Location and Stability of Rectus Muscle Pulleys
Muscle Paths as a Function of Gaze

Robert A. Clark,* Joel M. Miller,† and Joseph L. Demer*‡

Purpose. The paths of the rectus extraocular muscles (EOMs) are constrained by pulleys, connective tissue sleeves mechanically coupled to the orbital walls. This study sought to investigate, using high-resolution magnetic resonance imaging (MRI), the location and stability of EOM pulleys in normal subjects and those with strabismus.

Methods. Multiple contiguous coronal MRI scans spanning the anterior-to-posterior extent of the orbit during primary gaze, upgaze, downgaze, adduction, and abduction were analyzed digitally to determine the paths of the rectus EOMs. Pulley locations were inferred from EOM paths.

Results. Data for 10 orbits of six normal subjects established the normal paths of the rectus extraocular muscles in primary gaze. Muscle paths in primary position were highly uniform across normal subjects. In secondary gaze positions, rectus muscle paths at the level of the pulleys exhibited small but consistent shifts, relative to the orbit, opposite the direction of gaze, consistent with the expected mechanical effects of the intermuscular connective tissue suspensions of the pulleys. Twelve orbits of seven subjects with strabismus showed, as a group, no significant difference from normal in rectus muscle paths in primary gaze and no significant difference from normal in changes of muscle paths in secondary gaze. Two subjects with incomitant strabismus were found to have grossly abnormal rectus muscle paths in primary gaze, suggesting heterotopic pulleys. Computer simulations of these heterotopic pulley locations accounted for the observed patterns of incomitant strabismus in both.

Conclusions. High-resolution MRI can determine the location and sideslip of rectus EOM pulleys. Pulley position is highly uniform across normal subjects, consistent with the notion that musculo-orbital tissue connections determine the pulling direction of the rectus EOMs. In normal subjects and subjects with strabismus, pulleys exhibit small shifts with eccentric gaze that are consistent with secondary intermuscular, but not musculo-global, mechanical couplings. Heterotopic pulley position is a potential cause of incomitant strabismus. Invest Ophthalmol Vis Sci. 1997;38:227-240.

Recent studies exploiting high-resolution magnetic resonance imaging (MRI) of rectus extraocular muscles (EOMs) have facilitated a new understanding of EOM paths and their behavior during changes in gaze. Historically, the concept of rectus EOM behavior has evolved from the “shortest path” hypothesis,¹ by which the muscle belly was thought to have complete freedom to slip to the path of least tension, to the “permitted sideslip” hypothesis, by which unspecified constraints or musculo-global elasticities²³ were proposed to allow only limited muscle path displacement during gaze shifts.

The most recent evidence demonstrates that paths of the rectus EOM bellies are tightly constrained in the orbit during changes in gaze except, of course, for their most anterior, tendinous insertions.⁵⁻⁷ Rectus EOMs are now known to pass through connective tissue sleeves—composed of collagen, elastin, and smooth muscle—that function as pulleys.⁸⁻⁹ Serial histologic sectioning has demonstrated that these pulleys are coupled mechanically to the orbital walls, directly and indirectly, by intermuscular couplings in posterior Tenon’s fascia.¹⁰ The pulleys serve as the effective me-
TABLE 1. Clinical Profiles of Subjects With Strabismus

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Primary Deviation</th>
<th>Incomittance</th>
<th>Prior Strabismus Surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>CB</td>
<td>29</td>
<td>F</td>
<td>Right hypertropia 3°</td>
<td>Greater in left gaze</td>
<td>None</td>
</tr>
<tr>
<td>SB</td>
<td>58</td>
<td>M</td>
<td>Right hypertropia 2°</td>
<td>Greater in downgaze</td>
<td>Right inferior rectus recession (unknown amount)</td>
</tr>
<tr>
<td>AC</td>
<td>50</td>
<td>M</td>
<td>Esotropia 15°</td>
<td>&quot;A&quot; pattern</td>
<td>None</td>
</tr>
<tr>
<td>TL</td>
<td>30</td>
<td>M</td>
<td>Exotropia 3°, right hypertropia 5°</td>
<td>&quot;A&quot; pattern</td>
<td>Bilateral superior oblique tenotomies</td>
</tr>
<tr>
<td>RM</td>
<td>36</td>
<td>M</td>
<td>Exotropia 7°</td>
<td>&quot;A&quot; pattern</td>
<td>Bilateral medical rectus resections (unknown amount)</td>
</tr>
<tr>
<td>JM</td>
<td>60</td>
<td>M</td>
<td>Left hypertropia 8°, exotropia 3°</td>
<td>Greater in right gaze, &quot;V&quot; pattern exotropia</td>
<td>None</td>
</tr>
<tr>
<td>CP</td>
<td>28</td>
<td>F</td>
<td>Left hypertropia 5°</td>
<td>Greater in downgaze</td>
<td>None</td>
</tr>
</tbody>
</table>

