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The nonlinearity of passive extraocular muscles

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Passive extraocular muscles (EOMs), like most biological tissues, are hyperelastic, that is, their stiffness increases as they are stretched. It has always been assumed, and in a few occasions argued, that this is their only nonlinearity and that it can be ignored in central gaze. However, using novel measurement techniques in anesthetized paralyzed monkeys, we have recently demonstrated that EOMs are characterized by another prominent nonlinearity: the forces induced by sequences of stretches do not sum. Thus, superposition, a central tenet of linear and quasi-linear models, does not hold in passive EOMs. Here, we outline the implications of this finding, especially in light of the common assumption that it is easier for the brain to control a linear than a nonlinear plant. We argue against this common belief: the specific nonlinearity of passive EOMs may actually make it easier for the brain to control the plant than if muscles were linear.

Orbital mechanics

Understanding the mechanical properties of the biological tissues that collectively form the oculomotor plant is important for many reasons. First, it is well established that abnormalities in the eye plant can lead to strabismus, a common disorder of ocular alignment.[1] Misalignment of the eyes is an important health problem, as it is one of the leading causes of amblyopia (loss of vision in one eye). Although some forms of strabismus are associated with an excellent surgical outcome,[2] others are still difficult to treat. Undoubtedly, in those cases a deeper understanding of eye plant mechanics could provide guidance for treatment and result in improved outcomes. Second, scientists and clinicians alike can easily measure only the overall "output" of the eye plant: the eye orientation. Reconstructing from this one output the control signals for the six eye muscles (i.e., the output of the neural controller) is impossible unless the properties of the plant (i.e., how it responds to neural inputs) are known. Without

a formal understanding of the eye plant, expressed in terms of a mathematical model, we cannot even know *which* problems the brain needs to solve to control the orientation of the eyes, let alone *how* they are solved. This is a very important issue, because problems that were considered to require complex neural solutions have become less daunting once a proper understanding of the periphery emerged.[3–7]

Because of their importance, it is not surprising that measurements and mathematical models of the oculomotor plant go back over 50 years.[8,9] Unfortunately, understanding eye plant mechanics has proven much more difficult than expected, and even goals that appeared to be within reach long ago[10] have proven elusive. This lack of progress, and in particular the limited success of modeling efforts, has prompted a recent resurgence in the experimental approach to the problem, something advocated long ago by David A. Robinson.[11]

In this renewed quest, different groups have taken different approaches. Simonsz *et al.*[12,13] have focused on developing a finite element model of the

eye plant, with a special interest in the mechanical properties of orbital fat. Demer *et al.*[5,14–18] are investigating specializations within individual eye muscles as well as measuring the constitutive properties of passive muscles and orbital tissues. Whereas these efforts are aimed at finding constitutive equations for orbital tissues, others focus on directly measuring forces and deformations in the eye plant. For example, Miller *et al.*[19] have measured muscle forces in behaving monkeys using implanted force transducers. Dean *et al.*[20–22] measured the time-course with which an artificially rotated globe drifts back toward the rest position and inferred from that the mechanical properties of the plant. For our part, we measured the viscoelastic properties of passive EOMs in nonhuman primates, *in vivo*. In particular, we took advantage of improved measuring devices and somewhat more sophisticated experimental paradigms to address questions that earlier experiments[23] had left unanswered.

While these different approaches have not yet yielded a consistent model of orbital behavior, considerable progress has been made. We will now briefly review some of our findings and discuss their implications for this resurgent field.

