Understanding and misunderstanding extraocular muscle pulleys

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As evidence has mounted for the critical role of extraocular muscle (EOM) pulleys in normal ocular motility and disease, opposition to the notion has grown more strident. We review the stages through which pulley theory has developed, distinguishing passive, coordinated, weak differential, and strong differential pulley theories and focusing on points of controversy. There is overwhelming evidence that much of the eye's kinematics, once thought to require brainstem coordination of EOM innervations, is determined by orbital biomechanics. The main criticisms of pulley theory only apply to the strong differential theory, abandoned in 2002. Critiques of the notion of dual EOM insertions are shown to be mistaken. The role of smooth muscle and the issue of rotational noncommutativity are clarified. We discuss how pulley sleeves can be stabilized as required by the theory, noting that more work needs to be done in specifying the tissues involved.

Keywords: active pulley hypothesis, APH, extraocular biomechanics, extraocular connective tissue, extraocular muscle pulleys, EOM pulleys, Listing's law

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Status of the pulley concept

The notion that connective tissues function as extraocular muscle (EOM) pulleys elastically stabilized relative to the orbital wall, and consequently that muscle actions are gaze dependent (Miller, 1989), was a new idea in a field that supposed EOM actions to be basically understood and extraocular anatomy to hold no big surprises. Considered as a scientific revolution (Demer, 2002; Haslwanter, 2002), this was certainly a minor one, and yet it has substantially reoriented thinking in the field and stimulated much fruitful, innovative research in anatomy, modeling, mathematical analysis, imaging, and neurophysiology. Perplexingly, as evidence for pulley theories has mounted, and the scientific picture has clarified, opposition has grown more strident.

Converging support

Neurophysiologists have generally maintained that the implementation of Listing's law (which specifies torsion for each gaze) and solutions to problems posed by noncommutativity of three-dimensional rotations (such as how independent horizontal and vertical gaze centers could control nonadditive eye rotation; Porrill, Warren, & Dean, 2000), must lie in the brainstem (Angelaki & Hess, 2004; Crawford, Martinez-Trujillo, & Klier, 2003; Nakayama, 1975; Tweed, Haslwanter, Happe, & Fetter, 1999; Tweed & Vilis, 1987). Recently, however, Ghasia and Angelaki (2005) showed that cyclovertical motoneurons do not modulate their firing during eccentric pursuit, as would be necessary if the brainstem implemented Listing's law. Then, Klier, Meng, and Angelaki (2005, 2006) stimulated the abducens nerve and nucleus, downstream of all neural circuits that might contribute to the implementation of Listing's law, and found that eye movements nevertheless had Listing kinematics, proving that ocular plant mechanics are capable of implementing Listing's law without neural assistance. Thus, at the end of 2005, in addition to the modeling results that first predicted pulleys (Miller, 1989; Miller & Robinson, 1984), the imaging studies that confirmed the early muscle path predictions (Miller, 1989; Miller, Demer, & Rosenbaum, 1993), the mathematical analyses that then showed pulleys suitable for implementing commutativity (Quaia & Optican, 1998; Raphan, 1998) and separability of horizontal and vertical controllers (Porrill et al., 2000), the many imaging studies that determined their normal and abnormal positions and movements (e.g., Clark, Miller, & Demer, 1997, 2000; Clark, Miller, Rosenbaum, & Demer, 1998; Demer, Clark, & Miller, 1999; Demer, Miller, Glasgow, Rabiah, & Vinters, 1994; Demer, Poukens, Clark, Miller, & Porter, 1998), the histochemical studies that showed supportive elastin fibrils and smooth muscle (SM) cells to be concentrated in pulley tissues (Kono, Poukens, & Demer, 2002b; Miller et al., 2003), along with innervations to modulate tension in the latter (Demer, Poukens, Miller, & Micevych, 1997), the electron microscopic studies that showed pulley tissues to have an unusual, stout, cross-layered structure (Porter, Poukens, Baker, & Demer, 1996), and the studies in nonhuman species (mouse and monkey) that showed pulleys to be evolutionarily conserved (Demer et al., 1997; Khanna & Porter, 2001), there was now compelling neurophysiologic evidence from alert, behaving

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primates that most or all of the mechanism underlying the eye's fundamental Listing kinematics lay in the orbit. Because EOM pulleys are the only candidate orbital mechanism, their functionality would seem to be firmly established.

It was against this background that an astonishing article appeared, in which McClung, Allman, Dimitrova, and Goldberg (2006) expressed doubt that extraocular connective tissues had any role at all in oculomotility and insisted that extraocular mechanics was as "described in classic anatomic studies and books for over 70 years." A spirited exchange of E-letters followed (Demer, 2006a; Goldberg, 2006; Miller, 2006). A similarly oriented paper from Jampel and Shi (2006) and an e-letter response (Demer, 2006b) subsequently entered the literature.

We have long been aware of an undercurrent of discomfort concerning EOM pulleys, and by airing this malaise the papers by McClung et al. (2006) and Jampel and Shi (2006) have made effective responses possible. The body of this review will therefore consist of detailed analyses of specific points of controversy, embedded, as will be seen necessary, in an account of the field's development. We will also briefly consider how the pulley controversy relates to other scientific controversies, issues of effective scientific communication, and directions of basic and clinical oculomotor research.

Resolving the controversy

Following Thomas Kuhn (1996), the philosopher who proposed that science after a "paradigm shift" was incommensurate with science before, one might propose that old school loyalists parse theory and evidence so differently that they are unable to evaluate the new pulley ideas (Demer, 2002; Haslwanter, 2002). However, it has been pointed out (e.g., Weinberg, 1998) that Kuhn's notion is over stated with respect to the great scientific revolutions (modern physicists, for example, learn and routinely apply both Newtonian and relativistic mechanics without epistemological catastrophe), and it may be too pessimistic a view of the present situation. Nevertheless, we will find that radical critics have indeed misunderstood both the theory and the data related to pulleys.

