Measurement of Recti Eye Muscle Paths by Magnetic Resonance Imaging in Highly Myopic and Normal Subjects

Thomas H. Krzizok¹ and Bernd U. Schroeder²

PURPOSE. To analyze the path of extraocular muscles (EOMs) quantitatively in highly myopic subjects with and without restricted eye motility versus control. To elucidate the cause of the acquired motility disorder in patients with high myopia.

METHODS. Thirty-three orbits were imaged using a Magnetom or Siemens Vision (Siemens, Erlangen, Germany; both 1.5 Tesla) MRI (magnetic resonance imaging) scanner. Coronal T1-weighted, spin-echo images were obtained with repetition time of 550 msec and echo time of 15 msec. Subjects had to fixate in different positions of gaze. Orbits of three patient groups were analyzed: group 1 (n = 14), patients with high axial myopia and restricted eye motility (average axial length, 31.4 mm; refractive error more than −15 D); group 2 (n = 8), subjects with high axial myopia and normal eye motility (average axial length, 29.2 mm); control group (n = 11), emmetropic subjects with normal eye motility (average axial length, 23.6 mm).

RESULTS. Highly myopic patients showed significant displacements of recti EOMs in comparison with control subjects. Mean displacements, measured in the plane 3 mm anterior to the globe-optic nerve junction in primary gaze, were in group 1, lateral rectus (LR) 2.9 mm (2.5 downward, 1.4 medial), medial rectus (MR) 1.3 mm downward and in group 2, LR 1.4 mm (1.3 downward, 0.6 medial) and MR 1.2 mm downward. In groups 1 and 2 the inferior rectus (IR) was displaced 1.3 mm medially and upward. In both groups of myopic patients the superior rectus (SR) was displaced 1.5 mm medially and downward.

CONCLUSIONS. In patients with high axial myopia, displacements of all recti EOMs can be detected by MRI. Displacements of SR, MR, and IR were very similar in groups 1 and 2 versus control. LR displacement into the lateral and inferior quadrant of the orbit was greatest in patients with restricted eye motility. Thus, LR displacement is probably the major pathophysiological factor for the restrictive motility disorder in high myopia. EOM dislocations can be explained by myopia-associated alterations in the orbital connective tissues confining EOM positions in relation to the orbital wall. (Invest Ophthalmol Vis Sci. 1999;40:2554–2560)

Adult patients with unilateral or bilateral high (axial) myopia may acquire a typical restrictive motility disorder, resulting in esotropia and often hypotropia. Many different theories on the underlying cause of this restrictive disease can be found in the literature. Duke–Elder and Wybar¹ suggested structural changes in the ocular muscles—that is, a reduced number of muscular fibers included in fibrous tissue in the lateral rectus (LR). In some textbooks²,³ the deviation, especially the hypotropia, is characterized by the name heavy eye syndrome. Bagolini et al.⁴ inferred from echography, myopathic paralysis of the LR due to pressure from the lateral orbital wall. Demer and von Noorden⁵ found that rotation was limited due to contact between the posterior aspects of the elongated globe and the bones of the orbital apices. Herzau and Ioannakis⁶ observed during surgery an abnormal LR path but could not confirm this finding before surgery by computed tomography or magnetic resonance imaging (MRI).

More recent theories concerning restrictive strabismus take into account new aspects of the functional anatomy of the orbit.⁷ Thus, it has been shown by Demer et al.⁷ that the EOMs pass through connective tissue sleeves in the posterior Tenon’s fascia that constrain muscle paths during gaze shifts and act as functional origins of the muscles. These tissues, termed pulleys, structurally and functionally include many of the orbital connective tissues known as check ligaments, Lockwood’s ligament, intermuscular membranes, or orbital septa.⁷–¹⁰ Pulleys are composed of collagen, elastin, and smooth muscle and are connected to the orbital bones and to one another by connective tissue bands.⁷ In normal orbits, location of the pulleys is highly uniform. However, displacements of normal pulley positions of some millimeters have been found in cases with incomitant (A- or V-pattern) strabismus.¹¹