Mechanical origins of the EOMs, exerting a profound influence on EOM behavior. This effect is seen most clearly after muscle transposition surgery, in which pulleys markedly limit the path displacement of transposed muscles. Pulley ultrastructure is specialized for high internal rigidity, capable of resisting displacement during rectus muscle contraction, and the pulleys are suspended by connective tissue containing richly innervated smooth muscle bands. Computer simulation of orbital mechanics shows that binocular alignment is highly sensitive to pulley location. However, the early MRI studies of muscle paths were performed on small numbers of subjects using scanning techniques that have now been considerably improved. Quantitative normative data on human pulley locations are lacking.

These observations suggest that abnormal pulley sideslip or abnormal pulley position might cause or contribute to incomitant strabismus. For example, a V-pattern strabismus could result from a lateral rectus muscle pulley inferiorly displaced or a medial rectus muscle pulley superiorly displaced in the orbit. As another example, weakened pulleys could result in excess rectus muscle sideslip during changes in gaze, leading to incomitant strabismus caused by asymmetrical shifts in muscle positions and elastic tensions.

Histologic evidence indicates that the posterior orbital paths of the EOMs are determined entirely by their origins at the annulus of Zinn and by the locations of the pulleys near the equator of the globe. No other tissues exist that could influence posterior muscle paths significantly. Thus, despite the inability of MRI to image the pulleys directly in most cases, the paths of the EOMs that are so obvious on MRI imaging must lead directly to the pulleys, whose anteroposterior location is known from the histology to lie at and just posterior to the equator of the globe in primary gaze. The current study was designed to examine EOM paths to infer in this manner the location and sideslip of the fibromuscular pulleys during gaze shifts in normal patients. These findings were used to evaluate the position and sideslip of the fibromuscular pulleys in patients with known incomitant strabismus.

METHODS

Six normal volunteers were recruited by advertisement and were examined to verify normal ocular motility and the absence of strabismus. From patients enrolled in an ongoing clinical study, we selected seven subjects with incomitant strabismus (Table 1). Four of the seven subjects with strabismus had incomitant hypertropia, as measured with a Hess screen. One of the subjects with hypertropia also had a V-pattern strabismus, consisting of an exodeviation (divergence) in upgaze that shifted gradually to an esodeviation (convergence) in downgaze. An A-pattern strabismus is a horizontal strabismus with greater divergence or less convergence in downgaze. One subject had A-pattern esotropia, and the remaining two subjects had A-pattern exotropias. Qualitative data on subject TL have been briefly presented elsewhere. Three of the seven subjects with strabismus underwent strabismus surgery before analysis (Table 1).

After obtaining informed, written consent according to a protocol conforming to the Declaration of Helsinki and approved by the Human Subject Protection Committee at the University of California, Los Angeles, all patients underwent high-resolution MRI using a superconducting 1.5 T General Electric Signa (Milwaukee, WI) or Picker Vista (Cleveland, OH) scanner following techniques described in detail elsewhere. Briefly, subjects’ heads were stabilized using foam cushions and tape. A surface coil was placed over the scanned orbit, and multiple contiguous coronal images 3 mm in thickness were obtained with a 256 × 192 or 256 × 256 matrix over a 10 cm square field of view, giving a pixel resolution of 390 μm. A fixation target for primary position was attached to the inside of the scanner magnet, and other targets were used to maintain secondary gaze positions. The position of the globe–optic nerve junction in acquired images was used to determine the actual amount of globe...
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FIGURE 1. Coronal magnetic resonance images of a normal right orbit for plane 0 in primary and secondary gaze positions. The transverse diagram (bottom left) demonstrates the movement of the globe-optic nerve junction from point C in abduction to point D in adduction, allowing calculation of the total angle of rotation of the globe with change in horizontal gaze position. Similarly, the sagittal diagram (bottom right) demonstrates the movement of the globe-optic nerve junction from point A in upgaze to point B in downgaze, allowing calculation of the total angle of rotation with change in vertical gaze. Muscle and orbital borders have been outlined digitally in white. Video displays of computer images have greater contrast and resolution than print images. SR = superior rectus muscle; LR = lateral rectus muscle; IR = inferior rectus muscle; MR = medial rectus muscle.

rotation for positions of secondary gaze (Fig. 1). Images of primary gaze, upgaze, and downgaze were obtained for all patients, as were abduction and adduction for normal subjects.