The pulley literature can be roughly sorted into several groups. First, there are about 50 papers proposing pulley theories and bringing various sorts of evidence to bear on them; slightly more than half of these are from Demer et al. or my laboratories. Next, there are about 30 papers from mathematically sophisticated neurophysiologists on the question of whether the complexities of three-dimensional rotation must be solved by the brain. From an initial supposition that these problems were all solved in the brain (e.g., Tweed & Vilis, 1987), there is a clear trend toward attributing much of the basic kinematics to extraocular pulleys (e.g., Misslisch & Tweed, 2001). A selection of the most important papers from these two groups was cited above. We count 5 papers accepting the functional notion of EOM pulleys, but proposing quite different implementations than those favored by Demer and Miller (Schutte, van den Bedem, van Keulen, van der Heim, & Simonsz, 2006; Simonsz, Harting, de Waal, & Verbeeten, 1985; van den Bedem, Schutte, van der Helm, & Simonsz, 2005), or claiming that pulleys were identified long ago, and that there is nothing essentially new in the recent pulley proposals (Simonsz, 2001, 2003). We will evaluate these claims below. There are some 20 papers applying pulleys to clinical problems and another 25 on basic science issues, such as muscle fiber and motoneuron specialization, and extraocular connective tissues, which seem to have attracted interest because of their relevance to pulleys. Finally, there are 2 papers from Goldberg's group and 1 from Jampel's group, which offer radical critiques in the sense that they dispute most or all of the claims made concerning pulley functionality (Dimitrova, Shall, & Goldberg, 2003; Jampel & Shi, 2006; McClung et al., 2006). Examination of the points of controversy raised in these papers makes it clear that they do not turn on subtleties in weighting or interpretation of data.

To anticipate, we will find that the theory of pulley function is both innovative and well supported. In contrast, the current specification of pulley implementation is only qualitative and may be incomplete. We will find that most critiques of pulley theory are incorrect, being based on gross misunderstanding or directed at abandoned hypotheses. Pulley theory has been under active development: many ideas have been proposed and tested, and some have been laid aside. This is the normal routine of empirical science (Kuhn, 1996; Popper, 1968).

The traditional model of muscle action is rehabilitated

Robinson (1975) and Miller and Robinson (1984) sought to express extraocular mechanics in predictive, computational models. Reflecting what was known at the time, these models supposed each rectus muscle's action to be determined by its muscle plane, the plane containing the globe's center of rotation, the muscle's anatomic origin, and the muscle's point of effective insertion in the globe.

According to the classical notions (see, e.g., Boeder, 1962; Krewson, 1951), rectus EOMs were constrained only at their ends: each followed a great-circle path from its insertion to its point of tangency with the globe, and then a straight path to its origin in the orbital apex. Robinson (1975) observed that this model could not be correct: during normal eye rotation, such muscles would sideslip wildly about the globe, which would make eye rotation uncontrollable, and in any case did not occur. Miller and Robinson (1984) attempted to "rehabilitate" the classical model, moderating sideslip instabilities by

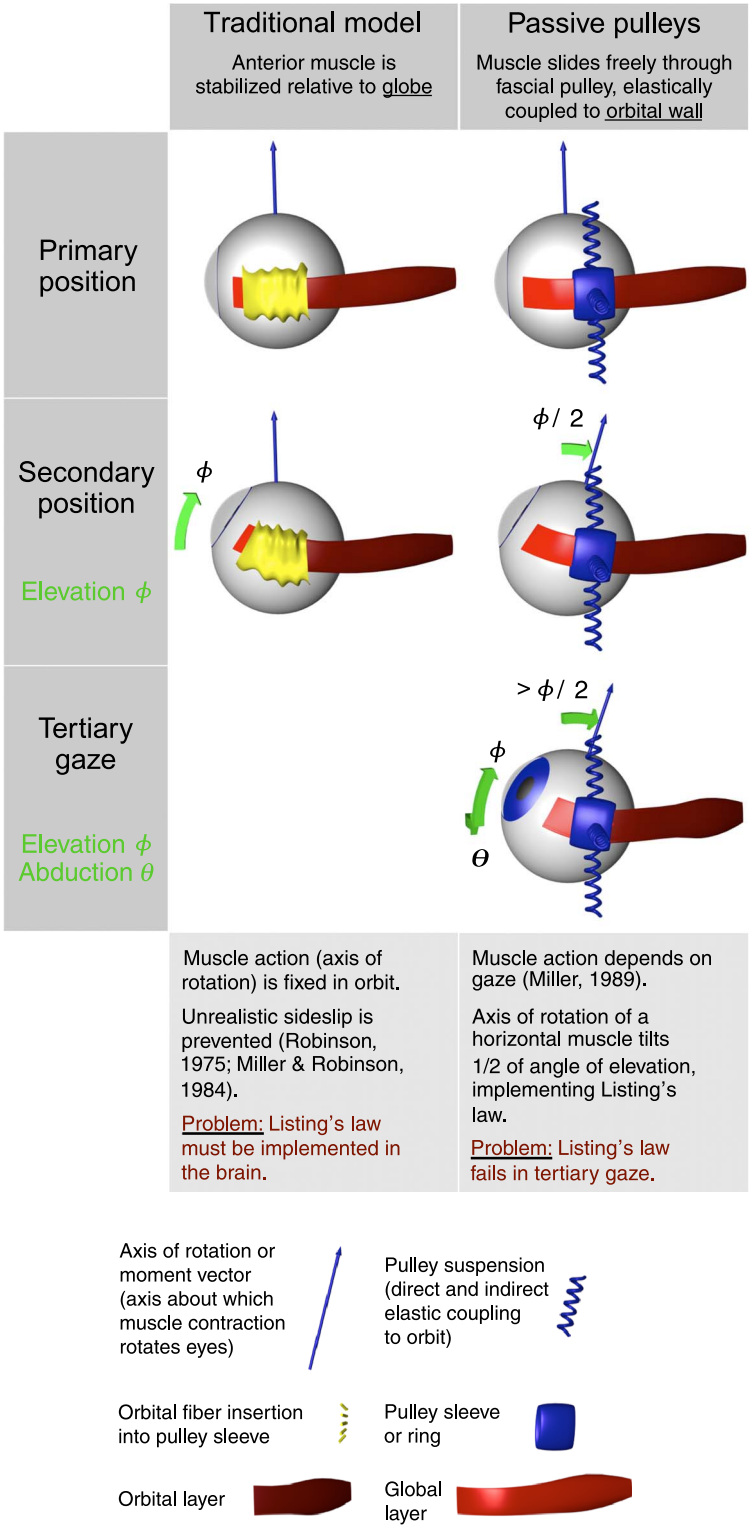

Figure 1. Traditional and passive pulley models of EOM action. The traditional model reflects the classical notion that EOMs are constrained only at their ends, as rehabilitated by Miller and Robinson (1984) to correct the sideslip instabilities discovered by Robinson (1975). The essential kinematic feature of the traditional model is that a muscle's axis of rotation (blue arrow) remains roughly fixed in the orbit for all gazes, leaving the brain to cope with rotational noncommutativity, and to enforce Listing's law. The passive pulley model (Miller, 1989; Miller et al., 1993) supposes that EOMs slide freely through connective tissue sleeves, which are elastically stabilized relative to the orbital wall. Passive pulleys make axes of rotation a function of gaze, making the eye appear commutative to the brain and implementing Listing's law near secondary gazes (Quaia & Optican, 1998; Raphan, 1998).

supposing there to be significant musculoglobal elasticities. This model, which we will call the *traditional model,* reasonably simulated normal and abnormal binocular alignment—with the notable exception of muscle transposition surgery—and formed the basis of the Orbit 1.0™ Gaze Mechanics Simulation (Miller & Shamaeva, 1993). The essential kinematic feature of the traditional model (Figure 1) is that a muscle's axis of rotation remains roughly fixed in the orbit for all gazes, leaving the brain to cope with rotational noncommutativity and to enforce Listing's law.