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Recently, Krzizok et al.\textsuperscript{12} described a significant inferior dislocation of the lateral rectus (LR) based on MRI scans. Our purpose was to analyze the path of all recti extraocular muscles (EOMs) quantitatively in highly myopic subjects with and without strabismus versus control by means of fixation-controlled MRI. We were looking for a specific pattern of myopia-associated EOM displacement related to restricted motility. Results of our study should not only contribute to a better understanding of the motility disorder associated with high myopia but should also to provide further anatomic data for improvement of strabismus surgery in these patients.

**MATERIALS AND METHODS**

We examined prospectively 22 orbits of 15 patients with unilateral or bilateral high axial myopia and 11 orbits of 8 emmetropic volunteers without strabismus. All subjects had given their informed consent according to a protocol approved by the institutional review board for the protection of human subjects (Declaration of Helsinki). Routine ophthalmologic and orthoptic examinations and A- and B-scan echography were performed in all subjects to determine axial length of the globes (Ocuscan; Alcon, Fort Worth, TX.). A simultaneous and alternate prism cover test at distances of 5 m and 0.33 m, in 25° up-, down-, and side gaze was performed in all patients with central (foveolar) fixation, before surgery and after surgery at 1 week and at least 3 months. The range of ocular rotation was measured with a synoptometer. Forced duction tests were performed during surgery to determine horizontal, vertical, and torsional motility.

Highly myopic patients unable to fixate in primary gaze because of very limited abduction or elevation had to be excluded from this analysis. Subjects were divided into three groups according to axial length of the globes and degree of motility restriction ( clinical characteristics are described in Table 1): control group (11 orbits): emmetropic subjects (spherical shaped globes with axial length of 21–24 mm) and normal eye motility (abduction

### TABLE 1. Clinical Data

<table>
<thead>
<tr>
<th>Number of Orbits</th>
<th>Age (y)</th>
<th>Gender (F/M)</th>
<th>Refraction (D)</th>
<th>Axial Length of Globe (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>14</td>
<td>55 ± 13</td>
<td>6/3</td>
<td>−19.2 ± 5.9</td>
</tr>
<tr>
<td>Group 2</td>
<td>8</td>
<td>46 ± 22</td>
<td>3/3</td>
<td>−17.1 ± 6.7</td>
</tr>
<tr>
<td>Control</td>
<td>11</td>
<td>39 ± 18</td>
<td>4/4</td>
<td>+0.25 ± 0.7</td>
</tr>
</tbody>
</table>

Data are mean ± SD. Subjects: group 1, n = 9; group 2, n = 6; control group, n = 8.

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**Figure 1.** Scheme of multiple contiguous coronal MRI planes of a normal orbit. Each image plane is 3 mm thick. The plane containing the globe–optic nerve junction is designated plane 0. Measurements of the paths of EOMs were performed in planes 0 and −1. (A) Method applied in the present study: coronal scan plane and orbital axis (o) are 116° apart. The visual axis (b) is 5° nasally to the primary gaze position (p). (B) Examinations of Miller\textsuperscript{13} and Clark et al.\textsuperscript{15}. Coronal scan planes are perpendicular to the orbital axis (o). Patient’s head is turned in the MRI scanner 26° to the opposite side. Visual axis (b’), shown here in 26° abduction, is parallel to the orbital axis (o). Visual axis (b”) and primary gaze position (p) are 26° apart.
\[ \geq 40^\circ \text{ and elevation } \geq 30^\circ \}; \text{ group 1 (14 orbits): highly myopic subjects (axial length } > 27 \text{ mm) with limited abduction (} < 40^\circ \) and elevation (} < 30^\circ \); \text{ and group 2 (8 orbits): highly myopic subjects (axial length } > 27 \text{ mm) without restricted motility (abduction } \geq 40^\circ \text{ and elevation } \geq 30^\circ \).

