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Functional Anatomy of Muscle Mechanisms Compensating Vertical Heterophoria

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Abstract

PURPOSE: Magnetic resonance imaging (MRI) of extraocular muscle function was used to evaluate the role of newly-recognized mechanisms underlying compensation of large heterophoria by vertical fusional vergence (VFV).

Design: Prospective case series.

METHODS: At one academic center, 8 adults with large hyperphoria and super-normal VFV underwent MRI during monocular and binocular fixation of a centered, near target. Contractility of the rectus and the superior oblique (SO) extraocular muscles in the hypertropic and hypotropic eyes was determined from changes in posterior partial volume (PPV).

RESULTS: Five of 8 patients could sustain binocular fusion in the scanner; in these patients, VFV corrected about 5° misalignment, about five-fold greater than normal VFV. Vertical strabismus was mainly compensated by significant contractility of the lateral more than medial compartment of the inferior rectus (IR) in both eyes ($P<0.005$). The superior rectus (SR) and inferior obliques had no significant contractile contribution, although the hypotropic SO relaxed significantly. The IR lateral compartment and SR medial compartment significantly co-relaxed when binocular fusion was attained from monocular target fixation ($P<0.01$).

CONCLUSIONS: While VFV protects patients from small muscle imbalances over the lifespan, even enhanced VFV may be inadequate to avert diplopia. Compensation of hyperphoria by VFV is accomplished mainly by IR muscle relaxation in the hypotropic eye, principally in its selectively innervated lateral compartment, while the SO contributes little. Fusion involves compartmentally-

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selective co-relaxation in hypotropic eye vertical rectus muscles. Our findings suggest a physiologic basis to prefer therapeutic surgical weakening of the medial IR in the hypotropic eye.

Introduction

Diplopia is a common symptom that brings patients to the ophthalmologist. Its binocular form is, by definition, caused by strabismus. Diplopia is diagnosed in 2.2% of Medicare beneficiaries[1]. Vertical diplopia due to vertical binocular misalignment causes about two-thirds of new onset diplopia in adults over age 40 years in California[2], and one third in older patients in Japan[3]. The two commonest causes of adult diplopia are sagging eye syndrome (SES) in 31%, and superior oblique (SO) palsy in 10% of cases[2].

However, it is notable that acquired binocular diplopia in adults is much less common than the potential causes of it. For example, nearly all older adults exhibit some age-related degeneration – sag – of the suspensions of their rectus muscle pulleys, yet only a minority experience diplopia as a result[4]. The most common presentation of SES is age-related distance esotropia (ARDE), also known as divergence paralysis esotropia[4]. Gradually progressive esophoria usually precedes symptomatic ARDE, which becomes clinically problematic only when the esophoria increases to exceed the capacity of fusional divergence to compensate it[5]. Horizontal fusional vergence is thus a defense against diplopia.

It has long been explained that many cases of otherwise idiopathic superior oblique (SO) palsy presenting after childhood represent decompensation of congenital SO palsy, even well into the sixth decade of life[6]. Congenital origin of SO palsy is typically inferred from super-normal vertical binocular fusional amplitudes[6]. However, while only about half of such cases actually exhibit SO muscle atrophy and diminished contractility on multipositional magnetic resonance imaging (MRI) that is the objective hallmark of SO palsy, the typical clinical evolution of such cases is insidiously progressive and lacks any recognizable etiologic event[7]. Like ARDE and cyclovertical strabismus associated with asymmetrical lateral rectus (LR) muscle sag in SES[4], the vertical alignment imbalance becomes symptomatic only when its magnitude increases to exceed the limit of compensatory fusional vergence, and the tiny range of sensory fusion[8] within Panum's area[9]. Vertical fusional vergence (VFV) is thus the major defense against diplopia, but the physiological basis of this defense mechanism is poorly understood.

It is at least generally recognized that normal maximal VFV amplitudes are a good deal smaller than horizontal fusional vergence amplitudes[10], the former being typically 2–3Δ[11,12] compared with the latter typically averaging about 35Δ for near convergence[13]. The amplitude of normal VFV is greater at near than at distance[8,14]. Beyond this, nearly everything about the mechanisms of VFV is controversial. Historically, the muscles implementing VFV have been inferred from patterns of externally observed eye movements. Analysis of the relationship between torsional and vertical eye movements has been used in context of customary concepts of muscle actions to infer mechanisms of VFV acting in normal subjects who developed experimental vertical phorias after adapting to progressively stronger monocular vertical prisms[15]. Based on video observations, Enright proposed that VFV is implemented exclusively by the SO muscles[16], and supported this proposal by an

afterimage study[17]. However, it was later pointed out that Enright's postulated SO mechanism is inconsistent with the finding that the associated torsional movements are independent of horizontal gaze angle, which does not correspond to the geometric variation in SO action[18]. Irsch *et al.* used scleral magnetic search coils to study torsion during VFV, and argued that Enright was nevertheless correct in attributing VFV to the oblique muscles[15]. However, other investigators found that during virtual reality viewing, neither conjugate torsion nor torsional vergence was correlated with normal VFV[8], and the torsion associated with VFV varied idiosyncratically rather than systematically among individual subjects[19].

