital. Each patient was referred by an ophthalmologist specializing in strabismus surgery, and each fulfilled the standard clinical criteria for SOP as set out by Parks.[19] In brief, each patient had a vertical-torsional diplopia with excyclotorsion of the hypertropic eye. The hypertropia was greater in adduction than in abduction and on head tilt toward the side of the hypertropic eye. Four of the patients were considered to have acquired SOP (mean age, 40 years; range, 35–68) on the following evidence: (1) diplopia for less than 3 months, (2) no head tilt on old photographs, (3) vertical fusional amplitude of 3 D or less, and (4) spontaneous resolution of hypertropia on examination 6 months after eye-movement recordings. Four of the patients were considered to have congenital SOP (mean age, 29 years; range, 15–58) on the following evidence: (1) diplopia for more than 1 year, (2) long-standing head tilt away from the side of the SOP on old photographs, (3) a vertical fusional range of 6 D or more, and (4) overaction of the inferior oblique so that the hypertropia on adduction was the same (two patients) or greater, (two patients) in elevation than on depression.

Written and informed consent was obtained from all patients before testing, according to the Declaration of Helsinki. The experimental protocol was approved by the Human Ethics Committees of the Central Sydney Area Health Service the South Eastern Sydney Area Health Service, and the University of New South Wales.

Recording System

The scleral search coil recording system and methods have been described in detail.[8] Three-dimensional head and eye position was measured using the scleral search technique developed by Robinson[20] and Collewijn et al.[21] with 1.88-m^3 magnetic field coils. The voltage signals corresponding to the positions of the head and eye coils were preamplified and passed through phase detectors. These signals were passed through anti-aliasing filters with cutoff at 100 Hz. The nine position signals were sampled at 1 kHz with 16-bit resolution by a computer running commercial software (LabVIEW, ver. 5.0 and I/O card; National Instruments, Austin, TX). The resolution of the coils systems was 0.2° (tested over the angular range of ±25° combined yaw, pitch, and roll positions) the differentiated signal noise was approximately 2.5 deg/s.

Both the head and eye coils were calibrated in vitro using a Fick gimbal in 5° steps over a range of ±20° in the yaw, pitch, and roll axes. To correct for nonorthogonality between the direction and torsion coils, we used an algorithm developed by Bruno and Van den Berg.[22] To correct for search coil misalignment with the eye, each patient was instructed to fixate on a laser dot 94 cm directly in front each eye. This misalignment was corrected using an algorithm developed by Tweed et al.,[23] which calculated the instantaneous rotation of the coil with reference to the coil's orientation when the eye was looking straight ahead. All the voltage data were converted into Fick angles using the offset and gain values determined during the in vitro calibration. The resultant Fick angles, describing the three-dimensional orientation in space of each eye and of the head, were converted to rotation vectors with roll, pitch, and yaw coordinates.[24]

Experimental Protocol

Dual search coils were placed on each eye of each patient after application of topical anesthesia (Alcaine; Alcon, NSW, Australia). The head coil was positioned on a mounting frame that was glued to a pair of lightweight spectacle frames. The spectacle frame was fastened securely to the patient's head with a Velcro strap without any adjustment during the experiment. The subject's head was held firm using a wooden head holder. The head holder was adjusted until the patient's mid interpupillary axis was positioned in the center of the magnetic field coils. The subjects viewed a target screen placed at either 94 or 15 cm from the mid interpupillary point. The displacement plane was measured as the patient fixated targets sequentially for 60 seconds on points located on a flat screen 94 cm and also 15 cm from the patient.[25] The far screen and the near screen each covered a vertical and horizontal visual range of −25° to 25° for each eye. The far screen was 90 × 90 cm and contained 144 dots separated by 7.5 cm; similarly, the near screen was 24 × 24 cm and contained 144 dots separated by 2 cm. If the patient could not fixate on the target screen, the data were excluded from analysis, and the measurement was repeated. To confirm that each patient was maintaining fixation on the near target screen, we used the vergence angles measured and the average measured interpupillary distance of 6.4 ± 0.3 cm to calculate the target screen distance of 15.8 ± 2.7 cm (mean across patients).

LP was calculated from the displacement plane, which is defined as the plane to which the static rotation vectors describing eye position are confined when they are calculated relative to an arbitrary reference eye position. In the special case where the reference gaze direction is perpendicular to the displacement plane, the reference position is Listing's primary position (LPP), and the displacement plane is LP.