**Magnetic Resonance Image Parameters**

All orbits were imaged using a Magnetom (SP 63) or Vision MRI scanner (Siemens, Erlangen, Germany; both 1.5 Tesla) with a head coil. For each orbit, an axial localizing image was taken at low resolution to select the optimal anteroposterior location, and the globe–optic nerve junction (Fig. 1). Modest resolution coronal T1-weighted, spin–echo images were obtained with repetition time, 550 msec; echo time, 15 msec; field of view, \( 21 \times 21 \text{ cm} \); pixel matrix, \( 256 \times 512 \); three acquisitions, slice thickness, 3 mm; and distance factor, 0.25. The number of coronal planes was 10 to 15 for each orbit. Pixel resolution of \( 820 \times 410 \text{ }\mu\text{m} \) was achieved. During the scanning procedure, the head of the subject was fixed. Subjects had to fixate (with the less restricted eye) for 4 minutes in different positions of gaze. 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. If strabismus was present, each eye had to be scanned separately to maintain primary gaze. The nonfixating eye was occluded.

**Geometric Analysis of MRI Scans**

The position of the centroid of the globe–optic nerve junction in coronal MRI scans (plane 0 in Fig. 1) was used to determine the exact eye position (e.g., primary gaze, upgaze, downgaze, abduction, and adduction). Tertiary positions were not necessary for this study. The geometric analysis of contiguous MRI scans was performed according to the description of Miller,\(^{13}\) except that coronal planes were not orthogonal (perpendicular) to the orbital axis—that is, 26° tilted to the coronal plane—but were perpendicular to the visual axis of each eye in primary gaze.

The original MRI films were scanned (Scanmaker E6; Mikrotek, Hsinchu, Taiwan) in tagged-image file format and analyzed quantitatively using NIH Image with personal computers (Macintosh; Apple, Cupertino, CA) or the same program, named PC-image, with IBM-compatible personal computers and Windows 95 (Microsoft, Redmond, WA). Both software programs were written by Wayne Rasband (National Institutes of Health) and are available as public domain programs on the Internet (anonymous - ftp://zippy.nimh.nih.gov). Images of left orbits were reflected digitally (flipping horizontally) to the orientation of a right orbit. Each image was calibrated according to the reference scale on the MRI scans: 10 mm equaled 38 to 55 pixels. Positional errors during the MRI examination were accounted for. Rotation in the coronal plane (head tilt) was checked and corrected by rotating the image to align the interhemispheric fissure of the brain with the scanner-defined vertical meridian.

A Cartesian coordinate system was used in the orbit. The \( z \)-axis consisted of a line, connecting the geometric centers of all coronal planes. After outlining the bony orbit of each scan with a trackball, the geometric middle, that is, the centroid, was determined (function XY Center of NIH Image software). This centroid is the reference or zero point for all measured coordinates of EOMs (Fig. 2). The plane containing the globe–optic nerve junction was designated plane 0. More anteriorly located planes have a negative prefix (Fig. 1). If the MRI scans did not fit exactly with these planes, data for plane 0, \(-1 \), and so on, were found by interpolation. The position of recti EOMs was determined by their centroid in this coordinate system.

A simulation with the computer model Orbit 1.6 of Miller and Shamaeva\(^{14}\) was used to examine the functional results of the different morphologic findings.