Similar analysis of vertical and torsional eye movements during VFV has been performed in patients with large hyperphoria due to SO palsy[20]. In 10 such patients, torsion during vertical vergence was quite diverse[20], motivating the authors to infer three different mechanisms operating in different patients: sometimes mainly the oblique muscles; or sometimes mainly the vertical rectus muscles; and even sometimes the paretic SO in the hypertropic eye along with the contralateral superior rectus (SR)[17]. This, of course, means that kinematic analysis of torsional and vertical eye movements based on traditional concepts may compel the conclusion that potent VFV is implemented by a paralyzed SO, constituting at best a paradox, and at least undermining credibility of inferences about muscle actions derived from observing eye movements.

Another method is now available to study muscle function during eye movements. MRI allows direct observation of function in individual muscles maintaining static eye positions, indicating contractility by increase in their maximal cross sectional area[21] and volume[22]. Analysis of muscle cross sections by MRI shows atrophy and reduced thickening in the fields of activation of denervated rectus muscles[23,24] and the normal and paretic SO[25–30] and inferior oblique (IO) muscles[27,31,32].

A recent anatomical discovery expands the repertoire of extraocular muscle actions, potentially providing a solution to the failure of conventional mechanical notions to explain the combined torsional and vertical movements observed during VFV. It has emerged that most the muscles are compartmentalized into selectively-controlled subunits that, for example, enable the rectus muscles to exert vertical and torsional actions[33]. Because human rectus tendons are quite wide[34–36], the fibers along their breadth insert at substantially differing scleral sites that have correspondingly different oculorotary actions. The motor nerve innervating each horizontal rectus muscle divides into superior and inferior branches supplying separate intramuscular regions[37,38]. These compartments contain parallel fibers[39,40] that behave with considerable mechanical independence[41,42]. The inferior rectus (IR) muscle is innervated diffusely by one intramuscular motor nerve branch, but its lateral third is innervated additionally but selectively by a separate nerve branch[38]. For example, the lateral rectus (LR) muscle exhibits differential compartmental contractility during ocular counter-rolling[43] and normal VFV[44], but not in convergence, where the medial rectus (MR) not only exhibits it, but does so differently from conjugate adduction[45]. During VFV induced in normal subjects by monocular near viewing through base up prism, the SO, in both aligned and infraducting eyes, exhibits differential compartmental contractility, but with

paradoxical vertical effects suggesting complex muscle interactions offsetting torsion produced by differential compartmental contraction in the LR and IR muscles[44].

The present study was conducted in context of the forgoing methodological and anatomical innovations, aiming to apply high resolution MRI as an objective measure of contractile function analyzed in separate compartments of the extraocular muscles as they compensate for latent vertical strabismus. We chose to study patients whose vertical heterophorias had increased to magnitudes greatly exceeding normal in order to optimize the chances of detecting the special muscle mechanisms that presumably prevent vertical diplopia in the larger population who must compensate for smaller muscle imbalances. This larger group doubtless includes many of adult patients seen in ophthalmic practice.

Methods

Subjects were paid to participate in a protocol conforming to the Declaration of Helsinki and approved by the Institutional Review Board for Protection of Human Subjects at the University of California, Los Angeles. These patients reported progressively decompensating intermittent vertical diplopia, and were invited to participate when presented for surgical correction. Eye examinations were performed by an author, verifying that refractive error permitted excellent uncorrected near visual acuity. No subject had dissociated vertical deviation. In addition to routine clinical motility examination, patients were tested in an examination room to verify that they could sustain fusional vergence to a near target at 20cm. Experimental procedures were performed prior to strabismus surgery, except in the case of two patients who had undergone prior IO procedures much earlier elsewhere.

As published earlier[46–48], MRI was performed using a transparent facemask containing a surface coil array (Medical Advances, Milwaukee, WI). Through the mask, patients could view a target consisting of a central black cross on a white background measuring 3 × 3 mm with 0.75 mm stroke width, in turn surrounded by 5 concentric squares of 0.25 mm stroke width progressing in dimensions from 9 × 9 mm to 20 × 20 mm. This accommodative target was 20 cm above the subject, midway between the eyes.

The fusional ability of each was initially verified subjectively by monocularly covering each eye in the scanner, and by querying before and after every imaging sequence. However, because sensory suppression occasionally averted diplopia in some patients, fusion was also objectively verified by MRI determination of appropriate gaze direction in quasi-sagittal image planes. One subject was unable to maintain motor fusion in the scanner without the aid of partial prism correction of the vertical deviation; the minimum base down prism necessary to enable to permit sustained fusion was placed before the hypertropic eye during both the fusion and monocular viewing scans in order to assure comparable eye positions. Scans were first performed during binocular fusion, then during monocular fixation by the right eye, and finally during monocular fixation by the left eye (Fig. 1). Analysis was performed only for scans when expected fusion was verified to have been achieved during MRI.

MRI technique was as described for our studies of normal fusional divergence[48], intermittent horizontal strabismus[49], and normal VFV in response to vertical prism viewing[44]. High-resolution, T2 fast spin echo[50] MRI was performed at 1.5 T (General Electric Signa, Milwaukee, WI) using published techniques[43,46,47]. For each viewing condition, the following contiguous image sets were obtained using a 256×256 matrix, 80×80 mm field of view, and 2 mm thick planes (312 micron pixels): 9 – 11 quasi-sagittal images of both eyes parallel to the long axis of the orbit, to verify vergence and for determination of IO muscle cross section (Fig. 1); and 18–19 quasicoronal images perpendicular to the long orbital axis for determination of rectus and superior oblique (SO) partial volumes (Fig. 2).

Analysis.

