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Location and Gaze-Dependent Shift of Inferior Oblique Muscle Position: Anatomic Contributors to Vertical Strabismus Following Lower Lid Blepharoplasty?

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Purpose. This study investigated, using high-resolution magnetic resonance imaging (MRI), inferior oblique muscle (IO) position relative to the adnexa in normal controls, subjects with and without vertical strabismus following lower lid blepharoplasty, and subjects with other hypertropia.

Methods. Sagittal plane MRI was obtained in central gaze, infraduction, and supraduction in 19 controls, 11 subjects with and 2 without hypertropia following bilateral lower lid blepharoplasty, and 13 subjects with hypertropia unrelated to blepharoplasty. In the plane passing through the center of the inferior rectus muscle (IR), we analyzed IO position relative to the globe, as well as the distance from IO to the skin or orbital floor.

Results. The IO was located approximately 1 mm more anteriorly and 1.2 mm more inferiorly in hypertropic than hypotropic fellow orbits of the blepharoplasty group and controls ($P < 0.05$). From central gaze to infraduction, IO shift in subjects with blepharoplasty was redirected inferiorly, rather than posteriorly as in all other groups. However, from central gaze to supraduction, IO motion was similar in all groups. There was scarring between the IO-IR pulley and orbital floor in the hypertropic eye after lower lid blepharoplasty.

Conclusions. Subjects with strabismus following lower lid blepharoplasty exhibit anterior and inferior IO pulley displacement in central gaze, as well as hindrance to normal posterior shift in infraduction. Proximity of IO to the orbital rim and lower eyelid skin is associated with strabismus following blepharoplasty, possibly because lower lid blepharoplasty may change lid forces on the IO-IR pulley system via scar tissue.

Keywords: blepharoplasty, hypertropia, magnetic resonance imaging

Lower lid blepharoplasty is widely performed aesthetic surgery that corrects involutional changes.[1] This surgery involves the region of the lower eyelid retractors, connective tissue bands extending from the region of the conjoined inferior rectus muscle (IR), and inferior oblique muscle (IO) pulleys. This region also includes Lockwood's ligament, a connective tissue structure that supports the globe. Complications of blepharoplasty have been reported, including diplopia due to strabismus.[2–8] While vertical strabismus is relatively infrequent, it is one of the most bothersome complications. The mechanism of this diplopia is not well understood.

The lower eyelid has an intimate anatomic relationship with the IO and orbital bones.[9,10] The IO pulley is partly coupled to the mobile IR pulley by elastic tissues. The lower eyelid normally moves in coordination with vertical eye position by roughly the same amount, as does the globe surface. However, while the IO pulley is shifted by the IR's orbital layer, the IO pulley moves only half as far at the IO pulley and lower lid.[11] This means that in infraduction, the IO pulley more closely approximates the lower lid skin surface than in other gaze positions. Elasticity of lower lid tissues contributes to the coordinated shifts of the lower lid, IO pulley, and eye. Fibrous adhesions of the IO-IR pulley to the orbital floor may produce restrictive hypertropia by hindering normal posterior pulley shift during infraduction.[12] It is therefore plausible that milder changes in elastic mechanical forces in the eyelids following blepharoplasty might be transmitted to IR and IO so as to contribute to strabismus following blepharoplasty. Such a putative effect would probably be related to individual variations in lid and bony orbital anatomy.

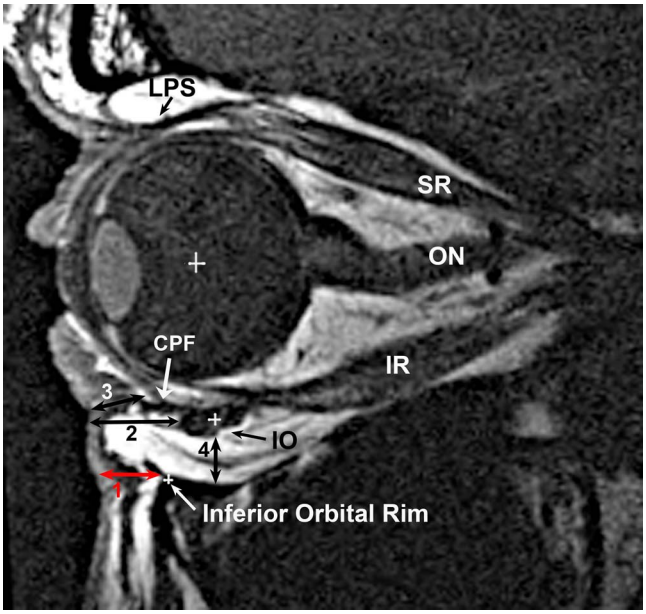

FIGURE 1. Quasi-sagittal, T1-weighted MRI of normal orbit illustrating definitions of anatomic distances. 1: Distance from the lower lid skin surface to the inferior orbital rim. 2: Distance from lower lid skin surface to anterior border of inferior oblique muscle (IO). 3: Distance from lower eyelid skin surface to anterior border of capsulopalpebral fascia (CPF). 4: Distance from inferior border of IO to the orbital floor. *White crosses* indicate centers of globe, IO, and inferior orbital rim, respectively. IR, inferior rectus muscle; SR, superior rectus muscle; ON, optic nerve; LPS, levator palpebrae superioris.

We hypothesized that the lower lid blepharoplasty could affect IO position, since the lower lid is intimately coupled to the IO-IR pulley system. If such an effect were to occur, it should be most pronounced in patients who have shallow orbits or other anatomic features bringing the IO-IR pulley assembly into proximity with the skin. This could cause vertical strabismus following lower lid blepharoplasty.

