MICELLANEOUS

Torsion in patients with superior oblique palsies: dynamic torsion during saccades and changes in Listing's plane

Heimo Steffen · Dominik S. Straumann ·
Mark F. Walker · Neil R. Miller · David L. Guyton ·
Michael X. Repka · David S. Zee

Abstract

Background The purpose was to assess intra- and post-saccadic torsion in superior oblique palsy (SOP) patients and the effect of surgery on torsion.

Methods Eleven patients with a presumed congenital SOP and five with acquired SOP performed 10° vertical saccades over a range of ±20°. Eye movements were recorded with dual search coils. Dynamic torsion was calculated by subtracting the expected change in torsion during the saccade (based upon static torsion before and after the saccade) from the maximum intrasaccadic torsion.

H. Steffen · D. S. Straumann · M. F. Walker · D. S. Zee
Department of Neurology, The Johns Hopkins Hospital,
601 North Caroline Street,
Baltimore, MD 21287, USA

M. F. Walker · N. R. Miller · D. L. Guyton · M. X. Repka ·
D. S. Zee
Department of Ophthalmology, The Johns Hopkins Hospital,
601 North Caroline Street,
Baltimore, MD 21287, USA

D. S. Straumann
Department of Neurology, University Hospital,
Rämistrasse 100,
8091 Zürich, Switzerland

H. Steffen (✉)
Department of Ophthalmology, University of Würzburg,
Josef-Schneider-Str.11,
97080 Würzburg, Germany
e-mail: h.steffen@augenklinik.uni-wuerzburg.de

Eight healthy subjects were controls. We also examined the effects of surgery on dynamic torsion and the orientation of Listing's plane in patients with congenital SOP who were operated on either by weakening of the inferior oblique muscle on the affected eye (n=5), by recession of the inferior rectus muscle on the normal eye (n=4) or by both procedures (n=2). Postoperative recordings were obtained at least 1 month after surgery.

Results Patients with congenital and acquired SOP showed an increased dynamic extorsion, primarily during downward saccades. Following a recession of the inferior oblique muscle in congenital SOP patients, half showed significant decreases in extorsion (up to 1.0°) during downward saccades by the affected eye. Following surgery all showed a temporal rotation of Listing's plane (up to 15° for primary position).

Conclusion Patients with a SOP show a characteristic pattern of dynamic torsion during vertical saccades differing from normals. Recession of the inferior oblique muscle leads to rotation of Listing's plane in all congenital SOP patients and causes large changes in dynamic torsion in a subgroup of them, perhaps reflecting the heterogeneity of congenital SOP.

Keywords Dynamic torsion · Superior oblique palsy · Listing's plane

Introduction

One of the most prominent symptoms in patients with superior oblique palsy (SOP) is vertical diplopia. Patients with an acquired SOP often also complain of torsional diplopia or tilting of images [1]. In the latter group symptoms are usually due to a weakness of the superior

oblique muscle resulting from damage to the trochlear nerve. In contrast, patients with a congenital SOP are a heterogeneous group. Only when orbital imaging shows a large difference between the cross-sectional areas of the superior oblique muscles is a muscle paralysis likely, though the cause is not necessarily neurogenic. For example, abnormalities of the tendon of the superior oblique muscle [2, 3], or of orbital pulleys [4] may produce a vertical strabismus with a pattern of misalignment similar to that of congenital SOP. Changes in torsional alignment are a well-known feature in patients with SOP-palsy [1] and changes in the primary position of the eye as defined by Listing's law have also been reported in the same group of patients [5–7]. Dynamic violations of Listing's law, as reflected in an increased thickness of Listing's plane, were reported in two patients with an acquired SOP of short (less than 4 weeks) duration [7].

The first aim of this study was to analyze changes in torsion *during* saccades in patients with presumed congenital or acquired SOP. We sought to know if patients with a SOP (either congenital or acquired) have dynamic changes in eye torsion during a saccade, so-called dynamic torsion or torsional 'blips.' Dynamic torsion is defined as the amount of torsion during a saccade that is over and above that predicted from the static changes associated with a change in eye position [9]. Our second aim was to investigate the effect of corrective eye-muscle surgery on dynamic torsion and upon the orientation of Listing's plane.

Methods

Subjects

We examined 11 patients with a presumed congenital superior oblique palsy and 5 patients with an acquired superior oblique palsy (see Table 1). Postoperative recordings were performed at least 1 month after eye muscle surgery.