Data Analysis

Each LP was calculated from 120 targets during a 60-second period. A best fit for LP was determined using a singular value decomposition algorithm[26]:

$$\boldsymbol{r}_x = \boldsymbol{f} + \boldsymbol{f}_v \boldsymbol{r}_y + \boldsymbol{f}_h \boldsymbol{r}_z$$

where $\boldsymbol{r}_x$, $\boldsymbol{r}_y$, and $\boldsymbol{r}_z$ are the components of the rotation vector representing the torsional, vertical, and horizontal components of the rotation and $\boldsymbol{f}$, $\boldsymbol{f}_v$, and $\boldsymbol{f}_h$ are coefficients. The *x*-axis is naso-occipital (forward, positive), the *y*-axis is interaural (left, positive) and the *z*-axis is rostrocaudal (up, positive). LPP was calculated to show the orientation of LP.[27]

RESULTS

The displacement plane was plotted for target screen distances, 94 and 15 cm. The yaw- and pitch plane-representations of approximately 120 random eye positions including LPP in a normal subject, a patient with acquired right eye SOP, and a patient with congenital right eye SOP are shown in Figure 1. The yaw-plane LP view during far- and near-viewing are shown in rows 1 and 2, respectively. LP in the normal (left) eye rotated temporally by approximately one third of the increase in vergence angle. In contrast, LP in the paretic (right) eye did not change with vergence angle. The pitch-plane LP view during far- and near-viewing are shown in rows 3 and 4, respectively. During near-viewing the range of horizontal eye movement were asymmetric, resulting in an apparent offset (in the opposite direction, along the rostrocaudal axis) between right and left eye LP. This asymmetry occurs during near-viewing because the adducting eye is more eccentric in position than the abducting eye. Once the adducting eye reaches

FIGURE 1. Displacement plane and LPP while viewing targets at 94 cm (FAR) and then at 15 cm (NEAR). Between far- and near-viewing conditions, LPP rotated temporally by approximately 8° in each eye of the normal subject (*top left*) and in the normal left eye of subjects with acquired or congenital SOP (*top row*; *middle* and *right* columns, respectively). In contrast, LPP was fixed in the paretic right eye of patients with SOP. In the patient with congenital SOP, it failed to rotate on near-viewing, whereas in the patient with acquired SOP it had already rotated 7° temporally during far-viewing and failed to rotate any farther during near-viewing.

TABLE 1. Listing's Primary Position in Four Acquired and Four Congenital Cases of Superior Oblique Palsy

	Normal (n = 10)		Acquired SOP (n = 4)		Congenital SOP (n = 4)	
LPP	Left Eye	Right Eye	Paretic Eye	Nonparetic Eye	Paretic Eye	Nonparetic Eye
Far (94 cm)	$X = 0.0 \pm 0.1°$	$X = 0.2 \pm 5.0°$	$X = 1.0 \pm 0.7°$	$X = 0.1 \pm 0.5°$	$X = 0.4 \pm 0.9°$	$X = 0.8 \pm 0.8°$
	$Y = -2.3 \pm 3.4°$	$Y = -0.7 \pm 3.3°$	$Y = 3.3 \pm 3.4°$	$Y = 1.1 \pm 2.4°$	$Y = 2.4 \pm 3.1°$	$Y = 0.3 \pm 1.5°$
	$Z = 4.4 \pm 4.7°$	$Z = -2.7 \pm 4.1°$	$Z = 6.0 \pm 4.0°$	$Z = 0.2 \pm 3.6°$	$Z = 0.1 \pm 2.2°$	$Z = 0.1 \pm 3.5°$
Near (15 cm)	$X = -1.4 \pm 1.5°$	$X = 1.4 \pm 4.8°$	$X = 1.4 \pm 1.3°$	$X = 1.4 \pm 1.1°$	$X = 0.6 \pm 2.7°$	$X = 1.2 \pm 1.2°$
	$Y = 1.5 \pm 5.5°$	$Y = 6.2 \pm 6.2°$	$Y = 2.3 \pm 2.4°$	$Y = 1.3 \pm 4.1°$	$Y = 3.3 \pm 3.7°$	$Y = 2.3 \pm 3.6°$
	$Z = 12.1 \pm 4.3°$	$Z = -10.2 \pm 4.7°$	$Z = 6.4 \pm 3.5°$	$Z = 7.1 \pm 4.5°$	$Z = 0.5 \pm 2.5°$	$Z = 7.4 \pm 4.2°$
Paired difference	$X = -1.4 \pm 2.9°$	$X = 1.2 \pm 5.1°$	$X = 0.4 \pm 0.6°$	$X = 1.3 \pm 0.9°$	$X = 0.2 \pm 1.1°$	$X = 0.4 \pm 1.3°$
	$Y = 3.8 \pm 3.5°$	$Y = 6.9 \pm 4.3°$	$Y = -1.0 \pm 4.4°$	$Y = 0.2 \pm 4.4°$	$Y = 0.9 \pm 5.1°$	$Y = 2.0 \pm 3.5°$
	$Z = 7.7 \pm 1.9°$	$Z = -7.5 \pm 1.6°$	$Z = 0.4 \pm 1.2°$	$Z = 6.9 \pm 2.7°$	$Z = 0.4 \pm 0.9°$	$Z = 7.3 \pm 2.3°$

The previously published normal data was collected under the exact same conditions as the SOP patients.

the limits of its oculomotor range the abducting eye, although less eccentric, is also limited, to maintain binocular vision.