Images were quantified using *ImageJ64* and customized programs in MatLab® (MathWorks, Boston, MA, 2011). Vertical and horizontal eye position was determined from the position of the globe-optic-nerve junction in quasi-coronal MRI as previously described[44]. It was reasonably assumed that during monocular viewing, each fixating eye was aligned on the target, so its direction was set nominally to zero for analysis. Vergence was defined to be the difference in gaze positions between the eyes.

The MRI analysis was largely as published for study of vertical fusional vergence in normal subjects[48], relying for compartmental distinctions upon histological demonstrations of compartmentally-selective intramuscular innervation in the LR, MR, IR[38], and SO[51]. Rectus and SO muscle bellies were outlined in quasi-coronal views[43]. The line of the maximum transverse dimension of each muscle was identified[43,52], so that superior and inferior horizontal rectus compartmental areas could be calculated above and below the perpendicular bisector of the maximum transverse dimension. This calculation omitted a region ±10% about the perpendicular bisector to avoid confounding by variations in the border between compartments. Medial and lateral compartments of the vertical rectus muscles were analogously computed[43,52]. The SR's lateral (SRl) and medial (SRm) 40% regions were tentatively considered "compartments" for purposes of analysis irrespective of neuromuscular anatomy that has neither demonstrated non-overlapping intramuscular innervation patterns, nor excluded the possibility of differential compartmental control. For the SO, it was earlier found that a line 30° or 60° to the long axis of the SO cross section optimally discriminates compartmental function, again omitting the central 20%[44]. As we have done elsewhere[48], we analyzed the SO using both the 30° and 60° angles, but also analyzed the whole muscle.

Change in posterior partial volume (PPV) was employed as an indicator of EOM contractility, because it correlates closely with angle of duction for vertical[52] and horizontal rectus muscles[21]. We computed PPV by summing the cross sections of each EOM compartment in the four contiguous image planes from 8 to 14 mm posterior to the globe-optic nerve junction[21]. We compared three PPV differences: 1) between the hypertropic state and the monocular fixation corresponding to the movement of redress required to correct the strabismus without binocular interaction; 2) between monocular fixation by the fellow eye and the fusing state, corresponding to the duction required to correct the strabismus but with binocular interaction; and 3) between monocular fixation by the same eye and the fusion

state, representing ideally no horizontal or vertical duction but specific to the binocular fusion state rather than merely monocular fixation in the same eye position.

The horizontal rectus muscles were not analyzed because patients often exhibited failure of convergence to the near target when binocular fusion was prohibited, either by breakdown of hyperphoria, or monocular occlusion. The eyes consequently were on average 6–8° divergent in the absence of binocular fusion, which is associated with substantial PPV changes in the MR and LR that would be impossible to distinguish from the effects of VFV.

As previously[48], the individual eye was used as the unit of sampling. Statistical comparisons and linear regressions were performed using GraphPad Prism (GraphPad Software, La Jolla, CA, USA). Graphs show all individual measurements, as well as the mean and standard error of the mean. Data were analyzed for the fusing state only when MRI confirmed that motor fusion was achieved during the scans, so not every subject contributed every scanning condition.

Results

Patient Characteristics.

Eight patients with large angle hyperphoria or intermittent hypertropia appeared on office evaluation to be able to fuse their deviations for a near target sufficiently to attempt fusion during MRI scanning. However, three of these, one man and two women, could not fuse at all in the MRI scanner, even with aid of base down prism before the hypertropic eye for partial strabismic correction. On MRI, one of these had profound unilateral SO atrophy consistent with congenital SO palsy, while the other two patients had normal SO size. These three patients who could not maintain fusing during MRI were not included in quantitative results of this report. Patient 3 could fuse during MRI, but only with assistance of base down prism in the path of the viewing eye; this patient was included in analysis.

Patients analyzed are listed in the Table. There were 2 women and three men, ranging in age from 27 to 53 years. All demonstrated stereopsis by Titmus testing, and three exhibited normal stereopsis of 40 arcsec despite 5 – 25Δ hyperphoria. While no patient recalled any significant head trauma or other clear precipitating event, only Patient 5 had a history of deviation dating back to childhood. Patients 2 and 4 had hypertropia strongly dependent on head tilt. Prior IO weakening surgeries had been performed, by left myectomy 15 months previously in Patient 1 and by bilateral recession 16 years previously in Patient 4.

All patients exhibited normal MRI appearance of the SO belly and tendon bilaterally. Patient 2, who had a striking increase in hypertropia during ipsilateral head tilt, exhibited bilaterally normal SO size and normal contractile SO thickening from supraduction to infraduction during MRI in those gaze positions. Time did not permit scanning in eccentric vertical gazes for the other patients.

Vertical Rectus Contractility.

The contractile difference between the vertically strabismic state under monocular occlusion and the monocular fixating state is plotted in Fig. 3A for both the hyper- and hypotropic eyes

of each subject. Remarkably, despite a mean 4.8±0.9° (standard error of the mean, SEM) infraduction of the hypertropic eye that was significantly nonzero at $P<10^{-6}$, mean contractility in the medial and lateral rectus compartments of the IR and SR did not differ significantly from zero change in PPV. Although refixation of the hypotropic fellow eye was associated by a numerically similar 5.2±0.8° supraduction, both the medial and lateral IR compartments relaxed significantly, with 8–10% lower PV (P<0.001) when the eye returned from hypotropia to the target.