Therefore, this study sought to investigate, using high-resolution magnetic resonance imaging (MRI), the position of IO relative to the adnexa in subjects with vertical strabismus following lower lid blepharoplasty, comparing these with controls.

METHODS

Subjects

All participating volunteers gave written informed consent according to a protocol conforming to the Declaration of Helsinki and approved by the local Institutional Review Board. Normal volunteers underwent complete eye examinations verifying normal corrected vision, normal ocular versions, orthotropia in all gaze positions, and 40 arc seconds stereopsis by Titmus testing. Strabismic subjects underwent complete ophthalmologic examinations by an author who is a strabismologist, including prism and cover testing.

We studied a control group of 19 normal, orthotropic volunteers recruited by advertisement. From an ongoing study of strabismus and amblyopia, we recruited 11 subjects with incomitant hypertropia following bilateral lower lid blepharoplasty (blepharoplasty group); 13 subjects with hypertropia unrelated to lower lid blepharoplasty but associated with dissociated vertical deviation, the sagging eye syndrome,[13] and idiopathic hypertropia (other hypertropia group); and 2 subjects who underwent bilateral lower lid blepharoplasty but did not develop hypertropia (blepharoplasty without hypertropia group). We recruited the subjects with strabismus from patients who visited our institution with complaints of strabismus or diplopia. Two subjects in the blepharoplasty group were included in an earlier report.[12] No subject had undergone strabismus surgery.

Operative notes for the surgical procedure were available for four subjects with hypertropia following blepharoplasty. Operative records could not be obtained for the other seven subjects in this group.

The two orbits of each subject in the "blepharoplasty group" were separately analyzed to evaluate differences that might account for the laterality of the hypertropia and permit mechanistic inferences concerning its etiology. The "other hypertropia" group was analyzed to identify possible nonspecific effects of hypertropia unrelated to blepharoplasty. Although rarely encountered in the study population, two subjects who had undergone bilateral lower lid blepharoplasty but did not develop hypertropia were included to evaluate the general effects of blepharoplasty.

MRI

A 1.5T General Electric Signa (Milwaukee, WI, USA) scanner was used for imaging using T1, or T2 fast spin echo pulse sequences. Both sequences provide equivalent measurements. Important technical aspects elsewhere published include use of a surface coil array (Medical Advances, Milwaukee, WI, USA) and fixation targets.[14–16] High-resolution (312 μm), quasi-sagittal images of 2-mm thickness and matrix of 256 × 256 parallel to the long axis of the orbit were obtained in target-controlled central gaze, supraduction, and infraduction for each eye. Because the scanned eye was centered on an afocal, monocularly viewed target that does not induce convergence, this procedure avoided confounding caused by strabismus.

Analysis

Image analysis was similar to published methods.[11,17,18] Digital MRIs were quantified using ImageJ (WS Rasband, National Institutes of Health, Bethesda, MD, USA; http://rsb.info.nih.gov/ij/, 1997–2009, accessed February 2009). To normalize position in the quasi-sagittal plane parallel to the long axis of the orbit, positions of IO, inferior orbital rim, and capsulopalpebral fascia (CPF) were translated to place the coordinate origin at the globe area centroid. The CPF is a fibrous band extending from the upper border of IO to the inferior margin of the inferior tarsal plate.[10] The image plane containing the middle of the IR was analyzed. Anterior and inferior positions were taken as negative, and posterior and superior positions as positive, in the quasi-sagittal image plane located closest to the IR center; this location corresponds closely to the IO-IR pulley complex location. The anteroposterior (AP) distance from globe center to IO center was quantified by the differences in the horizontal centroid coordinates. The AP distances from globe center to the inferior orbital rim, from globe center to the CPF, and from IO center to the orbital rim were defined correspondingly by differences in horizontal centroid coordinates.

The vertical distance from globe center to IO center was defined by differences in vertical centroid coordinates. Vertical distances from globe centroid to inferior orbital rim, from globe center to CPF, and from IO center to the orbital rim were correspondingly defined. We also measured the distances between the inferior IO border and orbital floor, between the lower eyelid skin surface and anterior IO border, between the lower eyelid skin surface and the inferior orbital

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Table 1. Characteristics of Subjects in the Blepharoplasty Group

Subject	Sex	Age, y	Side	Hypertropia, Δ, in Central Gaze	Limitation of Infraduction
1	Male	43	Right	15	−2
2	Male	29	Right	10	−2
3	Male	67	Left	8	−2
4	Female	71	Right	6	−1
5	Female	70	Left	4	−1
6	Female	63	Left	14	−2
7	Male	57	Left	18	−3
8	Female	41	Right	10	−2
9	Female	51	Left	8	−2
10	Male	43	Right	10	−2
11	Female	54	Right	6	−1

Normal infraduction was graded 0. Inability to infraduct more than 75% was graded −1. Inability to infraduct more than 50% was graded −2. Inability to infraduct more than 25% was graded −3. Inability to infraduct at all from central gaze was graded −4.

rim, and between the lower eyelid skin surface and the anterior CPF border. Figure 1 illustrates the measurement conventions.

Statistical Analysis

Statistical analyses were performed using SPSS (ver. 16.0 for Windows; SPSS Science, Chicago, IL, USA) with a 0.05 level of significance. Significant effects of groups were evaluated using ANOVA, with subsequent pairwise contrasts by unpaired *t*-tests.