The patterns of static vertical misalignment in these patients and the criteria for dividing them into congenital and acquired groups have been reported previously [5]. The diagnosis of congenital versus acquired was based on clinical information and the medical history. Orbital imaging was not available for this group of patients. All patients provided informed written consent according to a protocol conforming to the Declaration of Helsinki and approved by the Johns Hopkins Joint Committee on Clinical Investigation. In the congenital group, all patients were operated on, either with a weakening procedure of the inferior oblique muscle of the affected eye (n=5), a recession of the inferior rectus muscle of the normal eye (n=4) or both procedures (n=2). We had data from only one patient with acquired SOP who was operated upon, so results of surgery in acquired SOP will not be considered further. Recordings from the right eye of eight healthy subjects (mean age 37±10 years, range 22–58 years) served as controls.

Table 1 Clinical characteristics of patients

Patient no.	Age at recording	Clinical diagnosis	Vertical phoria (°)	Vertical fusional amplitude (°)	Eye operated*
1	57	Cong.	8.2	1.3	Paretic eye
2	54	Cong.	5.2	3.7	Paretic eye
3	17	Cong.	1.3	0.1	Non-paretic eye
4	55	Cong.	10.8	0.0	Non-paretic eye
5	47	Cong.	11.5	10.8	Paretic eye
6	41	Cong.	18.3	1.3	Paretic eye
7	53	Cong.	16.9	0.6	Both eyes
8	45	Cong.	2.0	4.0	Both eyes
9	36	Cong.	2.8	1.8	Paretic eye
10	58	Cong.	6.3	4.9	Paretic eye
11	38	Cong.	5.1	2.3	Non-paretic eye
12	53	Acq.	5.0	1.2	No post-op recording
13	52	Acq.	2.6	1.0	No post-op recording
14	54	Acq.	5.1	2.7	No post-op recording
15	15	Acq.	3.5	2.3	No post-op recording
16	59	Acq.	2.0	1.3	Spontaneous recovery

Cong., congenital; acq., acquired; *operation on the paretic eye was always an inferior oblique recession, operation on the non-paretic eye was always a recession of the inferior rectus muscle

Visual stimuli

Fixation points consisted of light-emitting diodes (LEDs) placed on a flat screen that was 124 cm in front of the eye of the subject. The LEDs were vertically separated by 10°. Subjects performed 10° vertical saccades in the midsagittal plane over a total range of 40°. The center LED (0,0), located straight ahead, served as the reference position.

Subjects made saccades to the targets on the LED array for five trials, starting at the center LED. The LEDs were consecutively lit for 1.5 s. The paradigm was performed under monocular viewing conditions with the affected eye viewing and the normal eye viewing. For the calculation of Listing's plane subjects viewed targets on a square ±20° centered around the LED at straight ahead.

Recording of eye movements and calibration procedure

Eye movements were recorded around all three axes of rotation (horizontal, vertical, and torsional) using the magnetic field search coil method with dual annuli. The annuli were placed on each eye after administration of a topical anesthetic [Proparacaine HCL 0.5%, (Alcaine ®)]. The subject's head (eyes) was precisely centered in the field coils using space-fixed horizontally and vertically oriented laser beams emanating from the location of the zero-position LED. The head was immobilized with a bite bar made of dental impression material.

The output signals were filtered with a bandwidth of 0–90 Hz and sampled at 500 Hz with 12-bit resolution. System noise was limited to 0.1°. Data were stored on a disc for later off-line analysis using Matlab (Mathworks, Inc). Further details of the calibration and recording procedures, as well as possible artifacts, can be found in the publications by Straumann et al. [5], Steffen et al. [8] and Bergamin et al. [10]. The positions of the eyes were calculated in rotation vectors and converted to degrees in a head coordinate system. For torsion, positive referred to extorsion and negative to intorsion. Primary position, as an index of the orientation of Listing's planes, was calculated as described previously [5, 6, 9]. The standard deviations of Listing's planes were calculated from the median values of torsion at straight ahead and the eight 20° eccentricities.

Data analysis

Identification of saccades

Each saccade was marked using a computer algorithm to identify the beginning of a saccade, when eye velocity reached 30°/s, and the end of a saccade, when eye velocity dropped below 30/s. Measures of the static alignment were based on the position of the mark at saccade onset The locations of the marks for each saccade were visualized by the experimenter and verified for correctness. Trials in which the subject did not gaze steadily at the target, saccades that were in the wrong direction or contaminated by eye closure were eliminated. This could amount to as much as about 30% of trials, but in most subjects the value was much less. Statistical analysis within subjects and among groups was based on our assumptions about whether or not we thought the data were distributed normally. If not we used nonparametric statistics. Pre- and post-surgery results within individuals were compared with paired 't' tests.