The nonlinear behavior of passive EOMs

With our experiments, we confirmed, and accurately quantified, the hyperelastic behavior of EOMs (Fig. 1)—that stiffness increases with muscle elongation.[24] We showed that a four-parameter
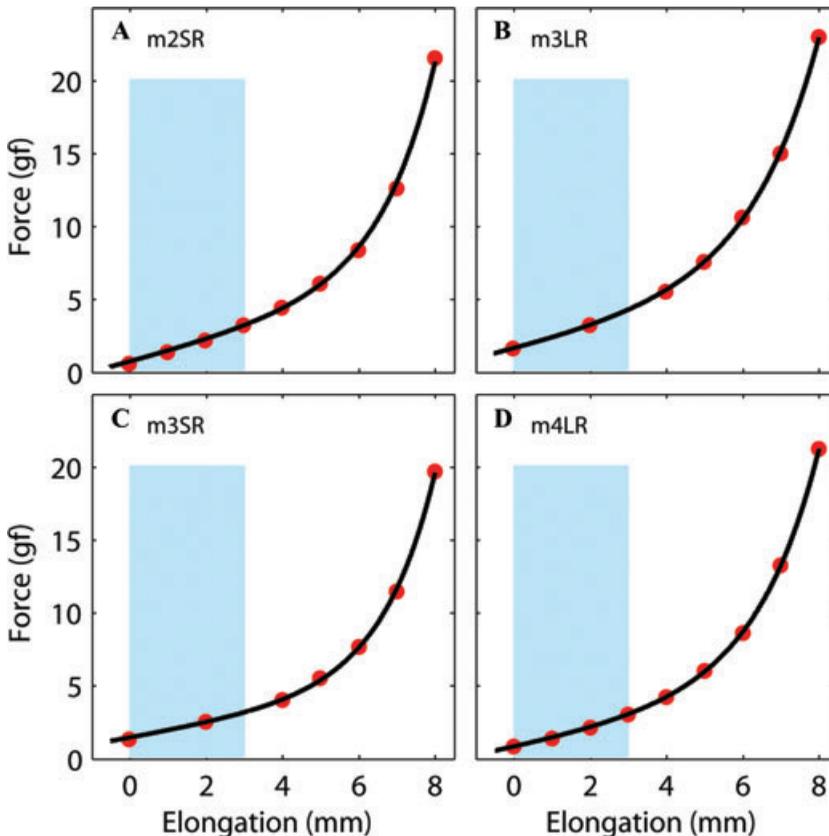

Figure 1. Length–tension relationship for individual passive EOMs (one for each panel, label identifies monkey and EOM). The red points are estimates of the equilibrium force reached by the muscle long after a small (0.5 mm) step-wise elongation at a given length. This estimate is computed by fitting the post-elongation decaying force with a sum of decaying exponentials (the fits were always excellent; an example is shown in Figure 2). The black line is a fit to the data. The fitting function is the sum of an offset, a linear term, and an increasing exponential term. It has four parameters (offset, slope of the linear term, gain of the exponential, and length constant of the exponential). The blue shading represents the elongation range over which a linear function would suffice to fit the data. This range extends to approximately 3 mm.
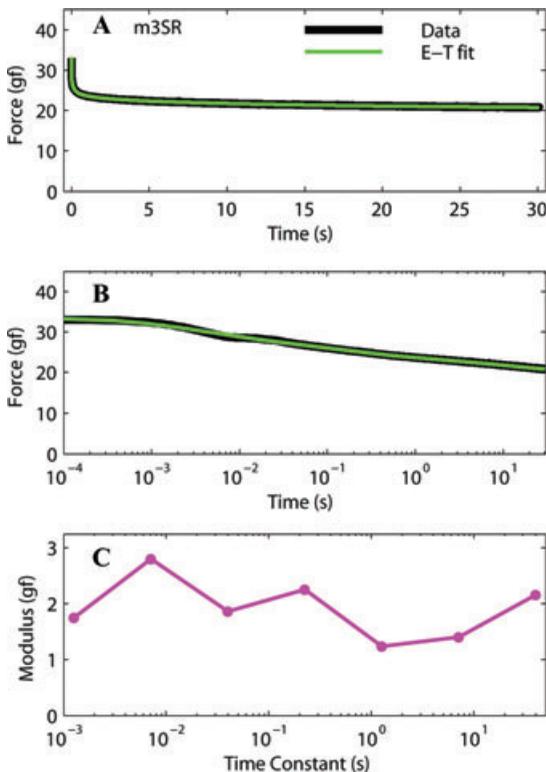
Figure 2. Typical relaxation responses in a passive monkey EOM. (A) Data (black) and fit (green). The fitting function is a sum of seven decaying exponentials, with time constants logarithmically spaced between 1 ms and 40 sec, and a constant (which is used to estimate the length–tension relationship in Fig. 1). The quality of this fit is representative of the entire dataset. (B) Same as A, but using a logarithmically spaced abscissa to improve visualization of the force at short times. (C) Moduli associated with each time constant in the fit. Note that there is no dominant time constant.