The contractile difference between the vertically strabismic state under monocular occlusion and the binocularly fusing state is plotted in Fig. 3B for both the hyper- and hypotropic eyes of each subject. Mean infraduction of the hypertropic eye was 2.2±2.1° and mean supraduction of the hypotropic eye was 4.1±0.9°. In this situation, the lateral compartment of the hypertropic IR significantly contracted at about 10% higher PPV, and the lateral compartment of the IR in the hypotropic eye was significantly relaxed at about 12% lower PPV. There was no significant PPV difference in the medial compartment IRm in either eye nor in either compartment of the SR in either eye.

The contractile difference between the monocularly fixating state with the fellow eye deviated and the binocularly fixating state is plotted in Fig. 3C for both the hyper- and hypotropic eyes of each subject. The vertical gaze change for the hypertropic eye was measured to be 0.4±0.3°, and for the hypotropic eye −0.1±0.6°, neither of which differs significantly from zero, as should be the case if eye position did not change from fixation to fusion. There was no significant contractile difference for this comparison in either compartment of the IR and SR of the hypertropic eye, but the lateral compartment of the hypotropic IR was about 10% less contracted in the fusing state than when viewing the same target during monocular occlusion of the fellow eye (P=0.008), and the medial portion of the SR (SRm) was about 8% less contracted (P=0.001). This represents a compartmentally-selective co-relaxation of the vertical rectus pair in the hypotropic eye.

Superior Oblique Contractility.

None of the patients had imaging evidence of SO palsy or paresis. There were no significant differences in contractile behavior between the medial and lateral SO compartments under any of the conditions tested, so PPV changes in the whole SO were analyzed in interest of robustness (Fig. 4). The hypotropic eye SO relaxed by an average of about 12% to return to central gaze under monocular conditions. There were no other significant contractility effects for the SO.

Inferior Oblique Muscle Size.

Analysis of the IO was limited to measurement of its maximum cross sectional area at the point where it crossed the middle of the IR muscle[31]. This limitation was motivated by recognition that two of the patients had previously undergoing IO weakening surgery, one bilaterally. As seen in Fig. 5, there was no significant difference in IO cross section in either eye in any viewing condition.

Discussion

The present study successfully employed multipositional, quantitative MRI to study extraocular muscle contractility in the majority of volunteer patients who had enhanced vertical fusional amplitudes compensating for large heterophorias that on average far exceeded the normal compensatory range of 2Δ at distance and 4Δ at near[44]. This study constituted a challenging effort, because three of the 8 patients in whom the study was attempted were unable to maintain fusion at all in the MRI scanner. This probably reflects the severity of the underlying condition that motivated these patients to seek surgical consultation for their intermittent vertical diplopia when fatigued.

Contrary to previous claims grounded in behavioral evidence, MRI here indicates that the fusional vergence that compensated for large vertical heterophorias in these five patients is implemented mainly by the lateral compartment of the IR muscle. There was little or no contribution from the SR muscle. While the SO in the fellow eye contracted significantly when the eye was hypotropic, the absence of any other contractile behavior in the hypertropic SO or in the transition from aligned fixation to fusion argues strongly against Enright's claim that vertical fusional vergence is accomplished by the SO exclusively[16]. The present study also failed to observe a contribution from the IO muscle, insofar as IO cross section in the region where its contractility is best demonstrated[31] did not vary with vertical tropia or fusion, and most tellingly, three of the total 10 IO muscles studied here had been surgically weakened even before MRI was conducted.

The current findings are generally consistent with our previous MRI study in normal subjects during hyperphoria induced by monocular viewing through a 2Δ base up prism[44]. That study, which was performed in 14 healthy volunteers, demonstrated that the compensatory VFV was implemented by ipsilateral contraction and contralateral relaxation of the IR medial compartment. However, the associated PPV changes in the normal IR were in the range of 1–2%, which is only about a fifth the magnitude observed here in patients with large hyperphorias. In the infraducting eye, the SO medial compartment relaxed; contralateral to prism, the SO lateral compartment contracted. There was no contractility in the SR in either eye. Normal subjects also demonstrated contraction of the superior compartment of the ipsilateral LR muscle, a phenomenon not studied in the current patients because manifestation of hyperphoria also caused a confounding breakdown of fusional convergence to the near target.

Since current MRI methodology does not require a change in gaze direction to interpret contractile muscle function, it was possible to compare the effect of binocular fusion with simple monocular fixation of a target in the same eye position. There was no significant horizontal or vertical eye position change due to fusion in the viewing eye, but horizontal vergence in the fellow eye would almost certainly have changed and such vergence is known to be associated with bilateral torsion[53]. As seen in Fig. 3C, in the hypotropic eye, the transition from monocular fixation to fusion was associated with a robust 8 – 10% co-relaxation in the lateral compartment of the IR, and in the medial region of the SR. This violation of Sherrington's law of reciprocal agonist-antagonist behavior seems likely to be

exploiting differential compartmental behavior in the vertical rectus muscles to implement torsion associated with fusion; the vertical effects would likely cancel.

We have elsewhere demonstrated by MRI a selective role for the MR superior compartment during physiological fusional divergence to near and far targets[48], the latter likely representing compensation for esophoria. We also showed that convergence controls large exophoria by greater contraction in the superior than inferior compartment of the MR muscle[45]. We further showed that compartmental horizontal rectus co-contraction also occurs in exophoria and exotropia, explaining in part why divergence mechanisms eventually fail to avert symptomatic strabismus when esophoria progresses sufficiently[49]. The present study extends to vertical strabismus the recognition of a contribution of differential compartmental contractility of extraocular muscles to enhanced VFV that compensates for heterophoria.