Passive pulleys make muscle actions a function of gaze

X-ray (Miller & Robins, 1987; Miller, Robinson, Scott, & Robins, 1984), CT (Simonsz et al., 1985), and MR (Miller, 1989) images as a function of gaze then showed that posterior muscle paths were even more stable relative to the orbit than this early modeling suggested. Miller (1989) proposed that, although it was possible to maintain the traditional model by supposing a fortuitous balance of muscle tension against musculoglobal elastic coupling, it was more likely that muscle paths were directly stabilized by sheaths that functioned as pulleys elastically stabilized relative to the orbital wall (Figure 1, passive pulleys). These were originally called "soft rectus muscle pulleys" to emphasize that they applied only to the rectus muscles and consisted of distributed, compliant connective tissues. We will refer to them here as *passive pulleys* to distinguish them from the subsequently proposed *active pulleys*. Miller (1989) pointed out that muscle planes (determined by a muscle's anatomic origin, its effective insertion, and the globe's center of rotation), used to describe muscle actions under the traditional model, would not describe the actions of muscles passing through pulleys, and that in an eye with pulleys muscle actions would be a function of gaze. Such an eye would require quite different brainstem control signals than one with traditional mechanics, in which muscle actions would be fixed in the orbit (see Figure 1). A test of the passive pulley model was described, using MRI data before and after muscle transposition surgery, and pilot results were cited in support of the model (Miller, 1989). A completed experimental test providing further confirmation was reported by Miller et al. (1993). Passive pulleys make axes of rotation a function of gaze. Appropriately located passive pulleys (see Figure 2) would cause a muscle's axis to tilt by half of the angle of eye rotation (elevation in the figure). In this connection, 1/2 is a "magic number" because Listing's law is satisfied if the axis of rotation shifts by 1/2 of a shift in eye orientation (Tweed, Cadera, & Vilis, 1990; Tweed & Vilis, 1990). Listing's law is

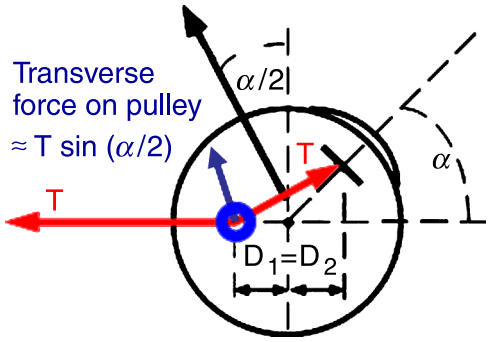

Figure 2. "Passive" pulley forces. A muscle (red line) under tension T, passing freely through a pulley (blue ring), exerts a transverse force (dark blue arrow) on the pulley that depends on its angle of deflection $\alpha/2$, where α is the eye's angle of eccentricity (e.g., elevation for a horizontal rectus muscle).

mathematically equivalent to the half-angle rule, so the assertion that

> The confusion surrounding Listing's law has yet to be resolved, and there is no experimental physiological demonstration of the half-angle requirement

(Jampel and Shi, 2006) expresses only the confusion of its authors. Passive pulleys then implement Listing's law, making the eye appear commutative to the brain, at least as the eye initially departs from secondary gaze positions (Quaia & Optican, 1998; Raphan, 1998). Passive pulleys were first implemented in the Orbit 1.5™ Gaze Mechanics Simulation (Miller, Shamaeva, & Pavlovski, 1995).

The notion that eye position contingent kinematics could be implemented by stabilizing posterior muscle paths relative to the orbit was the essence of this proposal, not any particular anatomic implementation, and certainly not any buzzword (whether "pulley" or "poulie") used descriptively by classical anatomists, who could not have shared our biomechanical concerns (Simonsz, 2001, 2003). Nevertheless, with respect to implementation, we were impressed by (e.g., Koornneef, 1983) descriptions of extraocular connective tissues, and we supposed that our pulley sleeves and their suspensions were related to the connective tissues he so brilliantly described. Characterization of pulley tissues began in earnest with Demer, Miller, Poukens, Vinters, and Glasgow (1995).

Passive pulleys move, but only as a consequence of their compliant suspensions responding to the transverse forces produced by deflection of the EOMs sliding freely through their sleeves (Figure 2). The subsequently proposed active pulleys differ from passive pulleys in that their sleeves are supposed to be moved by muscles inserting into them.

Contrary to assertions in the literature (Jampel & Shi, 2006), the passive pulley hypothesis applies only to rectus EOMs, not to oblique muscles, and the active pulley hypothesis (APH) was advanced by Demer, Oh, and Poukens (2000), not by Miller.

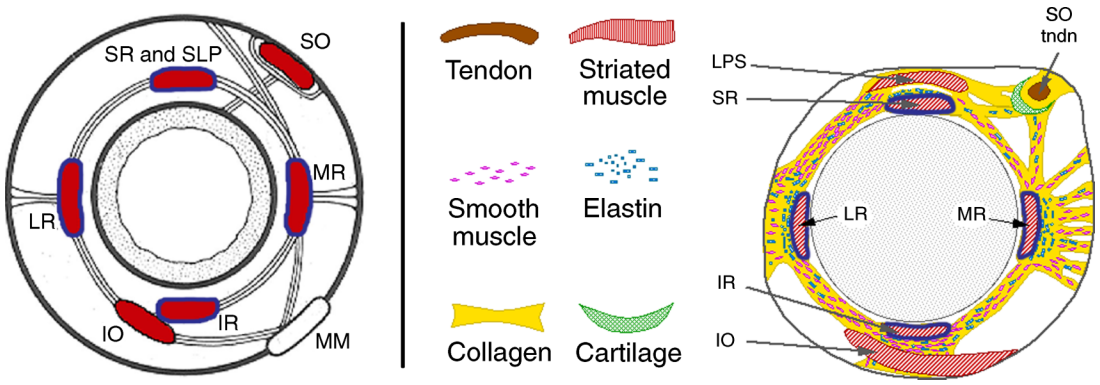

Figure 3. Schematic of equatorial connective tissues, adapted from Koornneef (1983) on left and from Demer et al. (1995) on right. Rectus muscle pulley sleeves are outlined in blue.