function, comprising a linear and an exponential term, fits the length–tension curve extremely well in all cases. This suggests the presence of an initial linear range (i.e., constant stiffness, shaded blue in Figure 1) of approximately 3 mm, corresponding to eye rotations of about 15°, followed by a hyperelastic range. The curves were remarkably similar across animals and muscles.

Next, we showed that, like virtually all passive biological tissues,[25] the relaxation response following a step-wise elongation can be fit by a sum of decaying exponential functions, with time constants ranging from 1 ms to 40 sec (Fig. 2). While there is quite a lot of variability across animals, muscles, and muscle elongations, all time constants were equally

represented (Fig. 2C). In fact, the range of time constants might be even wider, as our measurements were limited by resolution on the low end, and by the duration of the experiments on the high end. Evidence for longer time constants has in fact been found, albeit *in vitro*.[15] These findings contradict the long-held view that a passive EOM can be reasonably modeled as a first-order linear system.[20–23] Together with the above-mentioned hyperelasticity, our results thus indicate that no model simpler than a quasi-linear model with a wide relaxation spectrum[25,26] could possibly reproduce the force changes (stress) induced by length changes (strain) in passive EOMs. Such a model can be described[27] as a static nonlinearity (proportional to the length–tension curve) followed by a linear system (with an order equal to the number of time constants). This class of (nonlinear) models is often referred to as a NL (nonlinear-linear), or Hammerstein, cascade.

One property of NL cascades is that they obey superposition: if two elongations are applied sequentially, the forces induced do not interact, but simply sum. All models that are described by a single-integral form, of which the quasi-linear formulation is a special case, share this property. The terms *modified superposition* or *nonlinear superposition*[28,29] are commonly used to refer to this class of models. However, this property (which is also one of the cornerstones of linear systems) has received very little attention and to our knowledge has been tested in only a few man-made materials[28,29] but not in any biological tissue. This paucity of data is in a sense quite surprising, since most of our daily behavior involves a series of closely spaced movements, in which second- or higher-order interactions could play a prominent role. We felt that this was an especially crucial question for the eye plant, given the long time constants at play. For example, in passive EOMs, after a step-wise elongation it takes around 4 sec for the force to decay to 10% of its peak value, but successive refixations are usually separated by less than 300 ms.