**RESULTS**

Table 2 shows the average position of centroids of EOMs for all three groups.
**TABLE 2. Position of Recti EOMs in Primary Gaze**

<table>
<thead>
<tr>
<th>Plane 0</th>
<th>MR</th>
<th>LR</th>
<th>SR</th>
<th>IR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>12.4 ± 0.9</td>
<td>−1.7 ± 1.0</td>
<td>−9.5 ± 1.2</td>
<td>3.6 ± 1.1</td>
</tr>
<tr>
<td></td>
<td>(0.377)*</td>
<td>(0.049)</td>
<td>(&lt;0.001)</td>
<td>(0.004)</td>
</tr>
<tr>
<td>Group 2</td>
<td>12.6 ± 0.6</td>
<td>−1.6 ± 1.8</td>
<td>−10.3 ± 0.5</td>
<td>3.8 ± 0.8</td>
</tr>
<tr>
<td></td>
<td>(0.362)*</td>
<td>(0.055)</td>
<td>(0.018)</td>
<td>(0.076)</td>
</tr>
<tr>
<td>Control</td>
<td>12.5 ± 0.6</td>
<td>−0.4 ± 1.3</td>
<td>−10.9 ± 0.6</td>
<td>1.9 ± 1.3</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Plane −1</th>
<th>MR</th>
<th>LR</th>
<th>SR</th>
<th>IR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>13.1 ± 0.9</td>
<td>−1.5 ± 0.8</td>
<td>−10.8 ± 1.6</td>
<td>3.3 ± 1.3</td>
</tr>
<tr>
<td></td>
<td>(0.432)</td>
<td>(0.014)</td>
<td>(0.011)</td>
<td>(0.018)</td>
</tr>
<tr>
<td>Group 2</td>
<td>13.2 ± 0.5</td>
<td>−1.4 ± 1.7</td>
<td>−11.6 ± 0.7</td>
<td>3.3 ± 1.1</td>
</tr>
<tr>
<td></td>
<td>(0.440)</td>
<td>(0.067)</td>
<td>(0.097)</td>
<td>(0.053)</td>
</tr>
<tr>
<td>Control</td>
<td>13.1 ± 0.8</td>
<td>−0.2 ± 1.6</td>
<td>−12.2 ± 0.8</td>
<td>2.0 ± 1.6</td>
</tr>
</tbody>
</table>

Data are mean millimeters from the orbital center ± SD. Number of orbits: group 1, n = 14; group 2, n = 8; control group, n = 11.
* Statistical significance was determined between groups 1 and 2 and the control group.

**Control Group**

Recti EOMs had a regular, nearly symmetrical location in relation to the geometric center of the bony orbit. The medial rectus (MR) and LR were insignificantly shifted below the horizontal meridian, and the inferior rectus (IR) and superior rectus (SR) were insignificantly located medially to the vertical meridian. The entrance of the optic nerve into the globe in primary gaze was 2.9 mm medial and 1.3 mm superior to the orbital center (Table 2 and Fig. 2).

Figure 3 describes the muscle path of the significantly displaced LR of a subject of group 1 in contiguous MRI scans from the posterior to the middle right orbit. The measured inferior displacement of LR relative to the MR (difference of y-value of MR to LR) in different anteroposterior positions of the orbit constantly decreased from the anterior MRI scan in Figure 3 to the posterior MRI scan. This was primarily because of the displaced curved path of the LR, which shifted from the normal insertion site into the lateral and inferior quadrant of the orbit and proceeded to the origin and secondly according to the conical path of recti EOMs toward the apex of the orbit.

Recti EOMs, the optic nerve, the entrance of the optic nerve into the globe, the superior oblique muscle, the levator palpebrae superioris and the lateral levator aponeurosis can be reliably imaged in the orbit by means of MRI. Even with the highest quality orbital MRI, it is not yet consistently possible to image the pulleys of recti EOMs directly. This applies to the highest quality orbital MRI, it is not yet consistently possible to reliably image the pulleys of recti EOMs directly. This applies to the

**Groups 1 and 2**

In patients with high myopia deviations from the physiological, muscle position and path were evident. Figure 4 and Table 2 show in groups 1 and 2 a similar type and amount of dislocation medialward of SR and IR and downward of MR. In group 1, the mean displacement of SR in plane −1 (3 mm anterior to the globe–optic nerve junction) in primary gaze was 1.5 mm (0.6 mm downward and 1.3 mm medial) and was also 1.5 mm in group 2 (0.8 mm downward and 1.3 mm medial). Mean displacement of IR in group 1 was 1.3 mm (0.6 mm upward and 1.2 mm medial). In group 2 displacement of IR in plane −1 in primary gaze was also 1.3 mm (0.4 mm upward and 1.2 mm medial). The MR was displaced inferiorly 1.3 mm in group 1 and 1.2 mm in group 2.