Results

One orbit was imaged in each of 19 normal subjects of mean (±standard deviation [SD]) age 46.2 ± 18.3 (range, 22–68) years. In the blepharoplasty group, 22 orbits were imaged in 11 subjects of mean age 57.8 ± 12.9 (range, 29–70) years; the interval between blepharoplasty surgery and study measurements was 4.5 ± 1.8 (range, 2–7) years. In the other hypertropia group, 26 orbits were imaged in 13 subjects of mean age 50.3 ± 12.0 (range, 27–71) years. In the blepharoplasty without hypertropia group, one orbit was imaged in each of 2 subjects of mean age 60 ± 14 (range, 50–70) years.

In all four subjects of the blepharoplasty group for whom surgical records were available for review, transcutaneous lower lid blepharoplasty had been performed via an elliptical skin incision. Excess skin and fat were excised, but no canthopexy was performed. There were no indications of differences in technique between the hypertropic eyes and hypotropic fellow eyes, and no comments regarding possible injury to the IO.

Table 1 lists clinical characteristics of subjects in the blepharoplasty group, which exhibited 9.9 ± 4.0Δ mean hypertropia in central gaze associated with limited infraduction by the hypertropic eye. The other hypertropia group exhibited 8.7 ± 3.5Δ mean hypertropia without duction limitation. In the other hypertropia group, four subjects had dissociated vertical deviation, seven had idiopathic hypertropia, and two had sagging eye syndrome.

As illustrated by the example MRI in Figure 2, the IO was displaced inferiorly and anteriorly in the hypertropic eye of the blepharoplasty group (Fig. 2C) compared to controls (Fig. 2A) and hypotropic fellow eyes of the blepharoplasty group (Fig. 2B). These data are plotted for all subjects in Figure 3, which summarizes the AP and vertical positions of the IO centroid for every central gaze observation in the hypertropic eyes of the blepharoplasty, control, and blepharoplasty without hypertropia groups. Generally, the observations for the hypertropic eyes of the blepharoplasty group clustered in the lower left quadrant of the graph, below and to the left of the lower diagonal line (Fig. 3, triangles). In contrast, observations for both control groups clustered above and to the right of the upper diagonal line in the upper right quadrant of the graph (Fig. 3, circles). There was a small zone of overlapping observations between the two parallel lines, which were drawn to discriminate the observations for IO position. This implies that IO position alone did not always discriminate the hypertropic orbits from normal orbits.

Table 2 quantitatively compares anatomic relationships of the IO among subject groups. Statistical comparisons are omitted for the blepharoplasty without hypertropia group, which contains only two subjects. The IO was 0.49 ± 1.50 mm anterior to globe center in the hypertropic eye of the blepharoplasty group, but 0.55 ± 1.02 mm posterior in controls ($P = 0.043$) and even farther posterior at 0.80 ± 0.70 mm in the blepharoplasty without hypertropia group. The IO centroid was 15.79 ± 1.38 mm below globe center in the hypertropic eyes of the blepharoplasty group, significantly farther inferior than in controls at 14.69 ± 0.76 mm ($P = 0.004$) or the blepharoplasty without hypertropia group at 14.35 ± 1.03 mm. The AP distance from IO center to the inferior orbital rim was approximately 1 mm less at 5.08 ± 2.40 mm in the hypertropic eyes of the blepharoplasty group than in the control group at 6.17 ± 1.83 mm ($P = 0.046$) and less still than in the blepharoplasty without hypertropia group at 6.96 ± 0.94 mm. The vertical distance between the IO center and the inferior orbital rim was also approximately 1 mm less at 5.31 ± 1.58 mm in the hypertropic eyes of the blepharoplasty group than in the control group at 6.42 ± 1.42 mm ($P = 0.045$), and also less than in the blepharoplasty without hypertropia group at 6.50 ± 0.25 mm. The horizontal distance from the lower eyelid skin surface to the anterior IO border was approximately 1.75 mm less in the hypertropic eyes of the blepharoplasty group at 6.81 ± 1.96 mm than controls at 8.65 ± 1.34 mm ($P = 0.002$) and the blepharoplasty without hypertropia group at 8.10 ± 2.84 mm. However, none of the foregoing variables in the other hypertropia group differed significantly from controls. For example, the AP distance from globe center to IO center was 0.55 ± 1.50 mm in the other hypertropia group and 0.54 ± 1.05 mm in the control group ($P = 0.087$). The vertical distance from the globe center to the IO center in the other hypertropia group was −14.69 ± 0.76 mm, not significantly different from the −15.10