The current study has some immediate clinical implications that should motivate reconsideration of our traditional clinical approaches to chronic vertical anisophoria. First, such cases should not be uncritically assumed to represent congenital or decompensated SO palsy. Palsy of the SO is possible, of course, yet perhaps not all that common since only one of the 8 cases studied here exhibited SO atrophy that objectively secures this diagnosis, while the rest had normal SO muscles. If one regards MRI evidence of neurogenic SO atrophy and reduced contractility as the gold standard to confirm SO palsy, the 3-step test is only 70% sensitive[54] and 50% specific for actual SO muscle hypofunction[7].

A second clinical point is that the use of “hypertropia” as the preferred term to describe vertical binocular misalignment probably biases clinical thinking to presume that the abnormal muscle function is primarily in the higher eye. However, the MRI data indicate that in the current patients with large decompensating vertical alignment imbalances, the muscle activity involved in symptomatically releasing vs. compensating the strabismus is mainly in the IR (Fig. 3) and SO (Fig. 4) of the lower eye. This is consistent with an earlier MRI report of enhanced IR size and contractility contralateral to confirmed SO palsy, and the suggestion that the large magnitude of vertical misalignment in such cases may be due in large measure to contralateral IR over-contraction[28]. Since none of the current patients who contributed to the quantitative MRI analysis had SO palsy, it would appear that over-activity of the hypotropic IR, rather than a weak SO, may be the common factor in vertical strabismus with enhanced vertical fusional amplitudes. This finding provides further physiological basis for the surgical approach of including contralateral IR weakening in decompensating vertical deviations, including SO palsy.

The important role of the IR lateral compartment in fine tuning VFV would argue that surgical manipulations be targeted to the medial compartment when possible. While the current study was, of course, not designed to evaluate surgical alternatives, these findings have motivated us to perform graded partial tenotomy of the IR[55] from the nasal side, so as to maintain as much as possible the transmission of lateral compartment force and thus preserve VFV. It is possible for the strabismus surgeon to longitudinally split the IR to permit compartmental recession or tightening or to slant its scleral insertion after recession.

However, we do not have an adequate experience to report results of such techniques at this time.

Strengths and Limitations.

A strength of this experimental study is that it objectively evaluated the function of all of the compartments of the rectus muscles and the overall function of the obliques in patients with intermittently compensated but large angle vertical strabismus. Analysis did not make any geometric or kinematic assumptions about individual muscle function as reflected in observable eye positions; instead, the outcome measure was a direct indicator of contractility of each individual muscle. This approach avoids possible misinterpretations based on historical but incomplete concepts of muscle function developed before recognition of the role of the orbital pulley system and the compartmental diversity of muscle actions.

This demanding study was limited by the small number of patients who could perform the experiment and by the inability of some of them to sustain, supine in the MRI scanner, the same compensation for their large vertical phorias that they achieved while upright in the clinical examination room. Even among the five patients who could generally fuse large vertical deviations during MRI, data from some scans could not be included due to episodic fusion failure. One patient required partial prism correction to maintain fusion during MRI. The prior history of IO surgery in two patients limited the ability to draw strong conclusions about IO function, other than that it is evidently not absolutely necessary to implement compensation of large vertical heterophoria. None of the patients who contributed to the quantitative data had SO palsy, although this was only determined by the orbital MRI findings in the course of the experiment. The MRI employed here required special surface coil technique, scanning protocol, and target placement; the approach is not advocated for routine clinical use, but rather to provide insight into the pathophysiology of strabismus.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Biographies

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Joseph L. Demer received his MD and PhD in Biomedical Engineering from Johns Hopkins in 1983. He is Chief of Pediatric Ophthalmology and Strabismus at UCLA Stein Eye Institute, holds the Rosenbaum Professorship, is Professor of Neurology, and chairs the EyeSTAR residency-PhD program. ARVO awarded Dr. Demer its highest honor, the

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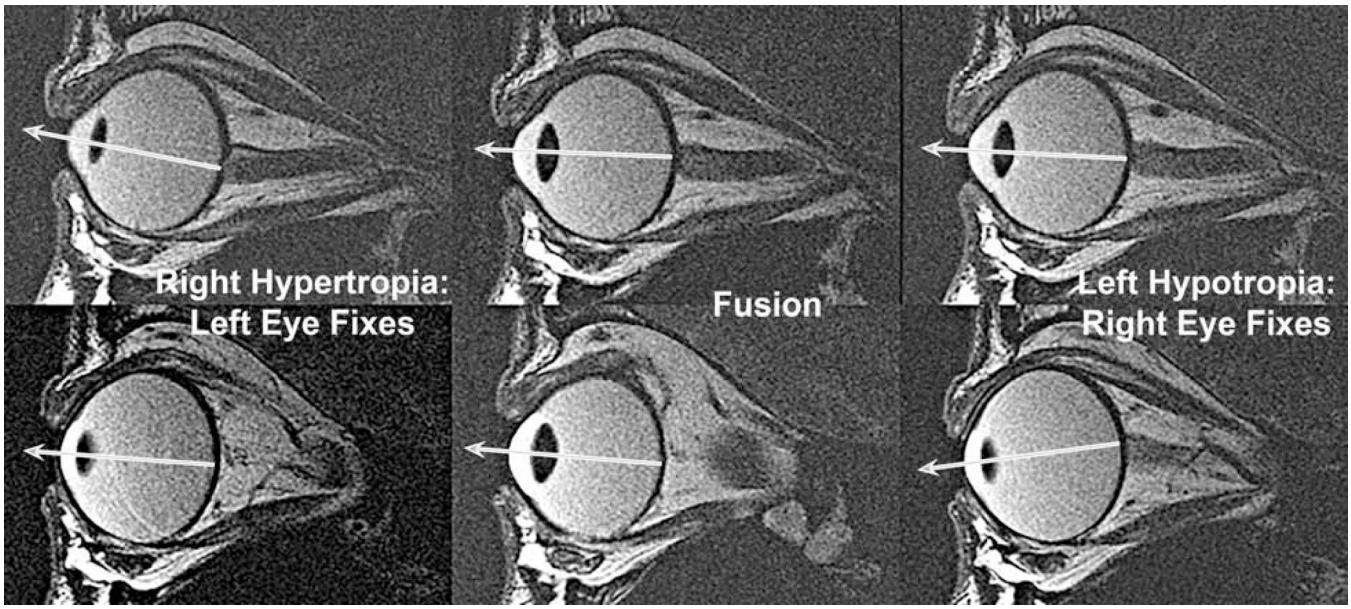