On the contrary, LR was significantly more displaced in group 1 (restricted motility) compared with group 2 (unrestricted motility). In plane −1, mean displacement of LR in group 1 was 2.9 mm (2.5 mm downward and 1.4 mm medial), which was significantly (Student’s t-test) more than in group 2 with only 1.4 mm (1.3 mm downward and 0.6 mm medial). The amount of dislocation of recti EOMs did not change significantly from plane −1 to plane 0: The dislocation decreased according to the conical path of recti EOMs toward the apex of the orbit. The dislocations in planes −1, 0, and 1 were always distinguishable from normal. The pattern of dislocation in planes 1 and 0 was not different from that in plane −1. In Table 2 the level of significance (Student’s t-test) is shown for x/y coordinates of all recti EOMs of group 1 and 2 in comparison with control. Between group 1 and 2, x/y coordinates of LR were significantly different (P = 0.036).

In our series, head tilt up to 12° during the scan procedure had to be corrected. Head elevation or depression was present to a lesser extent, up to 6°. Head turn to the right or left shoulder did not exceed 5°.

The positions of recti EOMs in normal subjects in our study compared with the results of Clark et al.4 are shown in Figure 2 and in Table 3. Differences in EOM positions between the two studies can be explained by the different orientation of scan planes used during MRI examination. Whereas Clark et al.15 preferred scan planes perpendicular to the longitudinal axis of the orbit, images in our study were obtained in the classic radiologic coronal plane, which is tilted approximately 26° from the plane used by Clark et al.15 (Fig. 1). Because of the different projection, cross sections of the bony orbit, the vertical recti and the lateral rectus muscles were more oblique to the image plane, resulting in different coordinates of the EOM position. Thus, in our study, EOM coordinates of the LR were measured slightly more laterally
We found a significant relative inferior dislocation of the horizontal recti EOMs were taken into account. In the present study, relative positions of the horizontal recti EOMs were measured more medially in comparison with the measurements by Clark et al. (Fig. 2 and Table 3). The SD in our control group was obviously higher than that in the study of Clark et al. The reason is the better resolution of the MRI scans in their study.

and the MR, SR, and IR coordinates were measured more medially in comparison with the measurements by Clark et al. (Fig. 2 and Table 3). The SD in our control group was obviously higher than that in the study of Clark et al. The reason is the better resolution of the MRI scans in their study.

**DISCUSSION**

In a previous study of 13 highly myopic patients with restriction-induced esodeviations or hypodeviations, Krzizok et al. found an inferior dislocation of the LR relative to the MR in the midorbital region, with a median value of 3.4 mm. The study did not include calculation of the centroids of the recti EOMs in relation to a reference point. In the present study, relative vertical positions of the horizontal recti EOMs were taken into account. We found a significant relative inferior dislocation of the LR in relation to the MR of 1.9 mm only in group 1 (Table 2; mean value of \( y = -1.5 \) [MR] versus \( -3.4 \) [LR]). No significant inferior dislocation of LR relative to MR was present in group 2 (0.8 mm [mean value of \( y = -1.4 \) [MR] versus \( -2.2 \) [LR]), and in the control group (0.7 mm [mean value of \( y = -0.2 \) [MR] versus \( -0.9 \) [LR]). This is consistent with an earlier finding (Krzizok et al.) of inferior dislocation of LR of 3.4 mm in myopes with very restricted abduction and elevation, equivalent to group 1 in the current study.