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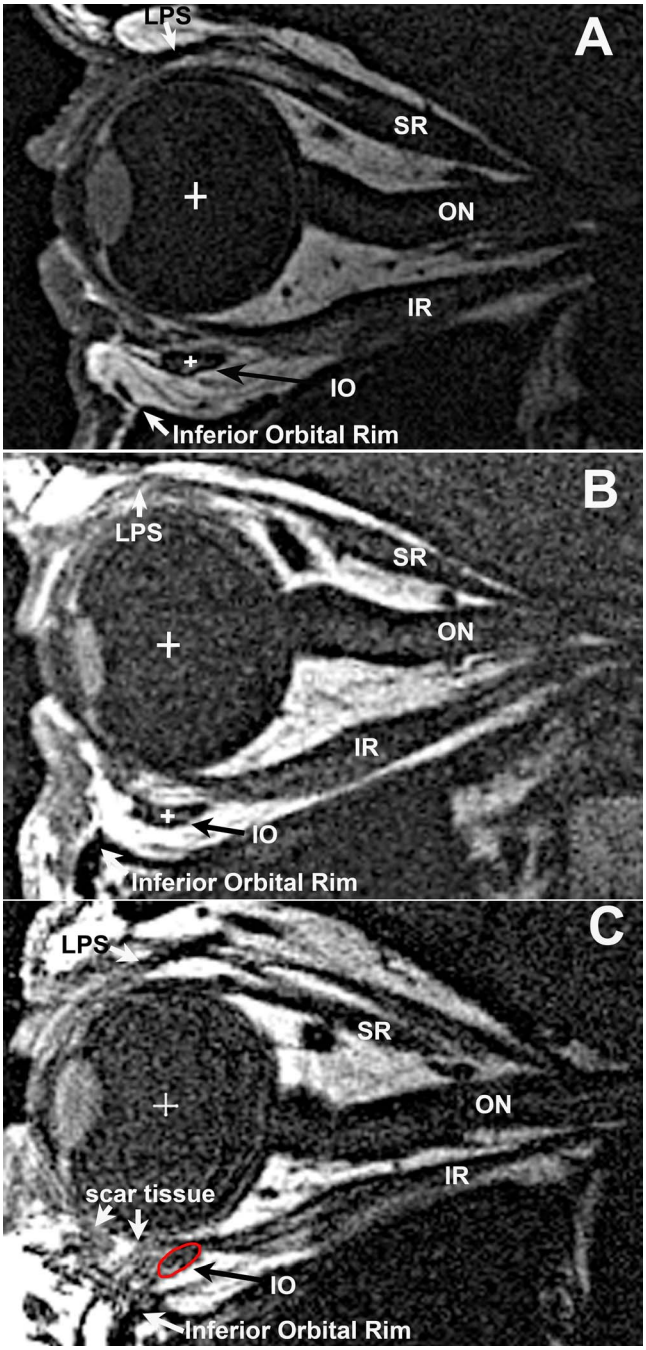

FIGURE 2. Quasi-sagittal MRI of the orbit in central gaze. (**A**) Normal control subject. (**B**) Hypotropic fellow eye in subject 4 of the blepharoplasty group that did not exhibit limited infraduction. (**C**) Hypertropic eye in subject 4 of the blepharoplasty group that exhibited limitation of infraduction; note anterior and inferior shift of the inferior oblique muscle (IO) with dark, band-like features suggesting traction by scar. ON, optic nerve; SR, superior rectus muscle; IR, inferior rectus muscle; LPS, levator palpebrae superioris. *Small white cross* indicates the center of IO or globe.

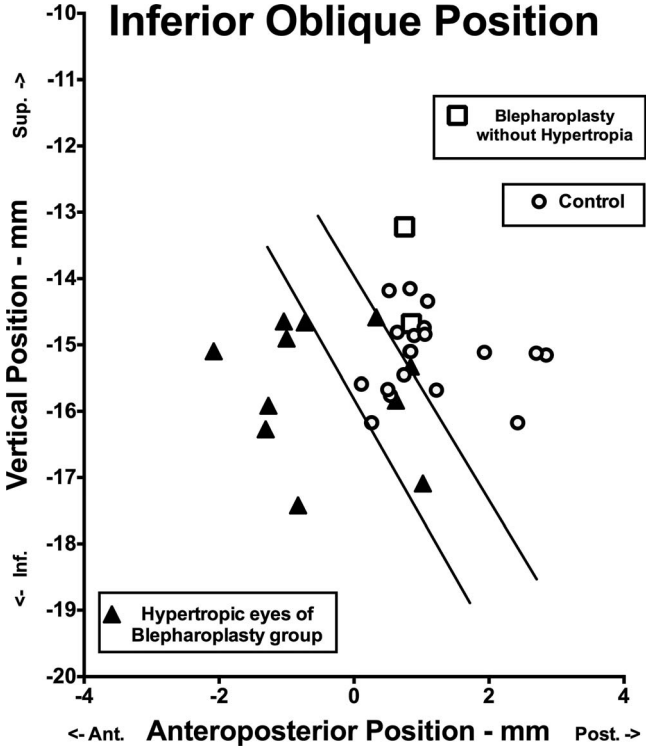

FIGURE 3. Quasi-sagittal plane coordinates of the inferior oblique muscle (IO) in central gaze for controls, the hypertropic eyes of the blepharoplasty group, and the blepharoplasty without hypertropia group. Each symbol indicates one case. The IO was located more anteriorly and inferiorly in the hypertropic eyes of the blepharoplasty group, for whom most observations were below and to the left of the *lower diagonal line*, than in controls and subjects who had undergone blepharoplasty but did not develop hypertropia, for whom most observations were above and to the right of the *upper diagonal line*. The region between *diagonal lines* is a zone of overlapping observations, indicating that IO position did not always discriminate hypertropic orbits.