Digital magnetic resonance images were transferred to Macintosh computers (Apple Computer, Cupertino, CA) and converted to 8-bit tagged image file format using locally developed software and were analyzed quantitatively using the program NIH Image (W. Rasband, National Institutes of Health; available by ftp from zippy.nimh.nih.gov or on floppy disk from NTIS, 5825 Port Royal Road, Springfield, VA 22161, part number PB95-500195GE1). Images of left orbits were reflected digitally to the orientation of a right orbit to allow uniform analysis of EOM positions.

To assess the effects of gaze changes on orbital anatomy, it was important to correct for small concomitant changes in head position. Normalization of image position and orientation facilitated quantitative comparisons and summaries across subjects. The magnetic field coordinates of the MRI scanner served as references for rotational corrections.

To normalize position in the coronal plane, all rectus muscle positions were translated to place the coordinate origin at the area centroid of the orbit. Normalization of rotation in the coronal plane by rotating the image to align the interhemispheric fissure of the brain with the scanner-defined vertical meridian (Fig. 2). This rotational correction ranged from 12° clockwise to 8.5° counterclockwise. In anterior image planes without a clear view of the interhemispheric fissure of the brain, the angle in the coronal plane
Figure 2. Coronal plane magnetic resonance imaging (MRI) of a normal right orbit demonstrates the two rotational angle corrections measured in each gaze position. The vertical correction was used to rotate all orbits to bring the interhemispheric fissure of the brain to the vertical position (as defined by the magnetic field coordinates of the MRI scanner). The horizontal angle measured was used to maintain coronal rotational alignment of bony structures during changes of gaze in image planes that lacked a distinct view of the interhemispheric fissure. Video displays of computer images have greater contrast and resolution than print images. SR = superior rectus muscle; LR = lateral rectus muscle; IR = inferior rectus muscle; MR = medial rectus muscle; SO = superior oblique muscle.

The orbital roof angle is a measure of the sagittal plane tilt (pitch) of the subject's head. The sagittal plane orbital roof angle was measured by reformatting the sequence of coronal images into a single sagittal image and measuring the angle between the orbital roof and the scanner horizontal axis (Fig. 3). The orbital roof angle varied less than 1° on average across gaze angles.

Table 2. Position and Sideslip of Rectus Extraocular Muscles in Plane-1 (mm from orbital center)

<table>
<thead>
<tr>
<th></th>
<th>Primary Gaze</th>
<th>Upgaze</th>
<th>Downgaze</th>
<th>Abduction</th>
<th>Adduction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>X</td>
<td>Y</td>
<td>X Change</td>
<td>Y Change</td>
<td>X Change</td>
</tr>
<tr>
<td>Normal Subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medial rectus</td>
<td>12.1 ± 0.5</td>
<td>0.1 ± 0.7</td>
<td>0.1 ± 0.3</td>
<td>0.3 ± 0.4</td>
<td>0.1 ± 0.3</td>
</tr>
<tr>
<td>Superior rectus</td>
<td>-1.4 ± 0.3</td>
<td>12.3 ± 0.5</td>
<td>1.0 ± 0.7</td>
<td>-1.6 ± 0.7</td>
<td>0.4 ± 0.6</td>
</tr>
<tr>
<td>Lateral rectus</td>
<td>-11.7 ± 0.3</td>
<td>-0.8 ± 0.4</td>
<td>0.0 ± 0.1</td>
<td>-0.7 ± 0.4</td>
<td>0.6 ± 0.2</td>
</tr>
<tr>
<td>Inferior rectus</td>
<td>1.7 ± 0.6</td>
<td>-12.3 ± 0.5</td>
<td>1.1 ± 0.3</td>
<td>-1.0 ± 0.3</td>
<td>-1.3 ± 0.3</td>
</tr>
<tr>
<td>Subjects With Strabismus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medial rectus</td>
<td>12.2 ± 0.5</td>
<td>-0.8 ± 0.9</td>
<td>0.1 ± 0.1</td>
<td>0.3 ± 0.4</td>
<td>0.2 ± 0.2</td>
</tr>
<tr>
<td>Superior rectus</td>
<td>-1.4 ± 0.10</td>
<td>11.9 ± 0.8</td>
<td>0.9 ± 1.0</td>
<td>-1.1 ± 0.8</td>
<td>0.6 ± 0.7</td>
</tr>
<tr>
<td>Lateral rectus</td>
<td>-12.9 ± 0.4</td>
<td>-0.4 ± 1.6</td>
<td>-0.1 ± 0.2</td>
<td>-0.9 ± 0.4</td>
<td>0.0 ± 0.2</td>
</tr>
<tr>
<td>Inferior rectus</td>
<td>1.5 ± 1.2</td>
<td>-12.5 ± 0.5</td>
<td>1.9 ± 0.3</td>
<td>-0.9 ± 0.4</td>
<td>-1.8 ± 0.7</td>
</tr>
</tbody>
</table>
FIGURE 4. Three contiguous anterior coronal magnetic resonance imaging planes of a normal orbit in primary position, demonstrating the procedure for defining the anteroposterior position of the globe center with respect to plane 0. Each image plane is 3 mm thick. Video displays of computer images have greater contrast and resolution than print images.