It is not essential that pulleys be directly coupled to the orbit to stabilize muscle paths relative to the orbit. Indeed, Abràmoff, Kalmann, de Graaf, Stilma, and Mourits (2002) have shown that pulley locations (determined by MRI) were close to normal shortly after orbital decompression surgery, in which connective tissue was thoroughly dissected from the bone. They conclude that extraocular connective tissue forms a "functional skeleton," which determines pulley positions. A substantial connective tissue skeleton has indeed been demonstrated (Figure 3).

Surgeons sometimes object that pulley tissues could not be critical because they are often sectioned and nothing untoward seems to happen. Consider, however, that even under the traditional model, cutting connective tissue attachments (particularly those of the lateral rectus muscle) should lead to catastrophic instabilities within the normal oculomotor range (Robinson, 1975). One must ask as well why these consequences are not commonly observed. Two explanations come to mind: (1) pulleys and other connective tissue attachments have little effect near primary position, which is where outcomes are typically judged shortly after strabismus surgery; and (2) connective tissues reattach soon after surgical dissection, and pulley structures may regrow. Empirical studies of these issues would be useful.

In a Crouzon's syndrome patient with "relatively good" monocular motility, van den Bedem et al. (2005) made an observation suggestive of the functional equivalence of intermuscular connective tissue and direct orbital coupling:

> A thick intermuscular membrane interconnected the superior rectus and levator muscles to the lateral rectus muscle and the latter to the inferior rectus muscle. Interestingly, the intermuscular membrane was particularly pronounced in regions where no orbital wall was present

(p. 2713).

It is possible that orbital fat, which fills spaces between connective tissue septa, helps stabilize posterior muscle paths (Schutte et al., 2006). However, the measurements and simulations required to demonstrate this theory are challenging, and compelling evidence remains to be developed. We suggest that this would be facilitated if proponents of the theory attempted to demonstrate a contributory role for encapsulated fat rather than on proving it to be the sole determinant of EOM paths.

Some strabismus surgeons had argued that extraocular connective tissues were not stiff enough to deflect EOM paths, and so, taking another cue from Koornneef (1983), Demer and I suggested that SM tonus could supplement connective tissue stiffness in alert subjects. Subsequent immunohistochemical studies supported this prediction by showing dense investments of SM, along with tough elastin fibrils, in pulley-related connective tissues (Demer et al., 1995, 1997; Kono et al., 2002b). Some SM and elastin was found to be organized in bands, suggesting that its tonic innervation might be modulated, perhaps to refine binocular alignment (Miller et al., 2003).

Pulleys have also been studied with a combination of light and electron microscopy and are found to be

> …comprised of a dense collagen matrix with alternating bands of collagen fibers precisely arranged at right angles to one another. This three-dimensional organization most likely confers high tensile strength to the pulley. Elastin fibrils were interspersed in the collagen matrix. Fibroblasts and mast cells were scattered throughout the relatively acellular and avascular collagen latticework. Connective tissue and *smooth muscle bundles suspended the pulley from the periorbita. Smooth muscle was distributed in small, discrete bundles attached deeply into the dense pulley tissue* [italics added]

(Porter et al., 1996, abstract).

What are active pulleys?

Demer et al. (2000) realized that pulleys could not account for normal ocular kinematics if they only moved

passively (Figure 2), and that to implement Listing's law far from secondary gaze positions they would have to move longitudinally (roughly, anteriorly, and posteriorly) with their EOMs while continuing to resist transverse movement (movement in directions other than longitudinal). This insight led to the APH. Whereas the notion of passive pulleys supposes that EOMs slide freely through their pulley sleeves, the APH supposes that EOMs insert in their pulley sleeves and move them longitudinally. There was clear evidence prior to Demer's proposal (e.g., Spencer & Porter, 1988), and there is more now (Oh, Poukens, & Demer, 2001; Ruskell, Kjellevold Haugen, Bruenech, & van der Werf, 2005), that predominantly orbital layer fibers terminate in pulley sleeves, coupling them to their EOMs.

McClung et al. (2006) are in substantial agreement with Demer et al. (2000) on the anatomy and on the essential point that each EOM moves its pulley longitudinally:

> [O]ur images showing the rectus muscles having a sleeve of connective tissue firmly anchored into the muscle belly as well as into this portal define a mobile pulley

(McClung et al., 2006, p. 204, column 2).

However, they misunderstand Demer's proposal by failing to recognize that the APH consists of two separate notions. This distinction was implicit in Demer et al. (2000) and explicit in Kono, Clark, and Demer (2002a), who named the two versions of the APH: one in which orbital and global fiber movements were coordinated, and the other in which differential contraction was possible:

1. *Coordinated active pulleys*: Pulleys move longitudinally with respect to their EOMs while elastically resisting transverse movement. Translational forces are applied to pulleys by orbital fibers inserting into pulley sleeves, whereas oculorotary forces are applied by global fibers inserting into sclera (Figure 4, coordinated pulleys).
2. *Differential active pulleys*: Orbital and global fiber contractions are mechanically independent (the two layers can slide relative to each other) and are independently controlled by the brainstem (Figure 4, strong differential pulleys).

McClung et al. (2006) also suggest that SM was supposed to provide the "action" of the APH when they write:

> The pulley function was further elaborated by noting the presence of smooth muscle with parasympathetic innervation within these tissues. This gave the pulley a dynamic neural control component

(p. 202, column 2).

It is true that Demer et al. (2000) hypothesized that SM might help move vertical rectus pulleys medially to account for the outward tilt of Listing's planes in convergence, but it is also true that they subsequently tested this theory, disproved, and abandoned it (Demer, Kono, & Wright, 2003). In most (but not all) pulley-related theorizing, striated muscle moves the pulleys, and SM simply contributes to pulley stiffness. Both sympathetic and parasympathetic innervations were actually described (Demer et al., 1997). The assertion that active pulleys are supposed to "function without the need for neural circuits" (Jampel & Shi, 2005) is obviously incorrect.

Coordinated pulleys support Listing's law in all gazes

There is nothing in the notion of coordinated active pulleys about independent control or differential motion of orbital and global lamina. Laminar distinctions are merely references to known anatomy. Nothing about coordinated APH kinematics would change if all fibers were coupled to both the pulley sleeve and the sclera.