± 0.84 mm-distance in controls ($P = 0.099$). Neither of the foregoing variables differed significantly between controls and the hypotropic fellow orbits in the blepharoplasty group. Notable differences were observed between hypertropic and hypotropic fellow orbits in the blepharoplasty group. As illustrated by the example in Figures 2B and 2C and summarized numerically for all subjects in Table 3, the IO centroid was farther anterior and inferior in the hypertropic than in hypotropic fellow eyes in central gaze. In hypertropic orbits, the IO centroid was 0.49 ± 1.02 mm anterior to globe center, while in hypotropic fellow orbits the IO centroid was 0.63 ± 1.26 mm posterior ($P = 0.032$). The vertical position of the IO centroid was 15.79 ± 1.38 mm below globe center in the hypertropic orbits, significantly farther inferior than in hypotropic fellow orbits at 14.57 ± 0.72 mm ($P = 0.013$). The anterior IO border was approximately 1.3 mm closer to the lower eyelid skin in hypertropic orbits at 6.81 ± 1.96 mm than hypotropic fellow orbits at 8.12 ± 1.23 mm ($P = 0.042$). However, none of the foregoing variables differed between hypertropic and hypotropic fellow orbits in the other hypertropia group.

Table 2 quantitatively compares anatomic relationships of the IO among subject groups. Statistical comparisons are omitted for the blepharoplasty without hypertropia group, which contains only two subjects. The IO was 0.49 ± 1.50 mm anterior to globe center in the hypertropic eye of the blepharoplasty group. However, the IO was significantly posterior to globe center in controls at 0.55 ± 1.02 mm ($P = 0.043$), the hypotropic fellow eyes of the blepharoplasty group at 0.63 ± 1.26 mm ($P = 0.032$), and the other hypertropia group at 0.54 ± 1.05 mm ($P = 0.043$). The IO centroid was 15.79 ± 1.38 mm inferior to globe center in the hypertropic eyes of the blepharoplasty group. However, the IOs were significantly less inferior in controls at 14.69 ± 0.76 mm ($P =$

Table 2. Anatomic Distances in Central Gaze

Distance, mm ± SD	Blepharoplasty Hypertropic Eye	*P* Value	Control	*P* Value	Blepharoplasty Hypotropic Fellow Eye	*P* Value	Other Hypertropia	Blepharoplasty Without Hypertropia
IO center to globe center								
AP	−0.49 ± 1.02	0.043	0.55 ± 1.50	0.032	0.63 ± 1.26	0.043	0.54 ± 1.05	0.85, 0.75
Vertical	−15.79 ± 1.38	0.004	−14.69 ± 0.76	0.003	−14.57 ± 0.72	0.021	−15.1 ± 0.84	−14.68, −13.22
Rim to globe center								
AP	5.75 ± 2.40	0.712	6.10 ± 2.72	0.059	6.54 ± 1.86	0.752	6.38 ± 2.82	6.53, 4.52
Vertical	−17.85 ± 2.09	0.131	−20.44 ± 1.71	0.283	−18.54 ± 1.59	0.368	−21.02 ± 1.64	−20.22, −19.11
IO center to rim								
AP	5.08 ± 2.40	0.046	6.17 ± 1.83	0.048	6.01 ± 1.43	0.012	6.98 ± 2.10	7.64, 6.30
Vertical	−5.31 ± 1.58	0.045	−6.42 ± 1.42	0.046	−6.31 ± 1.54	0.048	−5.87 ± 1.48	−6.25, −6.64
CPF to globe center								
AP	4.30 ± 1.04	0.587	4.93 ± 1.93	0.759	4.36 ± 1.27	0.467	5.13 ± 1.07	5.12, 4.09
Vertical	−13.27 ± 0.98	0.670	−13.12 ± 1.01	0.245	−13.63 ± 2.98	0.713	−13.18 ± 0.72	−13.90, −12.84
Skin to IO	6.81 ± 1.96	0.002	8.65 ± 1.34	0.004	8.12 ± 1.23	0.010	7.95 ± 1.92	8.67, 8.25
Skin to rim	6.92 ± 2.28	0.505	6.40 ± 2.02	0.645	6.87 ± 2.04	0.495	6.42 ± 2.98	7.22, 5.22
Skin to CPF	4.57 ± 1.28	0.557	4.80 ± 0.96	0.529	4.87 ± 1.04	0.645	4.72 ± 0.88	4.69, 4.22
IO to orbital floor	1.75 ± 0.72	0.952	1.77 ± 1.07	0.782	1.67 ± 0.48	0.438	1.93 ± 1.06	2.66, 1.88
CPF	4.06 ± 1.16	0.702	4.23 ± 1.16	0.674	3.84 ± 1.02	0.781	3.96 ± 1.43	3.45, 3.65

Each *P* value column is for comparison between the hypertropic eyes of the blepharoplasty group and the group in the column to its right (unpaired *t*-test). ANOVA demonstrated no significant differences among normal controls, hypotropic eyes of blepharoplasty group, and the other hypertropia group. Statistical comparisons are omitted for the blepharoplasty without hypertropia group, for whom both individual measurements are reported. Rim, inferior orbital rim; AP, anterior-posterior.

0.004), the hypotropic fellow eyes of the blepharoplasty group at 14.57 ± 0.72 mm ($P = 0.003$), and the other hypertropia group at 15.1 ± 0.84 mm ($P = 0.021$).