Finally, to normalize the anteroposterior position, the plane containing the globe–optic nerve junction was designated plane 0. The center of the globe in the sagittal plane was estimated with subpixel resolution by fitting a circle to three points on the globe images in planes −2, −3, and −4 (Fig. 4). Globes centers, so determined, were within 0.4 mm for all patients and all gaze positions. Rotation of the line joining the center of the globe–optic nerve junction and the globe center was used to estimate globe rotation in secondary gaze positions (Fig. 1). Our reconstruction methods assume minimal globe translation during changes in gaze. Biomechanical simulation suggests this to be a reasonable assumption because we calculate anteroposterior displacement to be less than 0.4 mm for up to 30° rotations of gaze in all directions.13,14

Pulley locations were inferred, and binocular alignment was simulated using the Orbit 1.5 Gaze Mechanics Simulation program (Orbit; Eidactics, San Francisco, CA) running on Macintosh computers (Apple Computer). Orbit simulates binocular alignment using static force balance equilibrium equations based on orbital parameters, such as innervations, globe dimensions, EOM insertions, lengths, stiffness, pulley positions, and contractile forces. The program then calculates the behavior of the EOMs and globes based on equations and methods given, in part, in Robinson,2 Miller and Robinson,3 and Miller and De Mer.15 Pulley positions (based on histologic studies) in Orbit’s description of a normal eye were taken as a starting point. Then the lateral–medial and superior–inferior coordinates of Orbit’s pulleys were altered to match Orbit’s simulated muscle paths to the paths observed in the MRI scans. To estimate anteroposterior displacement of the globe during changes in gaze, a translational compliance of 20 g/mm for small anteroposterior displacement14 was used in Orbit simulations, predicting a maximum anteroposterior shift in globe position of 0.4 mm for 30° secondary gaze positions in normal orbits. Translation might differ in subjects with strabismus, but predicted translation can be computed in Orbit simulations.

RESULTS
We studied 10 orbits in six normal subjects and 12 orbits in seven subjects with strabismus. For all subjects, images were obtained for primary position, upgaze, and downgaze. Abduction and adduction images were obtained in eight orbits of five normal subjects.
For each gaze position, six sequential planes were analyzed, beginning with plane –2 (6 mm anterior to the globe-optic nerve junction) and extending posteriorly to plane 3 (9 mm posterior to the globe-optic nerve junction) (Fig. 5). Plane –1 (3 mm anterior to the globe-optic nerve junction) represents the most anterior location at which muscle tendons were differentiated clearly from surrounding tissue. The average position in primary gaze of the area centroids of normal and strabismic EOMs are shown in Figure 6 for each of these image planes. Coordinates of the centroids are tabulated in Table 2 for image plane –1. In no image plane was there a significant difference in mean position of any rectus EOM between the normal group and the group with strabismus ($P < 0.05$ using the Bonferroni adjustment for multiple comparisons$^{16}$). This finding indicates that rectus muscle paths in the primary position are similar for normal subjects and subjects with strabismus.