Mathematically oriented neurophysiologists observed that EOM pulleys finally provided a plausible explanation of how the brain controls the noncommutative three-dimensional rotations of the globe (Quaia & Optican, 1998; Raphan, 1998) and does so with separate horizontal and vertical gaze centers (Porrill et al., 2000). Having provided the important insight that pulleys could only perform these essential functions in tertiary gazes (gazes with both horizontal and vertical coordinates nonzero) if they moved longitudinally in particular ways (e.g., in abduction, the LR pulley must move posteriorly and the MR pulley must move anteriorly), Kono et al. (2002a) then demonstrated by MRI that horizontal rectus pulleys actually moved as required.

Jampel and Shi (2006) have recently denied that there is any issue regarding commutativity: "all human movements are commutative," they say, and "it is not necessary to substitute the complex issues of commutative and noncommutative mathematics for Donders' law." Noncommutativity of three-dimensional rotations, however, is a mathematical fact of life, and Donder's law, or more precisely Listing's law, is an achievement of the oculomotor system that requires explanation (Figure 5).

Pulleys move anteriorly and posteriorly because they are attached to the EOMs, in addition to being stabilized relative to the orbital wall. EOMs develop whatever force is necessary to rotate the eye against antagonistic muscles and elastic orbital tissues, among which are the pulley suspensions themselves. According to the coordinated pulley model, the entire EOM (both orbital and global layers) contracts in coordination, rotating the eye and moving the pulley.

Extirpation of coordinated pulleys would therefore reduce the load on the EOMs. Dimitrova et al. (2003) showed, consistent with the notion of coordinated pulleys,

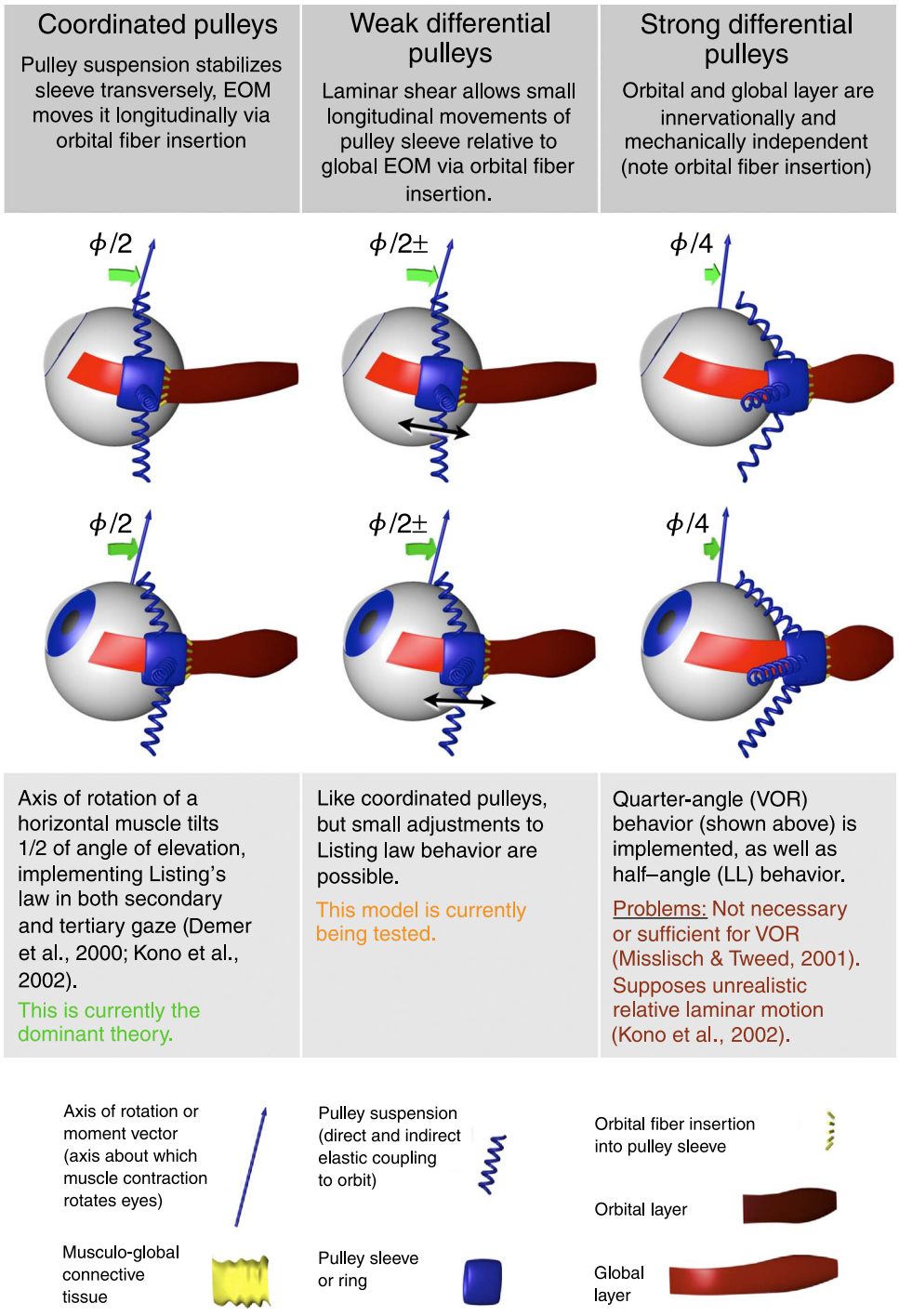

Figure 4. Active pulleys. Coordinated pulleys move longitudinally, with their muscles, extending the kinematic benefits of pulleys into tertiary gazes. Differential pulleys were hypothesized by Demer et al. (2000), based on the pattern of specializations of orbital and global muscle fibers and their attachments to the surrounding pulley sleeves. One can distinguish a strong version, which supposes complete mechanical independence of orbital and global layers, was intended to explain quarter angle vestibuloocular reflex (VOR) kinematics and was abandoned (Kono et al., 2002a) from a weak version, which supposes only relative laminar shear and which remains to be tested. Under the strong differential model, for a given eye position, the pulley sleeve could occupy the same anterior position as under the coordinated pulley model (see also Figure 2) to give half-angle behavior or, alternatively, the posterior position shown in the right column of this figure, to give quarter angle behavior. Under the weak differential model, only small movements of the pulley sleeve relative to the global EOM are possible, and consequently only small variations in the rotation axis.