The AP distance from IO center to the inferior orbital rim was 5.08 ± 2.40 mm in the hypertropic eyes of the blepharoplasty group. However, this distance was significantly greater in the control group at 6.17 ± 1.83 mm ($P = 0.046$), the hypotropic fellow eyes of the blepharoplasty group at 6.01 ± 1.43 mm ($P = 0.048$), and the other hypertropia group at 6.98 ± 2.10 mm ($P = 0.012$). The vertical distance between the IO center and the inferior orbital rim was 5.31 ± 1.58 mm in the hypertropic eyes of the blepharoplasty group. However, this distance was also longer in the control group at 6.42 ± 1.42 mm ($P = 0.045$), the hypotropic fellow eyes of the blepharoplasty group at 6.31 ± 1.54 mm ($P = 0.046$), and the other hypertropia group at 5.87 ± 1.48 mm ($P = 0.048$). The horizontal distance from the lower eyelid skin surface to the anterior IO border was 6.81 ± 1.96 mm in the hypertropic eyes of the blepharoplasty group. This distance was significantly longer in controls at 8.65 ± 1.34 mm ($P = 0.002$), the hypotropic fellow eyes of the blepharoplasty group at 8.12 ± 1.23 mm ($P = 0.004$), and the other hypertropia group at 7.95 ± 1.92 mm ($P = 0.010$). The number of subjects in the blepharoplasty without hypertropia group was insufficient for statistical comparisons.

None of the foregoing variables differed among the control, hypotropic fellow eyes in the other hypertropia, and the other hypertropia groups ($P > 0.05$, ANOVA).

Normally, the IO pulley, and therefore the IO at the IR center, shifts anteriorly in supraduction and posteriorly in infraduction. As illustrated in Figure 4, the posterior IO shift in infraduction was decreased following blepharoplasty, particularly when there was scarring around the IO. Figure 5 and Table 3 quantify changes of variables from central gaze to infraduction. In infraduction, the IO center shifted less posteriorly in the hypertropic eyes of the blepharoplasty group at 0.40 ± 0.43 mm than controls at 1.34 ± 0.81 mm ($P = 0.001$), the hypotropic fellow eyes of the blepharoplasty group at 1.25 ± 0.65 mm ($P = 0.001$), and the other hypertropia group at 1.31 ± 0.55 mm ($P = 0.001$), but shifted more vertically at 1.26 ± 0.22 mm in the hypertropic eyes of the blepharoplasty group than in controls at 0.73 ± 0.20 mm ($P = 0.001$), the hypotropic fellow eyes of the blepharoplasty group at 0.68 ± 0.23 mm ($P = 0.001$), and the other

Table 3. Distance Changes From Central Gaze to Infraduction

Distance, mm ± SD	Blepharoplasty Hypertropic Eye	*P* Value	Control	*P* Value	Blepharoplasty Hypotropic Fellow Eye	*P* Value	Other Hypertropia
IO center to globe center							
AP	0.40 ± 0.43	0.001	1.34 ± 0.81	0.001	1.25 ± 0.65	0.001	1.31 ± 0.55
Vertical	1.26 ± 0.22	0.001	0.73 ± 0.20	0.001	0.68 ± 0.23	0.001	0.60 ± 0.38
Skin to IO	0.49 ± 0.70	0.015	1.21 ± 0.56	0.018	1.09 ± 0.47	0.016	1.13 ± 0.68
IO to orbital floor	0.27 ± 0.54	0.310	0.14 ± 0.55	0.425	0.13 ± 0.22	0.430	0.18 ± 0.34

Each column of *P* values is for comparisons between hypertropic eyes of the blepharoplasty group and the other group in the column to its right.