To test for superposition, we thus applied sequences of two step-wise elongations, varying the interval between the two steps from 45 sec (when the two elongations can be considered approximately independent) down to 10 ms. Starting from the force induced by the two widely separated steps, superposition predicts the force following the second step for any other interstep interval. Intuitively, the
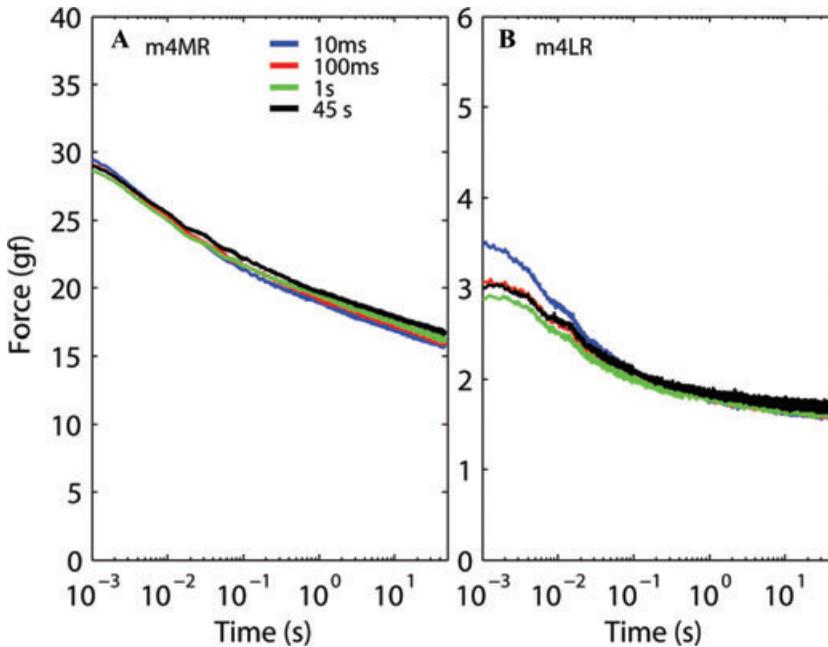
Figure 3. Force induced by the second step in a double-step sequence (semilog scale). (A) Data from elongations at long lengths (note large forces). The force induced by the second elongation step is not a function of the interstep interval (ISI, see legend). (B) Data from elongations at short lengths. With the exception of the first 20 ms after the shortest ISI, the force induced by the second elongation step is invariant. The muscle thus appears to have no memory of the previous elongation.

closer together the two steps are, the higher the force following the second step, since the force induced by the second step essentially "sits on top" of the decaying force induced by the first step. Our measurements[27] revealed that passive monkey EOMs do not obey superposition: the force induced by the second step is invariably lower than that predicted by superposition. Importantly, this occurs not only at large elongations, but also at small elongations, within the region in which the length–tension relationship is essentially linear.

We have thus demonstrated that passive EOM behavior is not captured by either linear or nonlinear superposition and that it falls instead in the class that is informally described as highly nonlinear. To model such materials, one must resort to a more general multiple integral nonlinear formulation.[29,30] Models of this type are, however, very difficult to formulate because finding their parameters (which are actually multidimensional functions called kernels) requires a prohibitively large number of experiments. This would seem to represent an insurmountable obstacle to finding a reasonably accurate model of eye muscles, but all hope is not

lost. It turns out that EOMs violate superposition in a very well-behaved way: the force following the second step is always the same (Fig. 3), regardless of the interstep interval (at least starting 20 ms after the end of the second step). This holds at both large (Fig. 3A) and small (Fig. 3B) elongations. In other words, the force decay after an elongation is determined almost exclusively by the antecedent elongation, regardless of the previous elongation history: passive EOMs have a very short memory.

To further characterize this nonlinear behavior, we measured the force induced by elongations of different extents, but with the final muscle length in common.[31] For these elongations, we did not use quick steps, but rather half-sinusoidal velocity profiles, approximating those experienced by the antagonist muscle during saccades. Peak speed and amplitude were drawn from the typical main sequence relationship. As expected from a viscoelastic material, speed and extent of the elongation greatly affected the force generated *during* the length change. However, once the elongation was over, the forces induced by the various elongations quickly converged (Fig. 4), and within 100 ms their
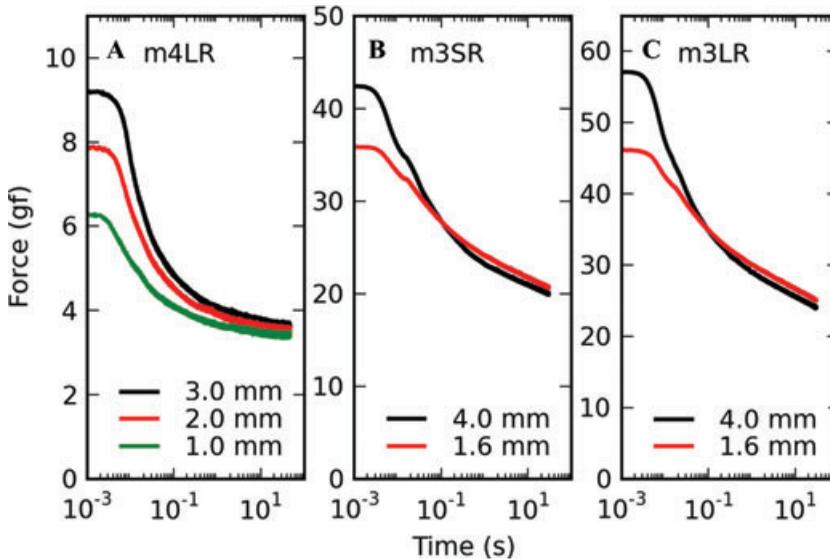
Figure 4. Forces induced by saccade-like elongations (i.e., elongations with a half-sinusoid velocity profile) characterized by different amplitudes but with a common final elongation. (A) 1 (green), 2 (red), and 3 (black) mm elongations terminating at an intermediate elongation. Note how the decay phase is almost independent of the elongation after 100 ms. (B) 1.6 (red) and 4 (black) mm elongations terminating at the largest elongation tested. Note how during the decay phase the forces quickly converge, becoming indistinguishable. (C) Same as B, but in a different muscle.