Figure 5. Commutativity and noncommutativity. A: Translations are commutative. Walking two blocks north, then one block east (red arrows) brings you to the same position as walking one block east, then two blocks north (blue arrows). Order of translations does not affect final position. B: Rotations are noncommutative. Rotating 90° about an up axis, then 90° about a left axis (red rotation vectors) does not get to the same orientation as rotating 90° about a left axis, then 90° about an up axis (blue rotation vectors). Changing the order of rotations changes the final orientation. (Rotation vectors give directions of rotation according to the familiar "right hand rule.")

that extirpation of the lateral rectus pulley increased the amplitude and velocity of horizontal eye movements elicited by brainstem stimulation. It would have been interesting if they had also measured three-dimensional eye position in tertiary gaze because all active pulley models predict torsional abnormalities in tertiary gaze, but this experiment has not yet been done.

If pulley suspensions are compliant enough to stretch under longitudinal, oculorotary muscle forces, how can they also be stiff enough to stabilize muscles against transverse forces? First, from the "half-angle rule," the muscle path deflection necessary to enforce Listing's law is equal to half the angle of "out of muscle plane" eye rotation (e.g., vertical rotation for horizontal muscle pulleys). For small angles, the force needed to deflect a muscle under tension is scaled by the sine of the angle of deflection (Figure 2). Thus, for an eye elevated 30°, the path of a horizontal rectus muscle would be deflected 15°, which would require the muscle's pulley suspension to provide a sideways force equal to only one fourth of the muscle's tension. Second, there is no reason why the pulley suspension must have isotropic stiffness: it could be longitudinally soft and transversely stiff. In support of this notion, van den Bedem et al. (2005) show that the so-called check ligaments (CLs; also known as faisseaux tendineux), which are aligned longitudinally with their EOMs, have very low stiffness within the normal oculomotor range. Third, there probably is some sliding of the muscle within the pulley sleeve (Ruskell et al., 2005). Finally, to the degree that encapsulated orbital fat stabilizes posterior muscle paths by providing a tunnel through which muscles slide (Schutte et al., 2006), longitudinal, and transverse forces would be independent. Much about the implementation of active pulleys remains to be quantified.

Differential pulleys

Rationale of differential pulleys

It has long been recognized that mammalian rectus EOMs consist of global and orbital layers, having distinct fiber types (Mayr, Gottschall, Gruber, & Neuhuber, 1975; Porter, Baker, Ragusa, & Brueckner, 1995; Scott & Collins, 1973; Shall & Goldberg, 1995). Global fibers extend from the annulus of Zinn to their global insertions, whereas orbital fibers terminate in the region of the pulleys (Oh et al., 2001; Porter et al., 1995; Spencer & Porter, 1988). Light microscopy clearly shows terminal orbital fibers intermingled with pulley collagen (Demer et al., 2000; McClung et al., 2006) toughened with elastin fibrils (Demer et al., 2000). Most orbital fibers are specialized for oxidative metabolism and fatigue resistance, whereas most global fibers are less fatigue resistant and more suited to generating force pulses (Porter et al., 1995). Finally, global fibers show both "pulse" and "step" changes in innervation during saccades, whereas orbital fibers show only step changes (Collins, 1975). Demer et al. (2000) inferred from this pattern of laminar specialization that global fibers were adapted to rapidly accelerate the viscously loaded globe, whereas orbital fibers were adapted to translate and hold elastic pulleys.

Differential pulleys fail to account for vestibuloocular reflex kinematics

The notion of differential pulleys was proposed by Demer et al. (2000) as an explanation that was parsimonious in

the sense that it provided an orbital mechanism for both the half-angle, Listing's law kinematics of fixation, saccadic, and pursuit movements, and also for the non-Listing, quarter angle kinematics of the dynamic rotational vestibuloocular reflex (VOR): pulleys would be positioned as shown in Figure 2 to support half-angle kinematics and would be pulled posteriorly by orbital fibers (modifying Figure 2 so that $D_1 = 3D_2$) to support quarter angle kinematics. The theory requires that orbital and global fiber contractions be mechanically independent, and that they be independently controlled by the brainstem. This is the differential APH in what we call its "strong" form. The *strong differential AHP* was soon shown to be neither necessary nor sufficient to account for VOR kinematics (Misslisch & Tweed, 2001) and was abandoned (without, we note, denying the existence of smaller pulley movements that can be demonstrated):

> The original proposal of differential control of rectus pulleys supposed larger anteroposterior shifts during the VOR than during visually guided eye movements. Although differential control of pulleys as originally proposed no longer appears to be a tenable explanation for the steady state VOR during low frequency head rotation, pulley repositioning transverse to the rectus EOM axes appears to occur during convergence albeit in different directions than originally proposed

(Kono et al., 2002a).

In their critiques of the APH, McClung et al. (2006) and Jampel and Shi (2006) overlooked the notion of coordinated pulleys, supposing that the APH was identical to the notion of strong differential pulleys. McClung et al. cite evidence that connective tissue insinuates global as well as orbital fibers and declare that it disproves the APH. Such evidence does not, of course, bear on the notion of coordinated pulleys and is only weak evidence against differential pulleys because, although global fiber coupling can be found, orbital fiber coupling with pulley tissues clearly predominates (Ruskell, 1989), and only the orbital coupling is strengthened with elastin fibrils (Demer et al., 1995).

According to the theory of passive pulleys, all muscle fibers pass freely through pulley sleeves (or rings), which are, in turn, independently supported by connective tissues. The arrangement is analogous to that of familiar rope and grooved-wheel pulleys, which we tend to think of when we hear the term. That is, passive pulleys are prototypical. Differential pulleys are also prototypical because global fibers slide freely through the pulley sleeve, whereas orbital fibers independently support the sleeve. In contrast, coordinated pulleys, in which global fibers passing through the pulley ring are not free to move relative to orbital fibers supporting the ring, are more like tethers than pulleys: coordinated pulleys are therefore not prototypical. We nevertheless refer to them as pulleys because they implement the critical pulley-like kinematic property of tilting the muscle's action vector as a function of eye rotation. Cognitive psychologists (e.g., Lakoff, 1987; Rosch, 1973) point out that members of a conceptual class differ in typicality (e.g., robins are more "birdlike" than penguins) and have found that prototypical members of a class come to mind more easily and lodge there more stubbornly. This may be why some have overlooked the nonprototypical member of the class of EOM pulley theories. This is a critical error because nonprototypical coordinated pulleys are the basis of the modern theory.