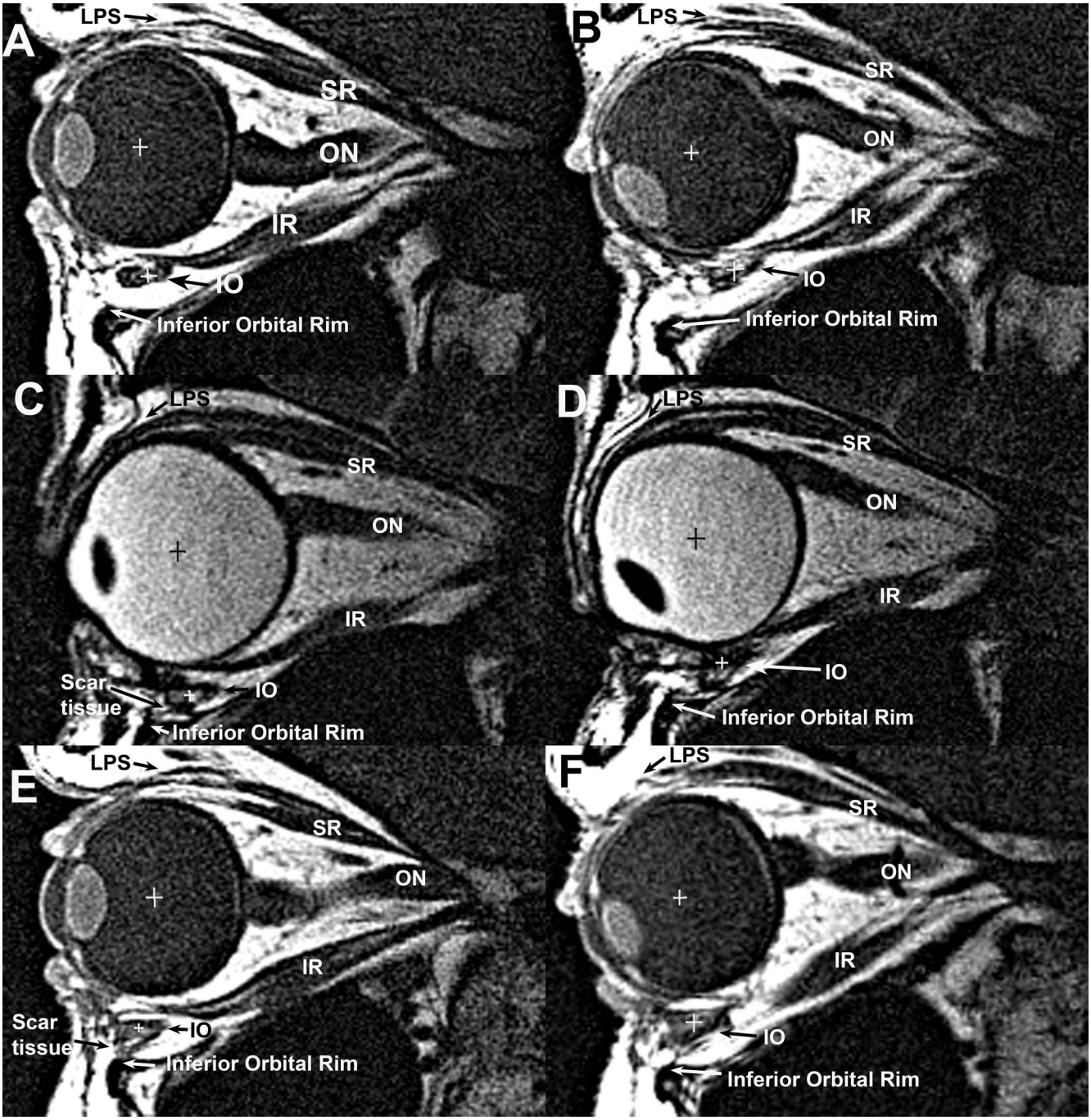

FIGURE 4. Quasi-sagittal MRI in central gaze (**A**, **C**, **E**) and infraduction (**B**, **D**, **F**). (**A**, **B**) Normal subject. Inferior oblique muscle (IO) moves posteriorly from central gaze to infraduction. (**C**, **D**) Subject 2 of blepharoplasty group who exhibits scar tissue around IO, inferior orbital rim, and floor. Inferior oblique muscle moves less posteriorly from central gaze (**C**) to infraduction (**D**) than normal. (**E**, **F**) Subject 5 of blepharoplasty group who exhibits modest scar tissue between IO and inferior orbital rim. Inferior oblique muscle moves less posteriorly from central gaze to infraduction than normal. Abbreviations as in Figure 2.

hypotropia group at 0.60 ± 0.38 mm ($P = 0.001$). Thus, IO motion was shifted from posterior to inferior in the hypertropic eyes of the blepharoplasty group. None of the foregoing changes from central gaze to infraduction was significantly different among the control, the hypotropic fellow eyes of the blepharoplasty group, and the other hypertropia group ($P > 0.05$, ANOVA).

Shift of the IO from central gaze to supraduction was also investigated in quasi-sagittal images. The effect of blepharoplasty on IO shift did not include supraduction. Neither the change in horizontal nor vertical position of the IO center differed significantly among controls, the hypertropic and hypotropic eyes of the blepharoplasty group, and the other hypertropia group ($P > 0.4$).

DISCUSSION

This study demonstrated by MRI abnormally anterior and inferior IO position in central gaze in hypertropic orbits that

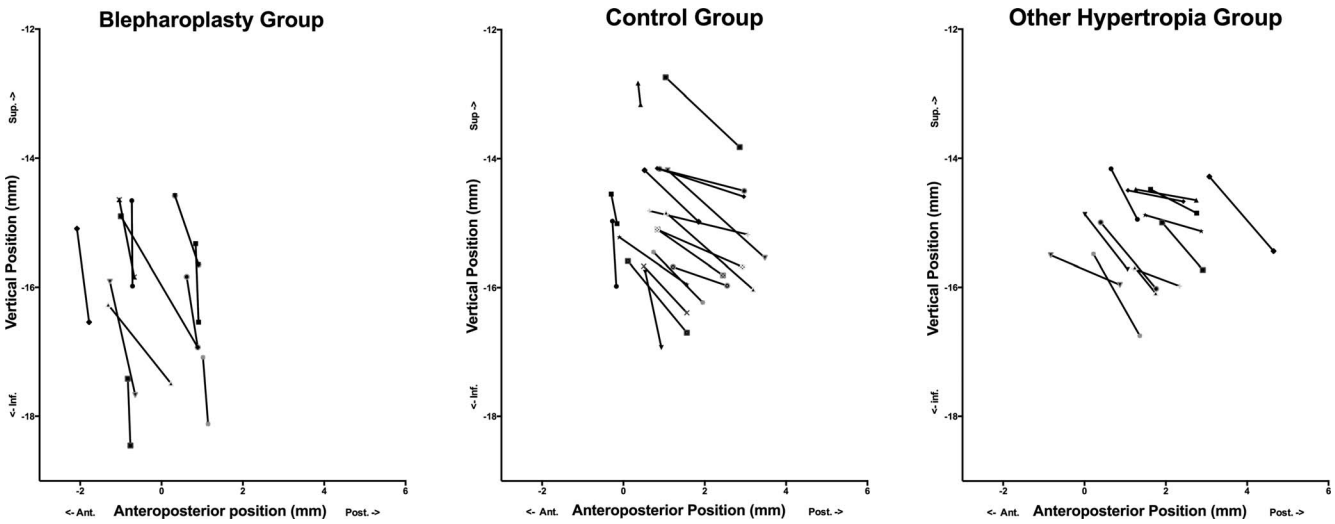

FIGURE 5. Comparison of changes of inferior oblique muscle (IO) position from central gaze to infraduction among control, blepharoplasty, and other hypertropia groups. The blepharoplasty group showed less posterior and more inferior shift of IO than the other two groups.