Calculation of dynamic torsion

Figure 1 demonstrates how the dynamic or 'blip' torsion was calculated for an individual vertical saccade. Figure 1 (top, right) shows the vertical eye position trace of the paretic eye for an upward saccade from 0° to up 10° in a patient with a congenital right SOP. The left dashed line represents the onset of the saccade and the right dashed line the end of the saccade. The middle trace demonstrates the associated torsion of the eye (positive is extorsion). Note that immediately before the saccade (left dashed line) there is relative extorsion of the right eye of about 0.2 and
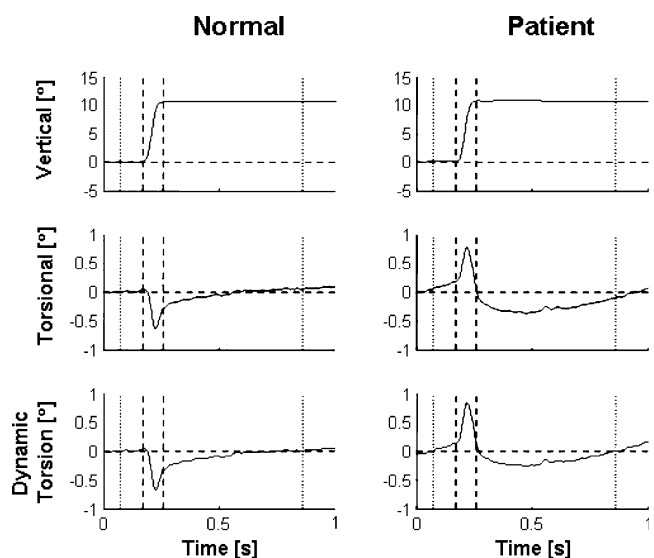
Fig. 1 Calculation of dynamic torsion. Patient on right, normal subject on left. The top trace, right trace shows the vertical position trace of the affected right eye of a patient with a congenital right superior oblique palsy for an upward saccade from 0° to up 10°. The left dashed line represents the onset of the saccade and the right dashed line the end of the vertical saccade. The middle trace shows the associated torsion of the eye (positive is extorsion). The bottom trace shows the dynamic torsion during this upward saccade. (See text for details of the calculation.) Dotted vertical lines indicate when eye torsion was assumed to be static before (left) and after (right) the saccade. Similar traces for the right eye are shown for a normal subject in the left panels

immediately after the saccade (right dashed line) relative intorsion of about -0.1°. This means that the static torsional orientation of the eye remained almost unchanged for this specific saccade.

In order to calculate the dynamic torsion for this individual saccade two eye positions were chosen, one 100 ms before the onset of the saccade and the other 600 ms after the end of the saccade (left and right dotted vertical lines). Then, assuming that at these two eye positions torsion was at a steady-state [9], a linear interpolation was made between these two points to predict what the expected static torsion would be at each point along the trajectory of the saccade, assuming that torsion changed linearly with eye position. The last step was to subtract this predicted static torsion (as determined from the linear interpolation) from the actual torsion measured during the saccade between the two dashed lines.

The bottom trace shows the result of this calculation which yields the dynamic torsion during this upward saccade. Note that dynamic torsion reaches its peak near the end of the saccade and then slowly returns to baseline following the saccade. A small amount of dynamic torsion and postsaccadic torsional drift can also be seen in normal subjects [9, 11].

Results

Preoperative data

Figure 2 shows the mean values of peak torsion of individual eyes for downward saccades in healthy subjects and in patients. Each data point represents the mean value of peak torsion based on all values from all the downward saccade trial types. There was considerable scatter amongst the congenital SOP group with about half of these patients overlapping with or even showing slightly higher values than most of the acquired SOP patients. Figure 3 shows the data from the same groups of patients for upward saccades. Again, there was also considerably more scatter within the congenital SOP patient group.