In both acquired and congenital SOP, LPP in the normal eye changed significantly between far- and near-viewing conditions (paired t-test, $P < 0.05$), whereas LPP in the paretic eye did not change between viewing conditions (paired t-test: acquired SOP, $P = 0.89$; congenital SOP, $P = 0.86$; Table 1).

As the average vergence angle increased from 3.8 ± 0.2° during far-viewing to 23.9 ± 3.0° during near-viewing, LPP rotated temporally in the normal eye by an average 7.1 ± 2.5°, a value similar to that in normal subjects.[8] In contrast, we found that LPP for the paretic eye was rotated temporally during both far- and near-viewing by only 0.3 ± 2.3° in the four patients with congenital SOP, whereas it was rotated by 6.2 ± 3.7° in the four patients with acquired SOP.

The fitted displacement planes had an average SD of 0.6°, and in patients in whom the displacement plane was measured more than once, the average day-to-day variability of LPP was slight (within ±0.3°, ±1.0°, and ±1.7°).

DISCUSSION

We found that in all patients with SOP, LP in the normal eye rotated temporally on near-viewing, as in normal subjects, but was fixed in the paretic eye. In congenital SOP it was fixed in the normal far-viewing orientation, whereas in acquired SOP, it was fixed in the normal near-viewing orientation. These findings agree with the results of a previous study that measured LP only during far-viewing and which showed that, in an eye with acquired SOP, LP was rotated temporally, whereas in an eye with congenital SOP, it was the same as in the normal eye.[14] We believe this difference in LP between congenital and acquired SOP occurs because patients with congenital SOP adapt better to the superior oblique muscle weakness. This hypothesis is supported by a previous study showing that during saccades, Listing's law is violated in an eye with acute SOP, whereas it is obeyed in an eye with chronic SOP.[28] The authors of that study concluded that neural adaptation could restore Listing's law by adjusting the innervations to the remaining extraocular muscles, even when one eye muscle remains paretic.

LP for the paretic eye did not change between far- and near-viewing in any of our eight patients with SOP, suggesting that the superior oblique muscle is responsible for the normal temporal rotation of LP during convergence. A previous study in monkeys provides direct support for this hypothesis. Mays et al.[29] showed that trochlear unit activity decreases during convergence. Furthermore, the magnitude of the decrease varies systematically with vertical eye position and is greater during downward gaze. Increased tension of the superior oblique for downward gaze directions requires a greater amount of relaxation of the muscle to assist with adduction required during convergence. That study showed that excyclotorsion increased with downward gaze, consistent with our findings in humans. To simulate the effects of the superior oblique muscle on LP, we used a software package that models the eye mechanically (Orbit 1.6; Eidactics, San Francisco, CA).[30] During convergence there is no contractile thickening of the superior oblique,[31] hence we simulated near-viewing by decreasing the superior oblique contractile muscle strength as a percentage of its normal value. The simulated eye was rotated in steps of 10° from −20° to +20° in all combinations of yaw and pitch (25 gaze directions). LP was determined by plotting the torsional position of the simulated eye for each gaze direction. The resultant deviations from normal LP are shown in Figure 2. The model results were similar to our normal eye data suggesting that the superior oblique muscle is modulated by vergence.

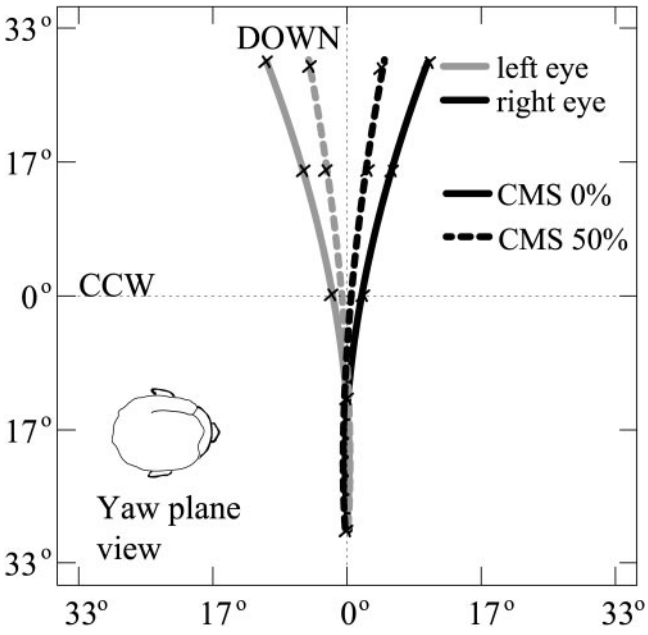


FIGURE 2. Deviations from LP due to superior oblique weakness. SOP in each eye was simulated by altering the contractile muscle strength (CMS) of the superior oblique. When the CMS was decreased to 0% of normal, LP was similar to that measured in the paretic eye of patients with acquired SOP during near- (15 cm) and far- (94 cm) viewing.

Our data suggest that the superior oblique muscle is necessary to rotate LP during near-viewing and implies that if SOP is present in early life, adaptive processes optimize the orientation of LP for far-viewing, whereas if SOP develops later in life, there is no such adaptation.

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