had limited infraduction following bilateral lower lid blepharoplasty, with the IO correspondingly closer to the lower eyelid skin surface and inferior orbital rim. Such proximity potentially reduces the mechanical independence of the IO from skin and bone structures. This was an asymmetric effect, since the abnormalities were not present in the hypotropic contralateral orbits of the same subjects following lower lid blepharoplasty, nor in subjects with hypertropia not related to blepharoplasty, nor in orbits in which blepharoplasty was performed but was not complicated by hypertropia. Moreover, in the hypertropic orbits of the blepharoplasty group, the physiologic posterior IO shift during infraduction was redirected inferiorly. The foregoing quantitative abnormalities were associated with scar tissue bands extending from the IO pulley to the orbital floor visible on MRI. This corresponds to the region of Lockwood's ligament. However, the normal anterior shift of the IO during supraduction was unimpaired after lower lid blepharoplasty.

The IO path in living humans exhibits an inflection in its path roughly at the lateral aspect of the IR crossing within the Lockwood ligament, due to the IO pulley.[11,19,20] The gaze-related inflection in IO path corresponds to its encirclement by a pulley composed of a dense ring of collagen, stiffened by elastin and smooth muscle and united with the IR pulley so that IR pulley position influences position of the IO pulley. Presumably the anterior and inferior shift of the IO pulley following lower lid blepharoplasty is due to anterior and inferior traction from foreshortened lower eyelid tissues. Therefore, there is likely to be greater than normal elastic resistance to posterior shift of the IR pulley in infraduction following blepharoplasty. If this effect were highly asymmetrical, hypertropia could result from vertical oculorotary force imbalance. If the IR were recessed on adjustable suture as a part of strabismus surgery performed for some separate indication in a patient who had previously undergone lower lid blepharoplasty, the abnormally anterior and inferior position of the IR pulley would distract the IR tendon inferiorly and anteriorly away from globe tangency, perhaps promoting nonattachment to the sclera that could result in late IR slippage. In addition, IR slippage is a common complication encountered following IR recession, especially in Graves' ophthalmopathy where the IR is stiff and fibrotic.[21] In hypertropic eyes of the blepharoplasty group, the IO moved less posteriorly, but more inferiorly, than in the controls and the hypotropic fellow eyes of the blepharoplasty group. There was less change in distance from the anterior skin surface to the IO anterior border in the hypertropic eyes of the blepharoplasty group than in controls and the hypotropic fellow eyes of the blepharoplasty group.

Several mechanisms may explain the displacement and abnormal motion of the IO pulley in hypertropic eyes following lower lid blepharoplasty. First, surgical removal of fat and connective tissue surrounding the IO and IR pulley could induce the scarring around the IO pulley that was observed here by MRI. For example, in subject 2 of the blepharoplasty group, extensive scar tissue was present around the IO, the inferior orbital rim, and the orbital floor, exhibiting visible tension lines that appeared to draw the IO inferiorly and anteriorly. Although subject 5 of the blepharoplasty group exhibited less scar tissue, the IO was located more anteriorly and inferiorly than in normal subjects; this might reflect presurgical anatomic variation, or perhaps surgical technique. Hypotropic fellow eyes of the blepharoplasty group did not show anterior and IO inferior displacement, suggesting that this phenomenon might be due to surgical technique. Differences between hypertropic and hypotropic fellow eyes probably reflect variation in technique of the surgeon, who likely intended to perform a bilaterally symmetrical operation, but for reasons perhaps not apparent even intraoperatively, failed to accomplish identical technique in the two eyes. Not surprisingly, available operative records provided no useful information regarding asymmetrical maneuvers or instances of direct IO injury.

We considered the possibility that degenerative changes that made the patients candidates for lower lid blepharoplasty in the first place could contribute to restrictive limitation to infraduction, but these increases in tissue laxity should make the IO pulley more mobile, rather than less, and were not evident in the hypotropic fellow eyes of blepharoplasty groups or in controls.

Because the rarity of strabismus after blepharoplasty makes it impractical to obtain detailed MRI studies of orbital anatomy preoperatively, the present investigation cannot conclusively confirm the possibility that a preexisting anatomic variation in the patients with strabismus following lower lid blepharoplasty may have made them susceptible to this complication. That possibility is consistent with normal quantitative findings in the two subjects who did not develop the hypertropia

following lower lid blepharoplasty; however, this control group is small. It is uncertain if the strabismus observed after lower lid blepharoplasty is entirely the consequence of technical aspects of lower lid blepharoplasty, or if preexisting anatomic factors may be responsible. For example, maxillary hypoplasia or shallow orbits could place the IO into proximity with the lower eyelid skin even without lid surgery. This potentially asymmetric anatomic factor might increase the possibility that surgical dissection during blepharoplasty might incorporate the connective tissues around the IO, and be responsible for the current observation of scar tissue bands from the orbital rim to the IO-IR pulley region. Blepharoplasty was performed via skin incisions in all four subjects with hypertropia for whom surgical records were available. Reliable information could not be obtained concerning the likely variable amount of fat excision, dissection, and skin excision related to the lower eyelid blepharoplasty performed in each subject. It is probable that surgical dissection close to the IO-IR pulley region would have been unrecognized at the time of surgery. It is unclear if greater technical attention to this dissection of fat pads near the IO could avoid scaring around the pulley region, although well-visualized technique would seem prudent.

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