Normal subjects and subjects with strabismus aver-
FIGURE 6. Average positions (relative to right orbital center and viewed as if facing the subject) of centroids of the rectus extraocular muscles for primary gaze in all coronal image planes in normal subjects and subjects with strabismus. Error bands = ±2 SD. SR = superior rectus muscle; LR = lateral rectus muscle; IR = inferior rectus muscle; MR = medial rectus muscle. (open circles) Normal subjects. (closed diamonds) Subjects with strabismus.

-aged 44° of ocular rotation from upgaze to downgaze (normal subjects, 44.1° ± 6.3° SD; subjects with strabismus, 44.6° ± 6.8° SD). These differences were not significant. Normal subjects averaged 40.7° (± 5.2° SD) of ocular rotation from abduction and adduction.

Rectus EOM paths underwent small but consistent changes with gaze. We term these shifts EOM path sideslip. The effect of changes in gaze on EOM position in plane -1 are summarized in Figures 7 and 8, and tabulated in Table 2, for normal subjects and subjects with strabismus, respectively.

In general, EOMs were displaced more during gaze changes in their plane of action (as agonists or antagonists), and were displaced less during gaze changes out of their plane of action. The displacements were small, averaging less than 2 mm even for EOMs in their plane of action. Displacements perpendicular to the plane of action of EOMs were always in a direction opposite the displacement of gaze; e.g., the lateral rectus (LR) path was displaced downward on upgaze. Thus, these shifts do not simply reflect the necessary movement of the EOM insertions as the globe rotates.

In normal subjects and subjects with strabis-
TABLE 3. Rectus Pully Position Displacements in Subject JM

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Right Orbit</th>
<th>Left Orbit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medial rectus</td>
<td>0.7 mm inferior</td>
<td>0.6 mm superior</td>
</tr>
<tr>
<td>Superior rectus</td>
<td>3.9 mm lateral</td>
<td>1.8 mm lateral</td>
</tr>
<tr>
<td>Lateral rectus</td>
<td>3.8 mm inferior</td>
<td>4.6 mm inferior</td>
</tr>
<tr>
<td>Inferior rectus</td>
<td>0.7 mm medial</td>
<td>1.1 mm medial</td>
</tr>
</tbody>
</table>

mus, the medial rectus (MR) muscle was the most stable, shifting less than 0.4 mm from its primary gaze position with horizontal rotations and less than 0.3 mm with vertical rotations (Figs. 7, 8). The superior rectus (SR) muscle was the most mobile, but only in its plane of action. In normal subjects, the SR shifted 1.6 mm inferiorly on upgaze and 1 mm superiorly on downgaze (for a total vertical excursion of almost 3 mm), inferiorly on adduction 0.5 mm, and superiorly on abduction 0.8 mm (Figs. 7, 8). Horizontal gaze did not affect the horizontal position of the SR in either normal subjects or subjects with strabismus. The LR muscle showed significant displacement during gaze changes perpendicular to its plane of action. In normal subjects, the LR shifted inferiorly 0.7 mm on upgaze and superiorly 0.8 mm on downgaze (for a total vertical excursion of 1.5 mm), superiorly on abduction 1 mm, and laterally and inferiorly on adduction 0.5 mm (Figs. 7, 8). The inferior rectus (IR) muscle showed significant vertical and horizontal displacement, but only in its plane of action. In normal subjects, the IR shifted medially 1.1 mm and inferi orly 1.0 mm on upgaze and laterally 1.3 mm and superiorly 1.3 mm on downgaze (for a total vertical excursion of 2.3 mm and horizontal excursion of 2.4 mm) while showing almost no shift in position, either horizontally or vertically, during adduction or abduction (Figs. 7, 8).

More posterior image planes showed a similar pattern in the direction of displacement of the EOM paths during changes in gaze, only varying from plane $-1$ by showing a different magnitude of displacement. The largest displacement inward of the horizontal EOMs occurred in plane 3 (MR, 0.6 mm; LR, 0.4 mm) and displacement outward occurred in plane 0 (MR, 0.7 mm; LR, 0.6 mm). The largest displacement inward of the vertical EOMs occurred in plane 0 for the

![Figure 7](image-url)  
**Figure 7.** Average positions of centroids of all rectus extraocular muscles (relative to right orbital center and viewed as if facing the subject) for normal subjects in plane $-1$ (3 mm anterior to the globe–optic nerve junction) in upgaze, downgaze, abduction, abduction, and primary position. SR = superior rectus muscle; LR = lateral rectus muscle; IR = inferior rectus muscle; MR = medial rectus muscle.