Statistical analysis of peak dynamic torsion was performed using a Kruskal-Wallis analysis of variance comparing the normal subjects, the affected eye of the SOP patients and the normal eye of the SOP patients. Because of the relatively small number of acquired SOP patients we combined all SOP patients together. For downward saccades, excluding one outlier with a very negative value from the congenital SOP group and which differed by more than 2.5 standard deviations from the mean, there was a statistically significant difference among the groups (p= 0.03) with the paretic eye being significantly different from the normal eye (multicompare, MATLAB, p<0.05). For
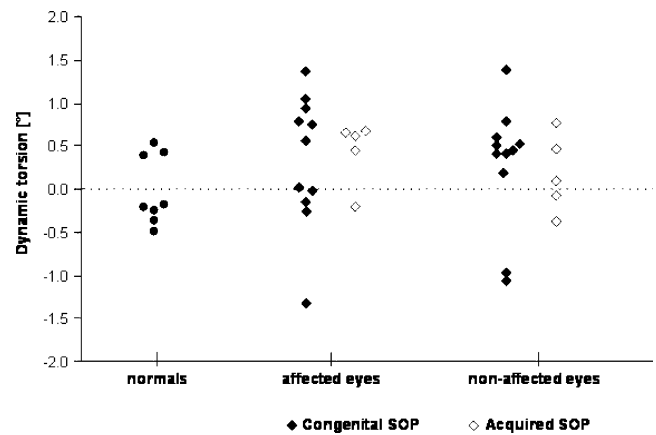
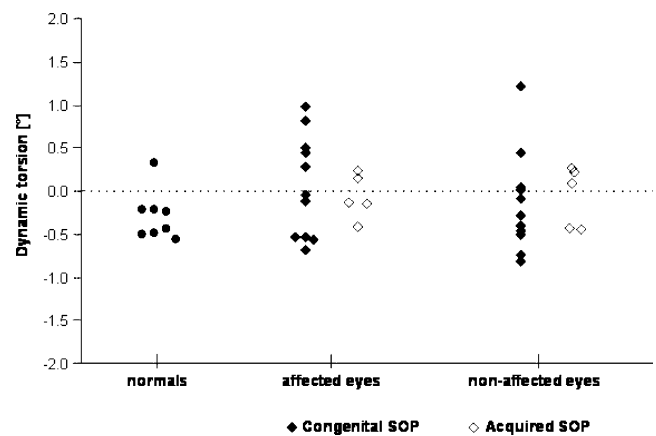**Fig. 2** Mean values of peak dynamic torsion of individual eyes for 10° downward saccades, from up 20° to down 20°, in normals and all patients. Each point represents the mean value for all downward saccades. A large value for peak dynamic torsion is prominent in the affected eyes of the patients with acquired SOP. Note also, however, that in the patients with congenital SOP a subgroup of patients appears to have high values for peak dynamic torsion. As a whole, changes in the normal eye of patients were not significantly different from normal subjects though there clearly is more scatter in the congenital SOP group. Positive is extorsion, negative intorsion of the eye

upward saccades there was no significant difference in peak dynamic torsion. The starting position of the eye had no significant influence on the amount of dynamic torsion.

Preoperative correlations of dynamic torsion

In the congenital SOP patients, who showed the wider range of values for peak dynamic torsion, we looked further for correlations between peak dynamic torsion and other parameters related to torsion or to vertical misalignment. For both the affected eye and the normal eye there was no correlation between peak dynamic torsion and the change

Fig. 3 Mean values of peak dynamic torsion of individual eyes for upward 10° saccades from down 20° to up 20° in normals and in patients. There is more scatter in the values for both the normal and the affected eye in the patients with congenital SOP. Positive is extorsion and negative intorsion
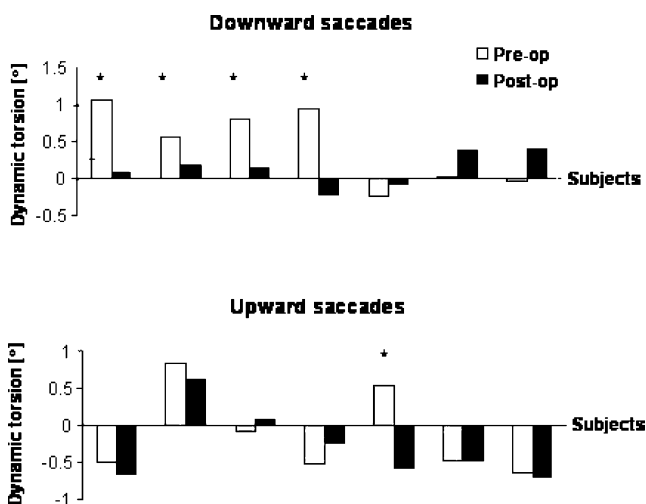
of static torsion between before and after the saccade. The horizontal and vertical components of primary position of the affected eye were also calculated. They did not correlate with the amount of peak dynamic torsion for upward or for downward saccades. Likewise, there was no correlation between the peak dynamic torsion and the vertical phoria, and no correlation between the peak dynamic torsion and the gradient (rate of change) of vertical phoria [5].