difference, which could be as large as 10 g at the end of the elongation, dropped to a fraction of a gram. It would thus seem that the post-elongation decay not only is little affected by previous history, but also is mostly independent of the kinematics of the last elongation. The final length, and presumably the direction of the last length change, mostly account for the decay in force.

We have since replicated these results in rabbit passive EOMs. Thus, this phenomenon is not species specific.

Implications for neural control

Historically, nonlinearities have never been particularly popular among scientists. The main reason for this unpopularity is that while we have very sophisticated tools to analyze and describe linear systems, our toolbox for studying nonlinear systems is very light, and the few tools we do have are very hard to use. Three strategies have been employed to get around these problems. The first approach is to focus on those nonlinear behaviors that are more amenable to examination. The large popularity of the QLV model and of nonlinear superposition in general, with its corresponding focus on a single

elongation, is an example of this approach. The second approach is to limit the study of the nonlinear system to a small range over which the behavior appears linear. This approach has been very successful, and there are examples of small-signal analyses in all fields of science. The third approach is to develop sophisticated linear analysis techniques that are tailored to a certain class of nonlinear systems (usually cascades of linear systems and static nonlinearities) and are capable of isolating the linear component of the underlying system. This approach is particularly popular in the analysis of neural responses. These techniques are ingenious, and their application has resulted in enormous progress in understanding natural phenomena. However, they also have a downside: they have contributed to the widely held belief that nonlinearities are inherently difficult and, correspondingly, that nonlinear systems are hard, or at least harder, to control than linear systems. For example, Anderson *et al.*,[22] while discussing the nonlinearity associated with EOM activation, concluded, "How these nonlinearities might be mitigated by, for example, recruitment to make eye-movement control simpler is an important topic for further work." We would like instead to argue

the opposite position, i.e., that proper nonlinearities might in fact lighten the burden of the neural controller.

The first reason for arguing in favor of nonlinearities is evolutionary: if nonlinearities in biological materials were detrimental, evolution would have disposed of them a long time ago. Conversely, if they are there they either serve a purpose or at least are irrelevant. A classic example of this type of specialization is represented by tendons and ligaments, which are composed of a set of collagen fibers arranged in parallel. The individual fibers behave linearly (at least within the physiological range of elongations); however, a "toe" region, where stiffness is low and increases nonlinearly, often characterizes the composite structure. This occurs because different collagen fibers have different lengths, so that as the load increases additional fibers are recruited.[32–34] Once all the fibers have been recruited, the whole structure behaves linearly. This mechanism, which helps to smooth out stress build-up and thus protects the muscle, is so important that many muscle–tendon groups operate mostly in the toe region. However, this compliance would be very detrimental for digital flexors and EOMs, whose length must be rapidly and precisely controlled: not surprisingly, evolution took care of that, and the tendons of those muscles are much stiffer.