Although vestibular system control of strong differential pulleys is no longer a viable hypothesis, a different sort of vestibular system control of pulley position has recently been demonstrated: MRI has revealed counterrotational movements of the pulley array in response to static head tilt, which appear to be controlled by the oblique muscles (Demer & Clark, 2005). This finding suggests that the rectus pulleys constitute an inner mechanism, which is rotated, like a gimbal, about the orbital axis by the oblique muscles. The utility of this arrangement is not yet clear.

Although abandoned as a theory of VOR kinematics, differential laminar movement appears possible under certain circumstances. It has been shown, for instance, that recession and resection surgeries, which relax and stretch the muscle, respectively, do not significantly alter longitudinal pulley location (Clark & Demer, 2006), and that rotating the globe to hyperextend a rectus muscle moves it relative to its pulley (Clark, Ariyasu, & Demer, 2004). Unless we suppose that these procedures detached the orbital layer pulley insertion, these observations show that orbital and global fibers are sufficiently independent to slide relative to each other, at least under surgical manipulation. The results of Dimitrova et al. (2003) are inconsistent with complete mechanical independence of oculorotary and pulley-translatory muscle lamina because they show that resistance to rotation is reduced when a pulley is removed, but they are compatible with the partial laminar independence of weak differential pulleys and, of course, with coordinated pulleys.

Weak differential pulley function remains a possibility

Finally, although complete EOM laminar independence is implausible, as we have noted, differential control of a less dramatic kind, in both innervational and mechanical senses, is not: There may be functionally significant shear through the depth of EOMs. For example, fibers on the orbital surface might contract slightly more or less than fibers on the global surface, with intermediate fiber contraction grading between. With EOM fiber insertions in a pulley sleeve arising predominantly from the orbital layer, the pulley would move longitudinally more or less than the global oculorotary fibers passing through it, perhaps thereby refining the kinematics that would otherwise be produced by rigidly coordinated control. We refer

to such mechanisms as *weak differential pulleys* (Figure 4). The existence of significant laminar shear remains an empirical question and is currently under investigation (Miller, Rossi, Wiesmair, Alexander, & Gallo, 2006).

What is an insertion?

Perhaps the most disconcerting critique of the APH concerns the notion of "dual insertions." Demer et al. (2000) write,

> [T]he orbital layer of each rectus EOM inserts on its corresponding pulley, rather than on the globe. Only the global layer of the EOM inserts on the sclera. This dual insertion was visualized in vivo by MRI in human horizontal rectus EOMs

(p. 1280, abstract).

Admittedly, it is possible to read these remarks about "insertions," think of long, discrete musculotendinous extensions, and to form a mental image of rectus muscle orbital fibers coursing anteriorly alongside global fibers, turning temporally to depart from those global fibers, becoming tendinous, and finally inserting some distance away in a pulley. One might then look at the MR images in Figures 1 and 2 of Demer et al. (2000), where the dual insertions are said to be visualized, to see dark tissue projections from the orbital side of the anterior recti (McClung et al. refer to them as "check ligaments"), and imagine that Demer is claiming these "CLs" to contain or consist of the departing orbital fibers, en route to some distant connective tissue pulley insertion. Our Figure 6 shows a detail view of one of Demer et al.'s images, with the EOM and the "CL" outlined for clarity of discussion. Thus, McClung et al. (2006) write,

> The CL is the band of tissue present on the MRI images, but was previously [i.e., in Demer et al. (2000)] described as the orbital layer insertion for the active pulley hypothesis

(p. 202, column 1). This misunderstanding leads them to look for orbital muscle fibers in the "CL" and, failing to find any, to mistakenly believe they have refuted Demer et al.'s notion of dual insertions:

> The CL can be seen coursing away from the orbital side of the muscle.… Note that no muscle fibers can be observed following the CL along its orbitally directed course

(p. 203, Figure 1 caption). Demer et al., however, contains many clarifications that might have dislodged this misunderstanding, for example,

> the *junction* [italics added] of the dark band [the "CL"] with the LR corresponds to the insertion of the LR orbital layer on its pulley

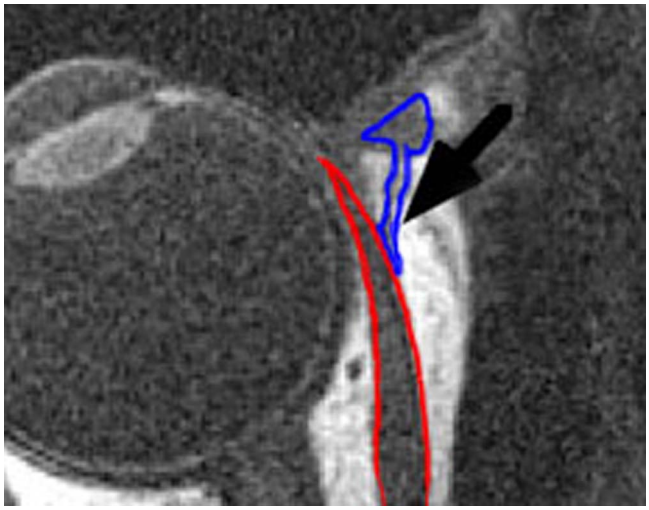

Figure 6. Medial rectus pulley insertion, adapted from Figure 1a of Demer et al. (2000). We have outlined the muscle in red, and the so-called "check ligament (CL)" in blue. McClung et al. (2006) misunderstand Demer et al.'s proposal to be that the blue-outlined "CL" consists of orbital muscle fibers running to a distant periosteal insertion. To the contrary, their proposal is that terminating orbital fibers and overlying connective tissues interdigitate at the junction of the red- and the blue-outlined regions. The large black arrow (in the original figure), intended to point to this junction, was apparently mistakenly seen as pointing to the "CL" as a whole.

(p. 1283, column 1). Finally, Figures 3, 5, 6, 7, and 8 by Demer et al. unambiguously show that the orbital fiber insertion consists of the interdigitation of terminal orbital layer fibers with immediately overlying pulley connective tissue.

Pulley sleeves (or rings) have been distinguished from pulley suspensions to avoid such confusions. McClung et al. (2006) reasonably describe the pulley sleeve as a "tubelike sheath," Tenon's capsule "reflect[ed] back as a sleeve around the muscle" (p. 202, column 1). The pulley suspension, in contrast, is the complex of connective tissues that suspend the pulley sleeve from the orbital wall, from the pulleys of other EOMs, and from other extraocular tissues. The "classical" description of this suspensory complex was given by Koornneef (1992; see our Figure 3, left panel). Our abstraction of this anatomy, with specification of constituent tissues based on immunohistochemistry, is diagramed in Demer et al. (1995; see our Figure 3, right panel). Anatomically, the "CL" would be considered part of the pulley suspension, although we doubt that it has the implied functionality (van den Bedem et al., 2005).