![Figure 8](image-url)  
**Figure 8.** Average positions of centroids of all rectus extraocular muscles (relative to right orbital center and viewed as if facing the subject) for subjects with strabismus in plane $-1$ (3 mm anterior to the globe–optic nerve junction) in upgaze, downgaze, and primary position. SR = superior rectus muscle; LR = lateral rectus muscle; IR = inferior rectus muscle; MR = medial rectus muscle.

TABLE 4. Rectus Pully Position Displacements in Subject TL

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Right Orbit</th>
<th>Left Orbit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medial rectus</td>
<td>2.6 mm inferior</td>
<td>4.2 mm inferior</td>
</tr>
<tr>
<td>Superior rectus</td>
<td>2.0 mm medial</td>
<td>2.6 mm medial</td>
</tr>
<tr>
<td>Lateral rectus</td>
<td>3.7 mm superior</td>
<td>5.1 mm superior</td>
</tr>
<tr>
<td>Inferior rectus</td>
<td>2.7 mm lateral</td>
<td>5.0 mm lateral</td>
</tr>
</tbody>
</table>
SR (1.9 mm) and plane −1 for the IR (1.3 mm), and displacement outward for both occurred in plane −1 (SR, 1 mm; IR, 1 mm).

The EOM sideslip in plane −1 for each EOM is summarized in Table 2. There were no statistically significant differences between the normal subjects and the subjects with strabismus in displacement of EOMs for various positions of gaze (P > 0.05). This finding indicates that rectus EOM sideslip is generally small and consistent in normal subjects and subjects with strabismus.

Although as a group the subjects with strabismus did not show abnormal EOM positions or sideslip, two subjects with strabismus had clearly heterotopic EOM positions. The first subject, JM, was a 60-year-old man with a 4-year history of intermittent vertical diplopia that manifested itself primarily when he was fatigued. Examination revealed an intermittent left hypertropia that was greater in downgaze and on right gaze, a superimposed V-pattern intermittent exotropia, and a marked inability to depress the left globe in adduction. Double Maddox rod testing revealed 5° of relative excyclotorsion. Left superior oblique palsy was diagnosed clinically on the basis of the left hyper-
tropia, left superior oblique underaction, and exotropia.\(^\text{17}\) In primary position, the area centroids of several of this subject’s rectus EOMs were displaced more than three standard deviations from normal. Notable were bilateral inferior displacement of the LRs and bilateral inferior and lateral displacement of the SRs, both exceeding three standard deviations in plane –1, plane 0, and plane 1 (Fig 9). No normal subject, and only one other subject with strabismus, had rectus EOM displaced from the mean normal position more than three standard deviations in any scan plane. The inferred stability of JM’s pulleys was normal during changes in gaze. The maximum midorbital cross-sectional area of JM’s right and left superior oblique (SO) muscles were similar in primary gaze (0.207 cm\(^2\) versus 0.212 cm\(^2\)). The contractile changes in maximum SO cross-section from upgaze to downgaze, 0.055 cm\(^2\) for the right SO and 0.068 cm\(^2\) for the left SO, are both within the normal range as previously described.\(^\text{18,19}\) An Orbit computer simulation, with pulleys positioned to simulate JM’s abnormal mean muscle positions (Table 3), exhibited JM’s V-pattern exotropia and the increasing left hypertropia in downgaze and right gaze, but it did not account for his left hypertropia in primary gaze. We reasoned that 4 years of intermittent hypertropia had stretched the left IR. Increasing its assumed resting length by 4 mm yielded a good simulation of JM’s clinical condition (Fig. 10).

The second subject with strabismus, TL, a 30-year-old man with a history of early childhood exotropia, had been treated with orthoptics and patching. Examination revealed right hypertropia, large A-pattern exotropia, and excessive depression of both eyes during adduction. Subject TL’s pattern of strabismus was surprisingly unaltered by bilateral SO tenotomy, motivating MRI examination. Rectus EOM positions in primary position are summarized for subject TL in Figure 11. All four rectus EOMs in the left orbit, as well as the SR and LR of the right orbit, were found to be more than three standard deviations from normal in several image planes, so that the array of right pulley positions appeared rotated clockwise and the left orbit counterclockwise, as viewed facing the subject. The inferred stability of TL’s EOM pulleys did not differ from normal. An Orbit computer simulation, with pulleys positioned to simulate TL’s abnormal mean muscle positions (Table 4), closely simulated the A-pattern exotropia and increasing right hypertropia in right gaze but did not account for the right hypertropia in primary gaze. Once again, a coincidental right IR abnormality (created by lengthening the right IR muscle 2.5 mm) was used to simulate TL’s right hypertropia in primary gaze, completing the computer simulation of his clinical condition. The simulation captures the observed A-pattern and most of the variation in the incomitant right hypertropia (Fig. 12).