Postoperative data

We now consider the effect of eye muscle surgery on peak dynamic torsion of the affected and normal eye in congenital SOP.

Effects of the corrective surgery on peak dynamic torsion

Figure 4 compares pre- and post-operative peak dynamic torsion for the affected eye in the congenital SOP patients in whom the inferior oblique muscle was recessed. In four patients-those who had the larger amplitudes of preoperative dynamic torsion-there was a significant decrease (paired t test $p < 0.05$) in peak dynamic torsion for downward saccades (left four patients, Fig. 4 top panel). The mean preoperative dynamic torsion in these patients was $0.8° \pm 0.2°$; the postoperative dynamic torsion was $0.2° \pm 0.2°$. There were no significant changes, however, in the other patients who underwent this surgery. For upward saccades (Fig. 4, bottom panel) only one patient had a significant change in peak dynamic torsion following corrective surgery. In contrast, there were no consistent changes in dynamic torsion of the normal, operated eye in the patients who
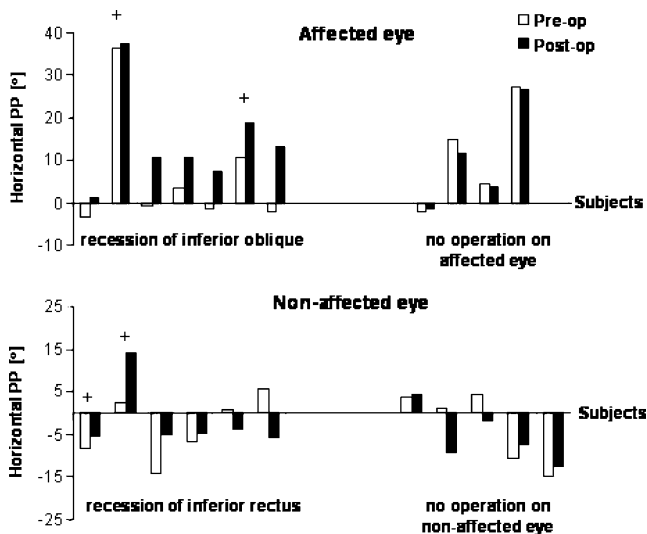
underwent recession of the inferior rectus of the eye contralateral to the affected eye. In those patients, however, the average peak dynamic torsion of the affected, non-operated eyes (n=4) increased by a small, but statistically significant amount ($0.2° \pm 0.08°$ SD, paired t test, p=0.01).

Effects of the corrective surgery on the orientation of Listing's plane

We also examined the effect of surgery on the orientation of Listing's plane by quantifying the pre- and postoperative location of Listing's primary position. The only significant change was in the group who had the recession of the inferior oblique muscle on the affected eye (Fig. 5, top panel, left seven patients). All subjects showed some degree of rotation of Listing's plane temporally as reflected in the more positive value of the horizontal component of primary position (paired t test, p=0.002). The average change in horizontal primary position induced by surgery was $7.9° \pm 4.9°$. In contrast there was no significant change in the vertical component of the primary position ($0.7° \pm 5.2°$). Note the '+' on the figure indicates the patients who had surgery on both eyes. Overall there was no significant change in the thickness of Listing's planes as reflected in the standard deviation of points from a plane fit to the nine median target positions at straight ahead and at the 20°

Fig. 4 Comparison of peak dynamic torsion in the affected eye in patients with congenital SOP, pre- and post-corrective surgery with a recession of the inferior oblique muscle of the paretic eye. Asterisks indicate a significant difference (p<0.05) between pre- and post-operative values

Fig. 5 Changes in Listing's primary position in the affected (top) and the normal (bottom) eye following corrective surgery. The most consistent change was in the patients who underwent a recession of the inferior oblique muscle in the affected eye (left seven patients). They showed a temporal displacement of Listing's plane of the affected eye as reflected in a more positive value of horizontal primary position. Patients who underwent a recession of the inferior rectus muscle in the normal eye (right four patients) showed no consistent change in Listing's primary position. 'Plus' signs indicate patients who had both a recession of the inferior oblique muscle in the affected eye and a recession of the inferior rectus muscle in the normal eye

eccentricities (0.86 preoperative, 0.94 postoperative, paretic eye).