Fixation-controlled MRI scans showed significant midorbital displacement of all recti EOMs in high myopia (groups 1 and 2). Surprisingly, the displacement of SR and IR medially and the displacement of MR inferiorly was detectable in the same amount in high myopes with (group 1) and without (group 2) restricted motility. The displacement of the superior and the inferior rectus muscles in high myopes without restricted motility is likely the result of the larger globe diameter in myopic patients. Because this displacement is symmetrical and small, no clinically detectable strabismus is associated.

However, inferior displacement of LR was significantly greater in high myopes with restricted abduction and elevation (group 1) compared with group 2. Thus, LR displacement can be considered to be the major pathophysiologic factor for the restrictive motility disorder in high myopia. Computer simulation of group 1 patients by using the Orbit 1.6 Gaze Mechanics Simulation program is consistent with the main clinical deviations: esotropia, hypotropia, and excyclorotation. LR displacement explains the acquired strabismus: the abducting function of the LR is reduced, and an unphysiological depressing and excyclorotating force is created.

The pathophysiology can be explained by stretching in the suspensory tissues that regulate the position of the LR pulley, permitting the pulley to slip inferomedially, and the globe to herniate superotemporally through the suspensory tissues that ordinarily confine the globe to the center of the orbit. Between SR and LR, this superotemporal tissue forms the lateral levator aponeurosis, consisting of dense collagen, elastin, and smooth muscle. However, the histopathologic changes and the trigger for them occurring in this particular anatomic site are not yet known. Demer et al. found in histologic studies that the MR pulley contains the most fibroelastic and smooth muscle tissue, resulting in the highest rigidity compared with the other recti EOMs.

If the inferior displacement of the LR is due to a weakening of the suspensory tissues of the LR pulley, then it would be expected that LR dislocation might be greatest when the LR contracts and increases its tension—that is, in abduction of the myopic globe. This is what we found in the simulation using Orbit 1.6. Clark et al. found in normal subjects a displacement of the LR superiorly 1 mm during abduction and explained this with the abducting action of the SR, which under increased tension in abduction may pull superiorly on the LR pulley through the lateral levator aponeurosis. Unfortunately, it is not possible to implement abnormal globe shape or a dehiscence of the lateral levator aponeurosis into computer simulations of highly myopic patients with the Orbit 1.6 Gaze Mechanics Simulation program. Thus, more fixation-controlled MRI scans in adduction and abduction must be analyzed.

Dislocation of recti EOMs in high myopia without restricted motility (group 2) has not yet been described. Because horizontal and vertical deviations in group 2 were small, the displacements of SR, MR, and IR were probably not a secondary adaptation to the misalignment in adduction and hypotropia. On the contrary, they...
were probably the result of abnormalities of orbital connective tissues (intermuscular membranes, lateral levator aponeurosis, pulleys, and Lockwood’s ligament⁸–¹⁰) in high myopia, resulting in pathologic EOM paths of different extents. Other than Duane’s syndrome, high myopia is the only nontraumatic disease with evident sideslip of EOMs.¹⁶

To have reproducible and comparable MRI scans, it is important to perform measurements in a defined (coronal) plane. A reliable reference point is the center of the bony orbit, but not the mobile optic nerve, which may lead to erroneous measurements. In high myopes, the optic nerve sometimes assumes a variable S shape near the orbital apex. Thus, the location of the optic nerve, even in constant gaze positions, depends on the scan plane. To determine and control the gaze position in the MRI scan procedure by means of the optic nerve position, the same coronal scan plane must be chosen. Most suitable for this purpose is scan plane 0, the globe–optic nerve junction. Although the mobile optic nerve is not a reliable reference point for measurements, the optic nerve in this plane may be helpful to determine the actual gaze position.