We believe that the nonlinearities that we have discovered in EOMs have also evolved to serve a purpose: simplifying neural control. To see how, let's consider a muscle whose viscoelastic behavior conforms to the QLV model, and thus within the first 3 mm of elongation is actually linear (but of high order). According to Dean *et al.*,[20–22] such a model actually describes the zero-load creep behavior of the whole orbit (i.e., the time course with which an eccentrically rotated eyeball passively returns to the rest position once released). Every time the eye rotates, the lengths of muscles and orbital tissues, as well as the forces that they exert, change. Once an eye rotation is over, the lengths do not change anymore, but the passive forces still do, and they do so for a long time. As all these forces are ultimately applied to the eyeball, this would inevitably lead to eye motion, that is, eye drift. Of course, this would be unacceptable, since vision would be severely degraded. There is only one way to avoid this: the brain must generate active forces that compensate for these passive forces. Hence, the inner-

vation to the eye muscles must keep changing long after the eye movement is over, or the eyes would keep moving. These innervational components have indeed been observed in motoneurons,[35,36] and the forces have been measured in muscles.[36,37] Generating these innervational signals would not be a trivial matter: because of superposition, the brain would need to build an internal model of the eye plant, separately for tissues and muscles, incorporating processes with long time constants. Only by doing so could it keep track of the ever-changing internal state of each muscle and tissue. With time constants of at least 40 sec, this would not be a small feat. Moreover, the level of accuracy required to use retinal slip (the error signal) to solve the credit assignment problem (i.e., to figure out which parameter needs to be tweaked) for such a model is extraordinary.[38] And yet the brain is capable of using the retinal slip to cancel postsaccadic drift: repeated presentation of an artificially induced postsaccadic retinal slip will, over time, induce a postsaccadic eye drift with a magnitude, and time constant appropriate to cancel that retinal slip.[39,40] Moreover, this drift can be induced not only in the same or opposite direction of the antecedent saccadic eye movement,[39] but even in a direction orthogonal to it.[41]

A nonlinearity such as the one we discovered in passive EOMs opens the door to a different strategy: the brain could simply issue a learned postsaccadic innervational command based on the final eye position and the direction of the antecedent movement. There would be no need to keep track of internal states of muscles and tissues, and thus there would be no credit assignment problem. It would also be very easy to dissociate the direction of the antecedent movement and that of the eye drift. Under this scheme, the floccular complex, which is required for retinal slip cancellation,[42] would be in charge of monitoring retinal slip and associating eye movements with an appropriate postsaccadic signal to be sent to the extraocular motoneurons.

One major caveat of this scheme is that it can only work if all the tissues that exert (significant) torques on the eyeball behave similarly. If any of the tissues were to behave linearly (i.e., had a long memory) the brain could not take this shortcut. Unfortunately, there is currently little evidence to support or reject this conjecture. EOM tendons are extremely stiff,[43] so they certainly do not play a significant role. However, the rest of the extraocular

tissues (usually referred to as orbital tissues, OTs) are not only poorly characterized from a mechanical standpoint, but also poorly defined: it is not at all clear which tissues contribute to OT torque and which do not. As we noted above, to the best of our knowledge the mechanical properties of the eye plant with all six EOMs fully detached have not been studied. Of course, such a procedure could not be carried out without causing damage to the conjunctiva, so its force contribution would have to be separately assessed. It has been shown,[44] and we have confirmed (unpublished observations), that when the horizontal recti are detached and the eye is rotated, it does not drift back all the way to its original resting position. This seems to imply that OT forces in central gaze are very small, but once again those experiments damaged at least the conjunctiva. As mentioned above, Dean *et al.*[20–22] have argued that the whole plant can be modeled as a linear system, but their conclusion was based on the results of zero-load creep. This type of paradigm is usually avoided in material testing because some nonlinear systems can behave linearly when the load is removed (removing the forcing function can turn some nonlinear differential equations into essentially linear ones). Furthermore, it has been long recognized that it is extremely difficult to predict relaxation from creep in biological tissues,[25,33,45–51] and while creep can be important in ligaments,[51] which are often subjected to repetitive load histories, it is hard to imagine under what physiological condition creep would be an important orbital phenomenon. Finally, Dean *et al.* have *inferred* linearity from a small set of single elongations, which were not designed to explicitly test superposition. Understandably, they thus had to introduce a set of assumptions to draw inferences from their data. However, if one or more of their assumptions were to be proven false, then their conclusions should be revisited. In light of our results, their assumption that passive EOMs are first-order linear systems seems particularly weak. Obviously, more experiments are needed to fully characterize the mechanical properties of the OTs, as well as those of active EOMs.