Postoperative correlations of dynamic torsion

There was no correlation between the change of dynamic torsion and the change of vertical alignment in any of the subgroups (operation performed on the affected eye, operation performed on the normal eye). In the case of the patients who underwent recession of the IO muscle there was no correlation between the amount of change in dynamic torsion and the change in the location of Listing's primary position.

Discussion

Our results indicate that as a group, patients with a SOP show an abnormal pattern of transient (dynamic) torsion during vertical saccades. Overall there was a transient extorsion during downward saccades in the affected eye of many of the SOP patients. In addition, a weakening procedure of the ipsilateral inferior oblique muscle in four of seven patients with a presumed congenital SOP produced a relatively large and statistically significant decrease of the peak dynamic extorsion of the affected eye during downward saccades. All patients who underwent a recession of the inferior oblique muscle showed a temporal rotation of Listing's plane. On the other hand, in the patients who underwent a recession of the inferior rectus muscle in the unaffected eye there was no significant change in either the orientation of Listing's plane or the peak dynamic torsion of the operated eye. How do we interpret these patterns of dynamic torsion in patients with a SO palsy and the changes in dynamic torsion and the orientation of Listing's plane induced by corrective surgery?

Dynamic torsion during vertical saccades with SOP

In patients with a SOP, any dynamic extorsion during downward saccades made by the affected eye may result from a loss of the normal contribution of the superior oblique muscle to the torsional position of the eyes during the saccade. During a downward saccade, the antagonist muscles (the superior rectus and inferior oblique muscles) are largely inhibited [12]. Thus, with the loss of the activity of the paretic superior oblique muscle, which normally acts as an agonist during downward saccades, the eye might be expected to show increased extorsion during saccades. In other words, the extorting action of the inferior rectus muscle during downward saccades would no longer be balanced by the intorting action of the (paretic) superior oblique muscle. At the end of the saccade, however, the discharge rate of the inferior rectus muscle decreases (relative to its rate during the saccade) to the appropriate tonic level for the new downward eye position, and its extorting contribution is lessened. This would lead to slightly less static extorsion than the dynamic extorsion during the saccade. The superior rectus and inferior oblique muscles resume discharging at the end of the downward saccade at discharge rates appropriate to the new downward position, and their contributions to torsion thus are now presumably balanced [13]. The final torsional position of the eye is then reached by a slow drift following the end of the saccade reflecting the mechanical properties of the orbital tissues. Thus, in a SOP, one might expect increased static extorsion at the end of downward saccades from loss of the intorting action of the superior oblique muscle and an additional superimposed, dynamic extorsion during downward saccades.

One can apply similar logic to interpret what happens during upward saccades. At the end of the upward saccade the paretic superior oblique muscle no longer contributes its usual intorting action toward the static orientation of the eye, although its contribution in up gaze is less than with down gaze. Hence the degree of relative extorsion in up gaze is less than in down gaze. On the other hand, any dynamic torsion during the upward saccade should be much less affected by the absence of contraction of the superior oblique muscle because it is largely inhibited during upward saccades.

Most of the saccades made by our five patients with acquired SOP showed a pattern of dynamic torsion that was consistent with the above interpretation. In the patients with congenital SOP, however, only some of the patients behaved in this way. This difference likely relates to the heterogeneity of causes and the long-standing nature of the strabismus in patients with the diagnosis of congenital SOP. This issue is discussed further below.

Postoperative changes in dynamic torsion

One can consider the changes in dynamic torsion following corrective surgery using similar reasoning. A recession of the inferior oblique muscle would not be expected to affect directly the amount of torsion during downward saccades because the inferior oblique muscle is largely silenced during these movements. Likewise, during upward saccades, one would expect to see little change in dynamic torsion because with a recession, the inferior oblique muscle presumably still contracts during the upward saccade. In some of our patients, however, there were large and significant changes in dynamic torsion following inferior oblique surgery, especially for downward saccades. These particular patients had a considerably larger amount of preoperative dynamic extorsion than did the other patients. The reason for this pattern of change in dynamic