Additional computer simulations were performed using Orbit to predict the sensitivity of binocular alignment to several rectus EOM path abnormalities. These computer simulations were performed to determine whether rectus pulley positions within normal variation, as defined by the measured standard deviations for normals, were compatible with normal binocular alignment. Worst cases were considered in which all pulley locations were altered by the measured standard deviations for normals, were compatible with normal binocular alignment. Worst cases were considered in which all pulley locations were altered to produce synergistic effects on binocular alignment. First, the array of the centers of all the rectus EOMs was rotated two standard deviations clockwise from normal for the right orbit and counterclockwise for the left orbit, or vice versa, simulating the types of symmetrical malrotation of both orbits. In both cases, the effect on primary position alignment was negligible, and the maximum deviation from orthotropia in extreme gaze (30° eccentric) was 10\(^\circ\) (5.7°) of horizontal deviation, 5\(^\circ\) (1.7°) of vertical deviation, and 4° of cycloversion, all probably within the range of binocular fusion.\(^\text{17}\) Simulations using malro-
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FIGURE 11. Average positions of centroids of the rectus extraocular muscles for primary gaze in all coronal planes (relative to right orbital center) in normal subjects and in subject TL. The left orbit has been reflected to the configuration of the right orbit. Error bands = ± 2 SD. Positions demarcated with a white dot in the center are more than 3 SD from the average normal position. (open circles) Normal subjects. (closed triangles) Subject TL right eye. (closed boxes) Subject TL left eye.

tations of three standard deviations also predicted a negligible effect on primary position alignment but deviation in extreme gaze increased to 14° (8°) horizontally, 5° (2.9°) vertically, and 6° of cyclotorsion, amounts challenging or overwhelming motor fusion.17 Finally, moving EOM pulleys three standard deviations radially inward (simulating a small orbit) or radially outward (simulating a large orbit) had an insignificant effect on binocular alignment in all gaze positions.

DISCUSSION
These high-resolution MRI data suggest that the positions of rectus EOM pulleys are defined precisely. Several additional simulations help estimate the range of pulley positions consistent with normal ocular motor function. Displacement of the pulleys radially from the orbital center does not appear to influence binocular alignment significantly. In contrast, displacement of the pulleys perpendicular to their planes of action by only a few millimeters may increase the risk of strabismus by altering EOM pulling directions to a degree that might exceed the compensatory capabilities of the central nervous system.20 In extreme cases, such as with subjects JM and TL, grossly abnormal rectus pulley positions seem likely to be the primary cause of incomitant strabismus.
These findings are consistent with findings in children with craniosynostosis syndromes (i.e., Apert, Crouzon, and Pfeiffer syndromes), who typically have marked V-pattern exotropia, small, laterally rotated orbits, and abnormally located extraocular muscles. In subject TL, all of the rectus EOMs were displaced clockwise in the right orbit and counterclockwise in the left orbit, giving rise to an A-pattern exotropia in addition to a right hypertropia.

In subject JM, only the SR and LR were displaced significantly from normal in both orbits. This subject had acute onset of diplopia and was diagnosed with left SO palsy because of left hypertropia that increased on adduction of the left eye. Dynamic MRI of this subject, however, did not confirm this diagnosis (using MRI criteria previously described). Alternatively, we propose that congenitally anomalous rectus pulley positions may have given rise to inconstant forces within the range of motor compensation during the subject’s youth. This compensation may have been disrupted by an event such as inflammation or trauma to the left IR, which would have unmasked the V-pattern exotropia with inconstant hypertropia and given the appearance of left SO palsy.