Here, we have focused our attention on the lack of superposition, not only because of its importance, but also because it can be easily grasped. In our experiments, we have chosen our elongation paradigms so that we could directly *measure* nonlinearity (see Figs. 3 and 4). In addition to this nonlinearity, which affects the post-elongation period, and the hyperelasticity (Fig. 1), which is measured at equilibrium, we found that forces during the elongation are also nonlinear: forces for larger elongations are considerably smaller than expected from a linear system given the forces induced by small elongations.[31]

In conclusion, we believe that there is enough evidence to support the idea that nonlinearities are pervasive in biological tissues, including the eye orbit. Evolution has made it easier for our brain to control our body, but it has not made it easier for scientists to understand how we achieve that control.

Acknowledgments

Conflicts of interest

The authors declare no conflicts of interest.

References

1. von Noorden, G.K. & E.C. Campos. 2002. *Binocular Vision and Ocular Motility*. Mosby. St. Louis.
2. Wisnicki, H.J., M.X. Repka & D.L. Guyton. 1988. Reoperation rate in adjustable strabismus surgery. *J. Pediatr. Ophthalmol. Strabismus* **25:** 112–114.
3. Quaia, C. & L.M. Optican. 1998. Commutative saccadic generator is sufficient to control a 3-D ocular plant with pulleys. *J. Neurophysiol.* **79:** 3197–215.
4. Miller, J.M. 1989. Functional anatomy of normal human rectus muscles. *Vis. Res.* **29:** 223–240.
5. Peng, M., V. Poukens, R.M. da Silva Costa, *et al.* 2010. Compartmentalized innervation of primate lateral rectus muscle. *Invest. Ophthalmol Vis. Sci.* **51:** 4612–4617.
6. Demer, J.L. 2006. Current concepts of mechanical and neural factors in ocular motility. *Curr. Opin. Neurol.* **19:** 4–13.
7. Schreiber, K., J.D. Crawford, M. Fetter, *et al.* 2001. The motor side of depth vision. *Nature* **410:** 819–822.
8. Westheimer, G. 1954. Mechanism of saccadic eye movements. *AMA Arch. Ophthalmol.* **52:** 710–724.
9. Robinson, D.A. 1964. The mechanics of human saccadic eye movements. *J. Physiol.* **174:** 245–264.
10. Simonsz, H.J. & H. Spekreise. 1996. Robinson's computerized strabismus model comes of age. *Strabismus* **4:** 25–40.
11. Robinson, D.A. 1981. Models of the mechanics of eye movements. In *Models of Oculomotor Behavior and Control*. B.L. Zuber, Ed.: 21–41. CRC Press. Boca Raton, FL.
12. Schoemaker, I., P.P. Hoefnagel, T.J. Mastenbroek, *et al.* 2006. Elasticity, viscosity, and deformation of orbital fat. *Invest. Ophthalmol. Vis. Sci.* **47:** 4819–4826.

13. Schutte, S., S.P.W. van den Bedem, F. van Keulen, *et al.* 2006. A finite-element analysis model of orbital biomechanics. *Vis. Res.* **46:** 1724–1731.