torsion after recession of the inferior oblique muscle is unclear. Firstly, as indicated above, the discharge activity of many of the motoneurons of the antagonist muscle during saccades does not completely cease throughout the entire saccade, so that there might indeed be some contribution of the inferior oblique muscle to torsion during downward saccades [12]. Secondly, it could be that the patients with the large change in dynamic torsion have a pattern of anatomical abnormalities producing their vertical strabismus that is more than just a weakness of the superior oblique muscle. Finally, it is also possible that following a recession, the inferior oblique muscle no longer generates the same pattern or timing of change in contractile force during saccades, either excitation as an agonist or inhibition as an antagonist, leading to a mismatch between the forces of the vertical recti and the inferior oblique muscles during vertical saccades. Certainly the completeness and degree of the recession of the inferior oblique muscle [14] and other mechanical factors related to the surgery on the inferior oblique muscle might also be playing a role, e.g., interruption or disruption of the ocular pulleys that determines the pulling directions of the ocular muscles [15–17]. In contrast, there were no consistent changes of dynamic torsion of the normal operated eye in the patients who underwent inferior rectus surgery although there was a small, unexplained, change in the affected non-operated eye.

The effects of corrective surgery on the orientation of Listing's plane

The effects of surgery on Listing's primary position were clear. All patients who underwent a recession of the inferior oblique muscle of the affected eye showed a temporal rotation of Listing's plane. At first glance, this seems paradoxical since an acquired SOP normally causes a temporal rotation of Listing's plane, and yet recession of the inferior oblique muscle is used to reverse both the vertical and the torsional misalignment created by an SOP. The inferior oblique muscle, however, has its strongest effect on torsion in up gaze. Thus, a recession of the inferior oblique muscle, while compensating for some of the static extorsion produced by the SO palsy in straight-ahead gaze, still produces a gradient of change in torsion such that the eye becomes even more intorted in up gaze relative to its torsional orientation in down gaze. Since it is the size of the vertical gradient of torsion that determines how far the horizontal component of primary position lies relative to the straight-ahead reference position [5], in SOP patients Listing's plane rotates even further in the temporal direction after IO recession. It is also worth reemphasizing that there was no relationship between the changes in intrasaccadic torsion during saccades and the changes in Listing's primary position during fixation following recession of the inferior oblique muscle.

Heterogeneity in congenital SOP

Because the number of patients was relatively small, especially in the acquired group, our results do not allow us to distinguish congenital from acquired SOP. Looking at the data, however, there was a trend for patients with acquired SOP to have larger amounts of peak dynamic torsion. There was also a suggestion of a separation of the congenital SOP patients into two groups, one of which had quite large amounts of peak dynamic torsion. This is not surprising considering the many possible causes of what appears to be a congenital SOP [2, 3, 17]. Clearly more patients with acquired SOP and more patients with congenital SOP who have MRI or surgical documentation of the cause must be studied carefully to confirm these suggestions.

One might also ask why our patients showed so much variability in their patterns of dynamic torsion and responses to surgery. Certainly, on the basis of clinical data alone we cannot classify patients definitively as having a congenital or an acquired SOP, and even in the so-called congenital SOP patients many types of anatomical or innervational abnormalities may cause patterns of vertical strabismus that are superficially similar [16–19]. Specifically the angle between the muscle plane and the sagittal line and the location and width of the insertion of both the superior and inferior oblique muscle may be considerably different among human subjects [19]. This anatomic variation may partially account for the different static deviations and the variability of the results of eye muscle surgery in SOP patients. Adaptive changes in eye alignment-both central (innervational) and peripheral (mechanical)-could also affect the patterns of torsion we observed, especially due to the long-standing nature of the strabismus in many of these patients. Furthermore, the inability to record eye movements immediately after corrective surgery and so document the immediate changes induced by the surgery before adaptation had a chance to intervene must be considered when interpreting patterns of torsion based upon mechanical considerations alone. Finally, we cannot as yet use the patterns of changes in dynamic torsion to assess the effectiveness of corrective surgery, since individual differences in the degree of static alignment and sensory and motor fusional capabilities are also likely important.

Even with these caveats, several novel and consistent patterns of change in torsion during saccades, both before and after corrective surgery, emerged in this study. These results can be compared with and serve as a guide for future clinical and experimental studies in which the anatomical abnormalities as a possible underlying cause for many cases of congenital superior oblique palsies and adaptive innervational changes are more clearly known.