A particularly severe muscle path abnormality has been documented recently by MRI in the “heavy eye syndrome.” In this syndrome, esotropia and hypertropia develop in eyes with extreme axial myopia. Krizok and colleagues have shown that such patients exhibit a large inferior displacement of the path of the LR muscle, with Orbit simulations consistent with the idea that most of the normal abducting action of the LR is thus converted to depression. This phenomenon is presumably associated with dehiscence of the lateral levator aponeurosis, allowing the LR pulley to shift inferiorly.

No significant differences in mean rectus pulley sideslip during gaze shifts were found here between normal subjects and subjects with strabismus. Previous work in four subjects showed that the EOM bellies all tend to bow in toward the center of the orbit during contraction and bow out from the center of the orbit during relaxation while maintaining relatively fixed during changes of gaze perpendicular to their plane of action. The largest bowing movements, almost 4 mm, were measured for the SR and the smallest bowing movements, 1.5 mm, were measured for the IR. In the current, much larger study, all rectus EOMs clearly showed similar bowing, but the horizontal EOMs showed much smaller and more posterior bowing compared with the vertical EOMs. This outward bowing of relaxed EOMs may be attributable to the radial forces generated by the pulley slings, which extend from the pulley rings located at the equator of the spheres as far posteriorly as the globe—optic nerve junction. The static tension within the pulley slings may pull the relaxed EOMs outward toward the orbital wall. Apparent inward bowing of the horizontal rectus muscles deep in the orbit in image plane +3 may represent an artifact of the contractile increase in muscle-cross-sectional area in a region constrained on the orbital sides by contact with the bony orbital walls, forcing the contracting muscles’ area centroids centrally.

Histologic studies are consistent with the current findings in that the MR pulley contains the least fibroelastic and smooth muscle tissue and should have the most rigidity, whereas the SR pulley has the least fibroelastic tissue and should be least rigid. The LR displaced little with horizontal gaze changes and approximately 1 mm opposite to the direction of vertical gaze changes (i.e., up during downgaze and down during upgaze). These movements increase LR path length and are opposite to predictions of all versions of the shortest path hypothesis. One possible explanation for this increase in path length is that intermus-
cular connective tissue posterior to the center of rotation may stabilize the LR relative to the globe. An alternative explanation points to the dense lateral levator aponeurosis, coupling the LR to the SR (Fig. 5, plane 0). The lateral levator aponeurosis contains dense collagen, elastin, and smooth muscle. During upgaze, as the SR bows downward 1.6 mm from its position in primary gaze, the lateral levator aponeurosis and posterior Tenon’s fascia sag inferiorly toward the LR, allowing, we propose, the LR to sag down as well. Conversely, as the SR bows superiority 1 mm on downgaze, the LR may be pulled superiority by the lateral levator aponeurosis. The displacement of the LR superiority 1 mm during abduction might be related to the abducting action of the SR, in that under increased tension, it might pull superiority on the LR pulley by the lateral levator aponeurosis.

The IR also exhibited unexpected behavior. It remained relatively immobile during changes in horizontal gaze. During changes in vertical gaze, the IR bowed superiority during contraction and inferiorly during relaxation as expected but also showed significant horizontal displacement as well. The IR shifted medially 1.1 mm during upgaze and laterally 1.3 mm during downgaze. A possible explanation could be intermuscular coupling between the IR and the inferior oblique (IO) muscle, termed Lockwood’s ligament. We propose that the IO, contracting in upgaze, draws its belly medially, pulling the relaxed IR medially as well. During downgaze, relaxation of the IO and contraction of the IR could cause lateral displacement of the inferior rectus.

In summary, the current study supports previous work showing that rectus EOM paths undergo small displacements during changes of gaze. The current study reveals some new complexities of the fibroelastic pulley system. The normal position and sideslip of the fibroelastic pulleys have been defined. Two subjects were discovered to have anomalous rectus EOM pulley positions that could form the structural basis for incomitant strabismus. In subject TL, both orbits appear rotated in a manner predicted by biomechanical simulation to produce an A-pattern strabismus. In subject JM, anomalous location of the SR and LR, coupled with an inferior rectus abnormality, simulated the clinical pattern of SO palsy. Anomalous pulley position can explain some cases of poor response to strabismus surgery for clinical oblique muscle dysfunction.

The system of orbital fibromuscular connective tissues provides mechanical coupling among the EOMs. The current MRI evidence indicates specific roles for coupling between LR and SR muscles and between IR and the IO muscles.

**Key Words**

binocular alignment, extraocular muscles, magnetic resonance imaging, pulleys, strabismus

**References**


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