**TABLE 3.** Comparison of Recti EOMs’ Position in Primary Gaze and Plane −1 in Normal Subjects in the Study of Clark et al. and in the Current Study

<table>
<thead>
<tr>
<th></th>
<th>MR</th>
<th>LR</th>
<th>SR</th>
<th>IR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>x</td>
<td>y</td>
<td>x</td>
<td>y</td>
</tr>
<tr>
<td>Clark et al., primary gaze⁹ ⁵</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>x</td>
<td>12.1</td>
<td>± 0.4</td>
<td>-11.7</td>
<td>± 0.3</td>
</tr>
<tr>
<td>y</td>
<td>0.1</td>
<td>± 0.7</td>
<td>-12.2</td>
<td>± 0.5</td>
</tr>
<tr>
<td>Clark et al., adduction</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present study, primary gaze</td>
<td>12.2</td>
<td>± 1.6</td>
<td>12.7</td>
<td>± 0.9</td>
</tr>
</tbody>
</table>

Data are mean millimeters from the orbital center ± SD.
* Because of the choice of the coronal scan plane of Clark et al., their technique means that there is a 26° abduction in the primary gaze. Thus the gaze position adduction approaches the physiological primary gaze, as was also used in the present study.

**FIGURE 4.** Average position of centroids of EOMs in coronal plane 0 (top) and −1 (bottom) for primary gaze in the highly myopic patients. Presentation as in Figure 2. Group 1 (first column, n = 14), high myopia with restricted motility; and group 2 (second column, n = 8), high myopia without restricted motility. The 2SD interval (95.4% confidence interval) of recti EOMs in our control group (Fig. 2) is designated by elliptically shaded zones.
position. It is important to position the head properly when quantitatively measuring positions of the EOMs.

Data for the path of recti EOMs of emmetropic subjects without strabismus have been determined by means of CT and MRI by Miller, Clark et al., and Miller and Robinson. In those publications coronal planes were orthogonal to the orbital axis, 26° tilted to a classic radiologic coronal plane (Fig. 1). We preferred exact coronal scan planes, 116° off the orbital axis. This procedure has two advantages: Both orbits can be scanned simultaneously; if coronal scan planes are perpendicular to the orbital axis, this means 26° abduction for the gaze position defined as primary gaze in the study of Clark et al., necessitating that the patient abduct 52° to reach the gaze position abduction. The disadvantage of our method is that the cross section of the LR is oblique to the imaging plane.

The choice of scan plane produces different data for normal subjects (Table 3), because of the projection. Thus, in our study, in plane −1 the LR was measured 0.5 mm more laterally, MR 1.0 mm more medially, IR 2.1 mm and SR 3.4 mm more medially in comparison with the measurements of Clark et al. Their study and ours have the consistent finding that recti EOMs have a stable, invariable path in the middle and posterior orbit in normal subjects.

Unfortunately, the most anterior portions of recti EOMs consist of tendons that are difficult to distinguish in MRI scans from intermuscular connective tissue and sclera. Thus, recognition of recti EOMs more anterior than plane −3 (Fig. 1) is not reliable. Nevertheless, Figure 3 shows a realistic impression of the LR path. Additional information about the LR path was available from former observations made during surgery. Thus, the LR has a displaced curved path, taking its course from the origin, shifting into the lateral and inferior quadrant of the orbit and proceeding to the normal insertion site.

Miller, Clark et al., Miller and Robinson, and Simonsz et al. demonstrated in vivo the stability of position and path of recti EOMs in the middle and posterior orbit during eye movements, allowing displacements only in the tendinous anterior part of recti EOMs. The stability of the recti EOMs' path is maintained by the pulleys, which encompass the anatomic structures previously known as check ligaments, and probably the so-called intermuscular membranes as well. Demer et al. described in a patient with Marfan syndrome deficient fibrillin in recti EOMs' pulleys, resulting in instability of the MR pulley in one patient in different gaze positions.

Thus, in the future it seems it would be very useful to examine those motility disturbances by means of standardized, high-resolution MRI, in which the orbital connective tissue is possibly altered. Another therapeutic step is to find a surgical treatment that can normalize a dislocated EOM or the pulley of an EOM.

Acknowledgments

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References