Thus, McClung et al. (2006) confused pulley sleeves with pulley suspensions: they misunderstood Demer et al.'s (2000) proposal to be that the pulley suspension, part of which is the tissue band they call the "CL," contains orbital fibers en route to an insertion in some distant

pulley. Demer et al.'s proposal is actually that terminal orbital fibers insert in the overlying tissues of the pulley sleeve (or ring), not the suspension. For example, referring to a gross anatomic sample, in which a pulley sleeve is stretched away from the orbital surface of its muscle, Demer et al. write,

> Figure 3 shows a surgical exposure of the MR, and illustrates multiple dense, white fibrous bands extending from the orbital surface of the MR muscle and inserting into the glistening white tissue on its nasal [orbital] side. This adjacent connective tissue was confirmed in cadaveric material to form the *pulley ring encircling the MR* [italics added]

(p. 1283, column 2).

Demer et al. (2000) are fully aware of the part of the suspension that McClung et al. (2006) call the "CL," and that it is distinct from the pulley sleeve:

> Favorable image planes … consistently demonstrated the presence of one or more dark bands running anteriorly and peripherally toward the orbital rim. Histologic evidence indicates that this dark band represents the *connective tissue suspension of the corresponding EOM pulley* [italics added]

(p. 1281, column 2).

It is interesting to compare the high magnification histology presented by McClung et al. (2006) in their Figure 1D with that presented by Demer et al. (2000) in their Figure 7B. The former are described by McClung et al. as "demonstrating the CL blending into the orbital side of the muscle by investing collagen filaments around the peripheral (orbital) muscle fibers," the latter by Demer et al. as the "insertion of rectus orbital layer fibers on their respective pulleys." The sections are actually very similar, with McClung et al. clearly showing orbital fiber termination short of the sclera, near the pulleys, and interdigitation of terminal orbital fibers with overlying connective tissue. Whether orbital fibers insert in pulley tissues (Demer et al., 2000) or pulley tissues invest orbital fibers (McClung et al., 2006) is, as the lawyers say, "a distinction without a difference." The interdigitation, clearly shown in McClung et al.'s histology, is precisely the insertion of orbital fibers in pulley connective tissues proposed by Demer. Thus, the data of McClung et al. strongly support both the coordinated and weak differential pulley models.

Conclusion

We have shown that McClung et al. (2006) challenged only the differential APH (the strong form of which was abandoned in 2002; the weak form of which is admittedly conjectural), overlooking the well-supported coordinated APH (Figure 4, coordinated pulleys), which is the basis of the modern theory. We have also shown that their arguments against separate orbital fiber insertions are as irrelevant to the APH as their histological data are supportive. We clarified that the presumptive role of SM is to stiffen pulleys and possibly to refine stiffness and position, and that a focus on direct musculo-orbital connections is misguided. We clarified that noncommutativity is a problem the oculomotor system must solve. We briefly sketched the developmental stages of modern extraocular biomechanics: (1) passive pulleys, (2) coordinated active pulleys, and differential active pulleys in their (3) strong and (4) weak forms, urging both supporters and critics to distinguish which theory they are addressing. We indicated something about the status of each theory and where additional work might usefully be done.

Is the brain necessary?

Coordinated EOM pulleys (Figure 4) account for half-angle, Listing's law kinematics of the eye during fixation, saccades, and pursuit with convergence relaxed and head upright and fixed. There is currently no reason to think the brain has any role in determining torsion in these situations and strong evidence that it does not (Ghasia & Angelaki, 2005; Klier et al., 2006). It is possible, although it remains to be demonstrated, that small adaptive and other changes to half-angle behavior can be effected through pulleys (Figure 4, weak differential pulleys) and by modulating the tonus of extraocular SM (Miller et al., 2003). The kinematic role of the classical brainstem pathways then becomes that of overriding Listing's law in convergence and during head movement.

Similarly, although coordinated pulleys "commutize" the orbit, making it possible for the eye to be controlled without tight coupling of horizontal and vertical gaze centers, the brain is not thereby relieved of noncommutative computations in other visuomotor connections (e.g., Crawford & Guitton, 1997; Crawford et al., 2003; Klier et al., 2006; Tweed et al., 1999).

Buzzwords and sound bites

We have shown that much of the controversy surrounding EOM pulleys has been due to misunderstanding. Where concepts are stable and novelty unlikely, it may be convenient to assume that familiar words and phrases refer to familiar notions. But where concepts are developing and nomenclature strains to cover new meanings, it is inappropriate to think in terms of buzzwords (such as "insertion" and "pulley") because their conventional, prototypical referents predate and therefore tend to obscure new ideas.

Neuroscience context

Pulley theory developed in response to mounting problems with the central bias of oculomotor neuroscience (Porter, Karathanasis, Bonner, & Brueckner, 1997). It was widely appreciated that three-dimensional eye movement kinematics placed very complex demands on a central system tasked with controlling a simple plant, and that these demands were fundamentally different from those successfully met by the earlier one-dimensional models (e.g., Tweed & Vilis, 1987, Tweed et al., 1999). Robinson (1994) worried that modeling of central mechanisms might have reached a point of diminishing returns because many control mechanisms seemed essentially distributed and not amenable to models that implicated localizable physiologic mechanisms. Pulley theory has reoriented oculomotor physiology in an important way: functionality once assumed buried in the brainstem is now known to be exposed in the periphery. Consequently, researchers can look to peripheral biomechanics for answers previously sought in brainstem neurophysiology. Suitable methods to support these efforts must now be developed, refined, and propagated (e.g., Gallo, Ai, Alexander, Miller, 2006; Miller, Bockisch, & Pavlovski, 2002, Miller et al., 2003, Miller et al., 2006).

Clinical context

This reorientation has clinical implications. If the plant were simple and most oculomotor mechanisms supernuclear, most disorders would be inaccessible to direct treatment. In contrast, functions localized in the oculomotor periphery are more readily subject to pharmacologic, surgical, and genetic manipulations. A start has been made in applying the new view of extraocular connective tissue mechanics to surgical treatment of strabismus (Clark et al., 2004; Clark & Demer, 2002; Clark, Isenberg, Rosenbaum, & Demer 1999; Pirouzian, Goldberg, & Demer, 2004). Detailed understanding of muscle stretching and contraction, of muscle fiber and connective tissue specializations, and of other overlooked articulations of the peripheral oculomotor system might make it possible to effect more subtle changes in muscle action than those currently available to treat strabismus and related disorders.

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