Fig. 1.

Quasi-sagittal MRI in Patient 4, who had intermittent right hypertropia. Right eye shown in upper row, left in lower row. Left column: right eye hypertropic under cover, left eye fixes. Center column: Fusion. Right column: left eye hypotropic under cover, right eye fixes. Arrows indicate approximate lines of sight.

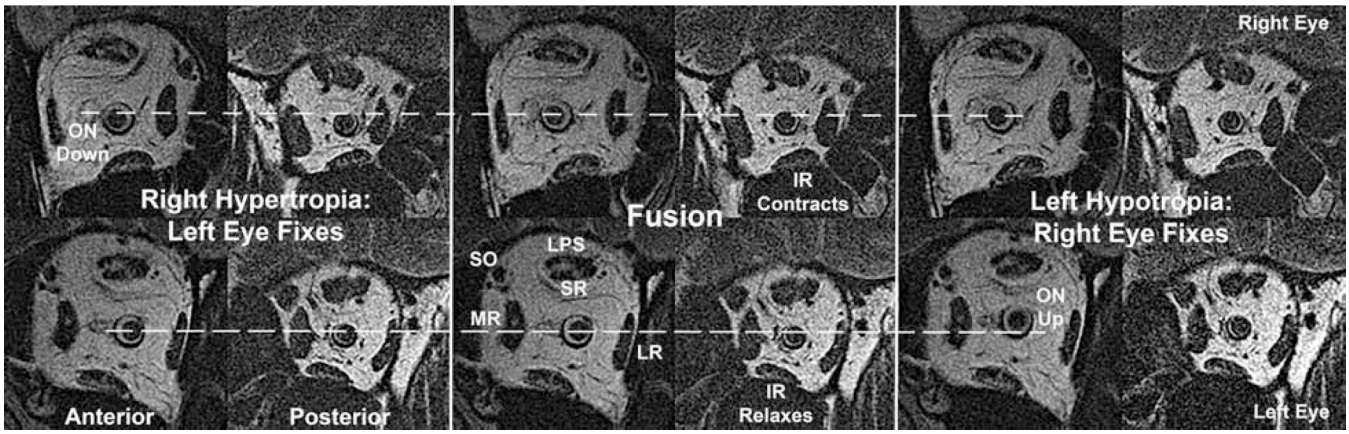

Fig. 2.

Quasi-coronal MRI in Patient 4, who had intermittent right hypertropia and whose quasi-sagittal MRI appears in Fig. 1. Conventions are similar to Fig. 1, except that each column contains the anterior image plane at the globe-optic nerve junction used for quantitative analysis of vertical eye position, and a mid-orbital image plane in the region used for analysis of muscle contractility. IR- inferior rectus muscle. LPS – levator palpebrae superioris muscle. LR – lateral rectus muscle. MR – medial rectus muscle. ON – optic nerve. SO - superior oblique muscle. SR – superior rectus muscle.