References

1. Noorden von GK (1996) Binocular vision and ocular motility, 5th edn. St. Mosby, St. Louis pp 411–414
2. Helveston EM, Krach D, Plager DA, Ellis FD (1992) A new classification of superior oblique palsy based on congenital variations in the tendon. Ophthalmology 10:1609–1615
3. Plager DA (1992) Tendon laxity in superior oblique palsy. Ophthalmology 99:1032–1038
4. Clark RA, Miller JM, Demer JL (1998) Displacement of the medial rectus pulley in superior oblique palsy. Invest Ophthalmol Vis Sci 39:207–212
5. Straumann D, Steffen H, Landau K, Bergamin O, Mudgil AV, Walker MF, Guyton DL, Zee DS (2003) Primary position and listing's law in acquired and congenital trochlear nerve palsy. Invest Ophthalmol Vis Sci 44:4282–4292
6. Migliaccio AA, Cremer PD, Aw ST, Halmagyi GM (2004) Vergence-mediated changes in Listing's plane do not occur in an eye with superior oblique palsy. Invest Ophthalmol Vis Sci 45:3043–3047
7. Wong AM, Sharpe JA, Tweed D (2002) Adaptive neural mechanism for listing's law revealed in patients with fourth nerve palsy. Invest Ophthalmol Vis Sci 43:1796–1803
8. Steffen H, Walker MW, Zee DS (2000) Rotation of Listing's plane with convergence: independence from eye position. Invest Ophthalmol Vis Sci 41:715–721
9. Straumann D, Zee DS, Solomon D, Lasker AG, Roberts DC (1994). Transient torsion during and after saccades. Vision Res 35:3321–3334
10. Bergamin O, Ramat S, Straumann D, Zee DS (2004) Influence of orientation of exiting wire of search coil annulus on torsion following saccades. Invest Ophthalmol Vis Sci 45:131–137
11. Lee C, Zee DS, Straumann D (2000) Saccades from torsional offset positions back to Listing planes. J Neurophysiol 83:3241–3253
12. Robinson DA (1970) Oculomotor unit behaviour in the monkey. J Neurophysiol 33:393–404
13. Seidmann SH, Leigh JR, Tomsak RL, Grant MP, Dell' Osso LF (1995) Dynamic properties of the human ocular reflex during head rotations in roll. Vision Res 35:679–689
14. Yalcin B, Ozan H (2004) Insertion pattern of the inferior oblique muscle. Am J Ophthalmol 139:504–508
15. Demer JL (2006) Current concepts of mechanical and neural factors in ocular motility. Curr Opin Neurol 19:4–13
16. Demer JL, Oh SY, Clark RA, Poukens V (2003) Evidence for a pulley of the inferior oblique muscle. Invest Ophthalmol Vis Sci 44:3856–3865
17. Demer JL, Ortube MC, Engle EC, Thacker N (2006) High-resolution magnetic resonance imaging demonstrates abnormalities of motor nerves and extraocular muscles in patients with neuropathic strabismus. J AAPOS 10:135–142
18. Kono R, Demer JL (2003) Magnetic resonance imaging of the functional anatomy of the inferior oblique muscle in superior oblique palsy. Ophthalmology 110:1219–1229
19. Fink WH (1962) Surgery of the vertical muscles of the eye, 2nd edn. Thomas, Springfield pp.48–52

UniversitätsSpital
Zürich

**Neurologische
Klinik und Poliklinik**

Prof. Dr. med. R. Baumgartner
Abteilung Neuroangiologie
Neurologische Klinik und Poliklinik
UniversitätsSpital Zürich
Frauenklinikstr. 26
CH-8091 Zürich

sabine.zwahlen@usz.ch
Sucher   01 255 55 56
Sekretariat   01 255 55 56
Telefax   01 255 88 64

Zürich, 06.07.2007

**Bescheinigung über durchgeführte neurosonologische Untersuchungen
für den Fähigkeitsausweis «Zerebrovaskuläre Krankheiten (SGKN)»**

Hiermit bescheinige ich, dass Herr Dr. med. Hakan Sarikaya 6 Monate im Ultraschalllabor
der Neurologischen Klinik und Poliklinik des Universitätsspitals Zürich unter meiner
Leitung tätig war. Während dieser Zeit hat Herr Dr. Sarikaya über 500 Patienten klinisch
und mit extra- und intrakranieller Farbduplexsonographie selbstständig untersucht und
eine abschliessende Gesamtbeurteilung, die auch den Ultraschallbefund beinhaltet,
abgegeben. Er hat die oben genannten Untersuchungen gründlich und mit grosser
Kompetenz durchgeführt, sodass ich sein Gesuch zur Erlangung des Fähigkeitsausweis
"Zerebrovaskuläre Krankheiten" gerne unterstütze.

Mit freundlichen Grüssen

Prof. Dr. Ralf W. Baumgartner