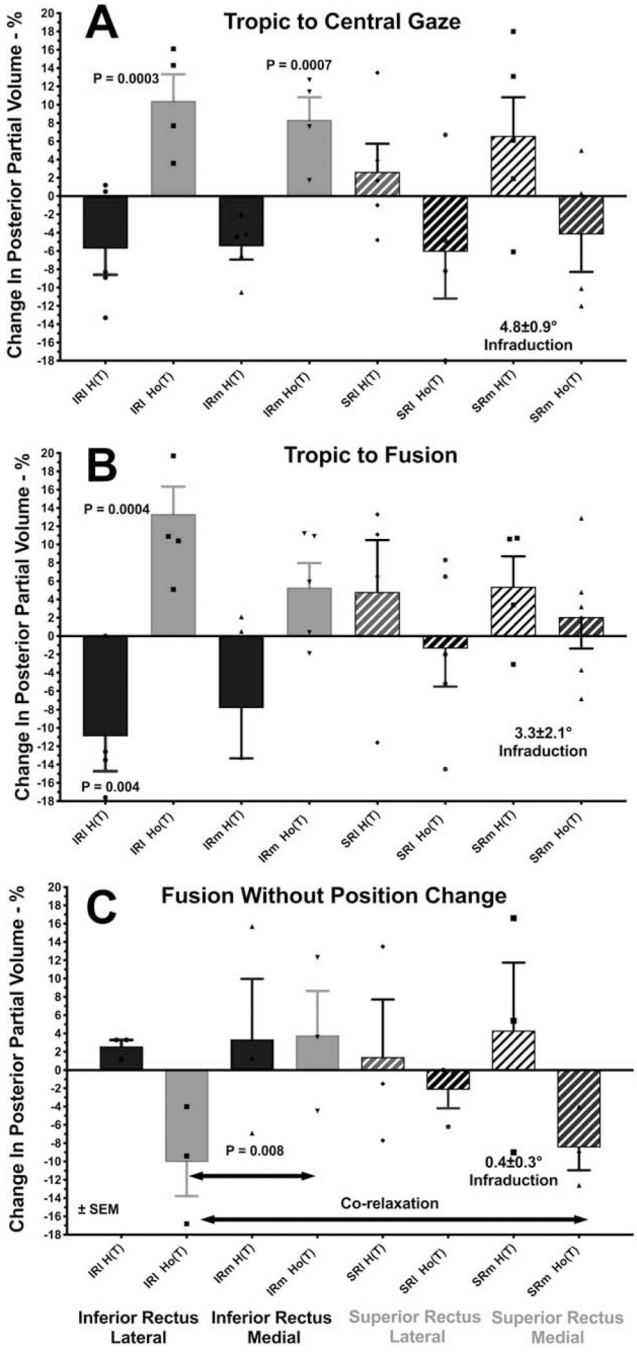

Fig. 3.

Mean compartmental vertical rectus muscle contractility compensating for intermittent hypertropia, as indicated by percent change in posterior partial volume. A. Contractility returning to monocular central gaze from tropic position. B. Contractility returning to binocular central fusion from tropic position. C. Contractility in binocular fusion relative to monocular fixation of the same target in central gaze. P values indicate significant differences from zero. H(T) – intermittently hypertropic eye. Ho(T) – hypotropic fellow in intermittent hypertropia. IRm – medial compartment of inferior rectus muscle IRl – lateral

compartment of inferior rectus muscle. SRm – medial region of superior rectus muscle. SRl – lateral compartment of superior rectus muscle. SEM – standard error of mean.

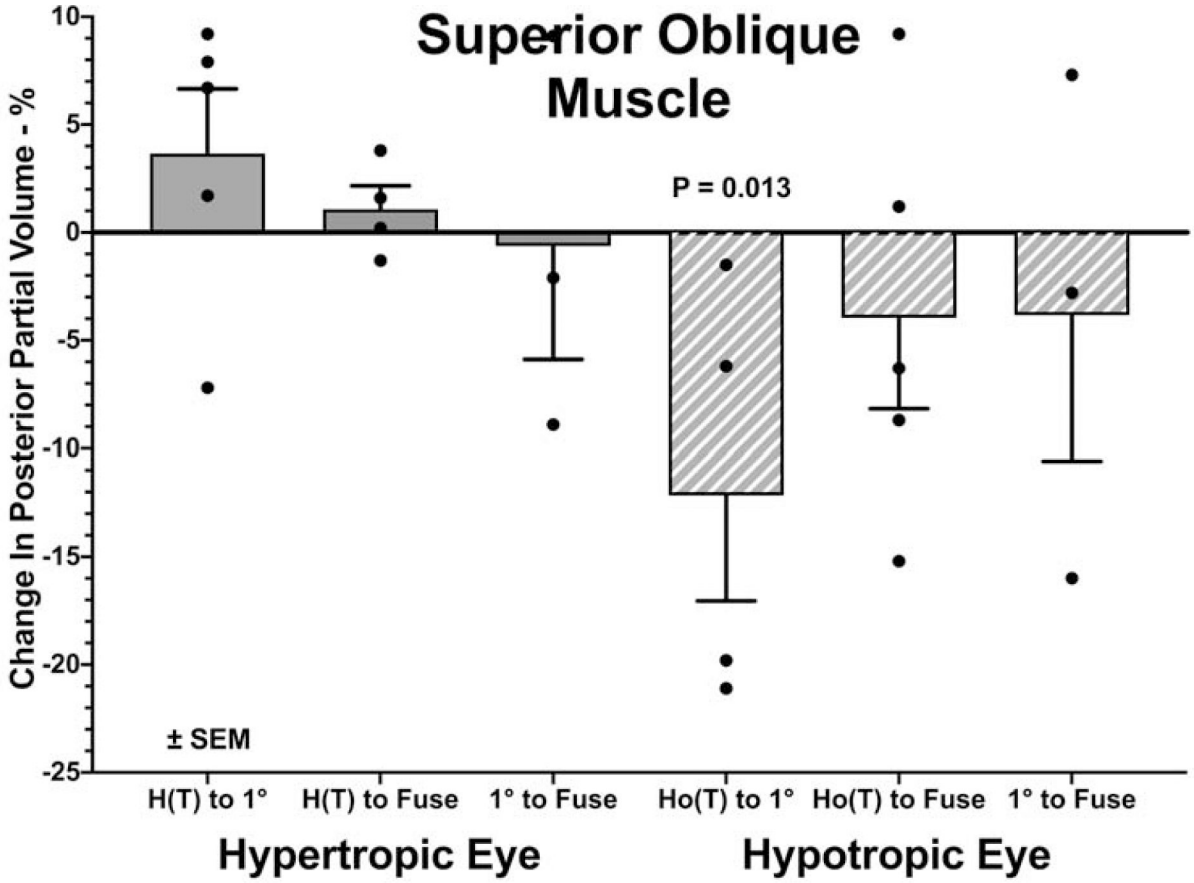

Fig. 4.

Contractile change in superior oblique muscle, as indicated by percent change in posterior partial volume. H(T) to 1° - return to monocular central gaze from hypertropic position. H(T) to Fuse - return to binocular fusion in central gaze from hypertropic position. 1° to Fuse - Contractility in binocular fusion relative to monocular fixation in central gaze. Ho(T) to 1° - return to monocular central gaze from hypotropic position. Ho(T) to Fuse - Contractility in binocular fusion in central gaze from hypotropic position. SEM – standard error of mean.

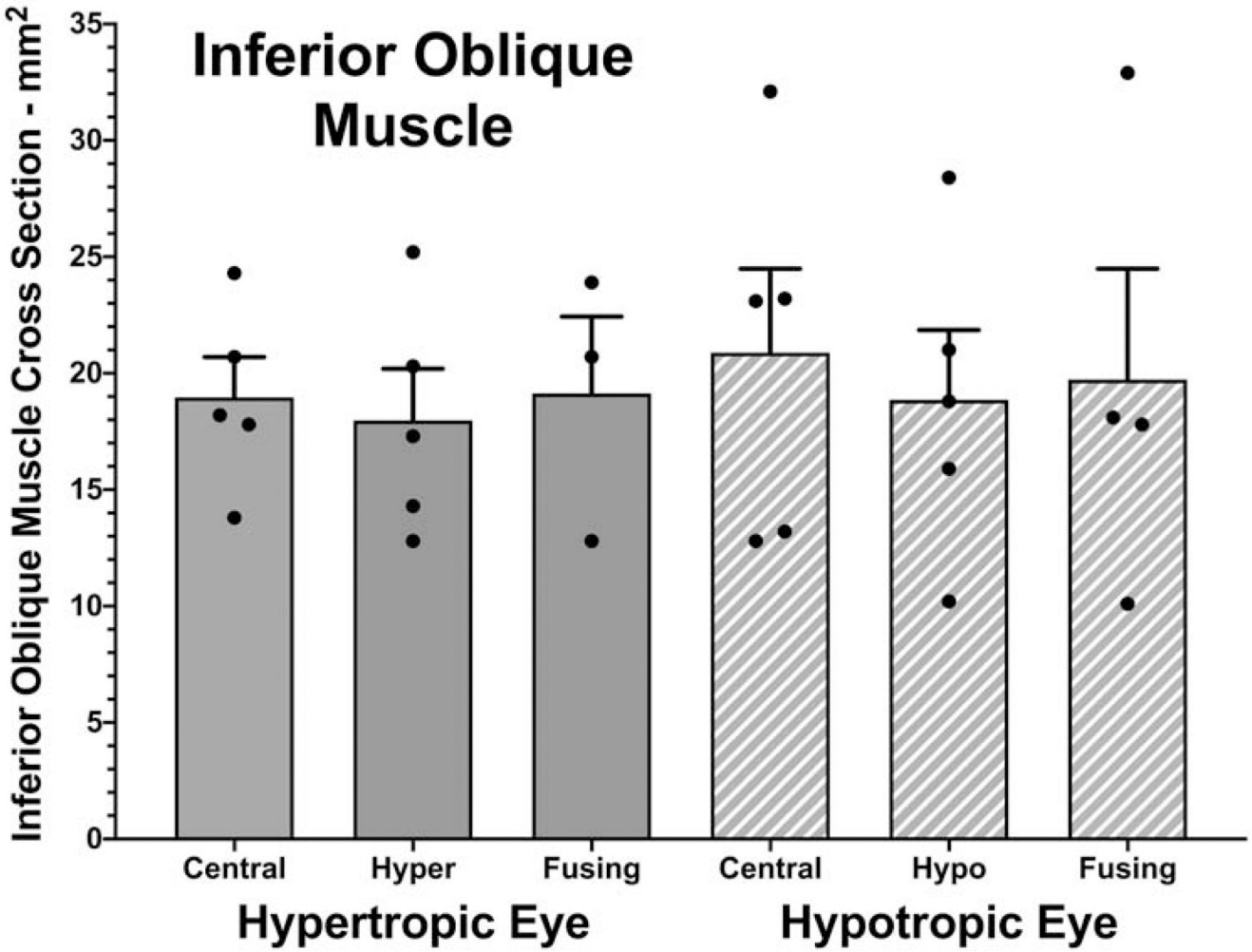

Fig. 5.

Mean cross section of the inferior oblique muscle of both eyes in central gaze, during monocular occlusion, and during binocular fusion. Values are mean ± standard error of mean.

Table.

Patients With Intermittent Hypertropia

Patient	Age (years)	Sex	Hypertropia Laterality	Duration (years)	Prior Surgery	Central Hyper (Δ)	Right Tilt Far (Δ)	Left Tilt Far (Δ)	Near Exophoria (Δ)	Stereopsis (arcsec)	Scan Prism (Δ)
1	39	male	right	2	Left IO myectomy	25	---	---	4	40	----
2	27	male	left	2	-----	5	1	25	0	40	----
3	53	female	right	6	----	5	6	6	0	100	2
4	31	male	right	15	Bilateral IO recession	25	30	ortho	10	40	----
5	40	female	left	35	----	18	18	20	−20	140	----