14. Lim, K.H., V. Poukens & J.L. Demer. 2007. Fascicular specialization in human and monkey rectus muscles: evidence for anatomic independence of global and orbital layers. *Invest. Ophthalmol. Vis. Sci.* **48:** 3089–3097.

15. Yoo, L., H. Kim, V. Gupta, *et al.* 2009. Quasilinear viscoelastic behavior of bovine extraocular muscle tissue. *Invest. Ophthalmol. Vis. Sci.* **50:** 3721–3728.

16. Demer, J.L., V. Poukens, H. Ying, *et al.* 2010. Effects of intracranial trochlear neurectomy on the structure of the primate superior oblique muscle. *Invest. Ophthalmol. Vis. Sci.* **51:** 3485–3493.

17. Yoo, L., V. Gupta, C. Lee, *et al.* 2011. Viscoelastic properties of bovine orbital connective tissue and fat: constitutive models. *Biomech. Model Mechanobiol.* doi: 10.1007/s10237-010-0281-z.

18. Yoo, L., J. Reed, A. Shin, *et al.* 2011. Characterization of ocular tissues using micro-indentation and hertzian viscoelastic models. *Invest. Ophthalmol. Vis. Sci.* **52:** 3475–3482.

19. Miller, J.M., C.J. Bockisch & D.S. Pavlovski. 2002. Missing lateral rectus force and absence of medial rectus co-contraction in ocular convergence. *J. Neurophysiol.* **87:** 2421–2433.

20. Sklavos, S., J. Porrill, C.R.S. Kaneko & P. Dean. 2005. Evidence for wide range of time scales in oculomotor plant dynamics: implications for models of eye-movement control. *Vis. Res.* **45:** 1525–1542.

21. Sklavos, S., D.M. Dimitrova, S.J. Goldberg, *et al.* 2006. Long time-constant behavior of the oculomotor plant in barbiturate-anesthetized primate. *J. Neurophysiol.* **95:** 774–782.

22. Anderson, S.R., J. Porrill, S. Sklavos, *et al.* 2009. Dynamics of primate oculomotor plant revealed by effects of abducens microstimulation. *J. Neurophysiol.* **101:** 2907–2923.

23. Collins, C.C. 1971. Orbital mechanics. In *The Control of Eye Movements.* P. Bach-y-Rita, C.C. Collins & J.E. Hyde., Eds.: 283–325. Academic Press. New York.

24. Quaia, C., H.S. Ying, A.M. Nichols & L.M. Optican. 2009. The viscoelastic properties of passive eye muscles in primates. I: static forces and step responses. *PLoS One* **4:** e4850, 1–15.

25. Fung, Y.C. 2004. Biomechanics. *Mechanical Properties of Living Tissues.* Springer. New York.

26. Fung, Y.C.B. 1971. Stress–strain–history relations of soft tissues in simple elongation. In *Biomechanics: Its Foundations and Objectives.* Y. Fung, C.B. N. Perrone & M. Anliker, Eds.: 181–208. Prentice-Hall. Englewood Cliffs, NJ.

27. Quaia, C., H.S. Ying & L.M. Optican. 2009. The viscoelastic properties of passive eye muscle in primates. II: testing the quasi-linear theory. *Plos. One.* **4:** e6480, 1–16.

28. Pipkin, A.C. & T.G. Rogers. 1968. A non-linear integral representation for viscoelastic behaviour. *J. Mech. Phys. Solids* **16:** 59–72.

29. Findley, W.N., J.S. Lai & K. Onaran. 1989. *Creep and Relaxation of Nonlinear Viscoelastic Materials.* Dover. New York.

30. Green, E. & R.S. Rivlin. 1957. The mechanics of non-linear materials with memory. *Arch. Ration. Mech. Anal.* **1:** 1–21.

31. Quaia, C., H.S. Ying & L.M. Optican. 2010. The viscoelastic properties of passive eye muscle in primates. III: force elicited by natural elongations. *PLoS One* **5:** e9595, 1–19.

32. Viidik, A. 1972. Simultaneous mechanical and light microscopic studies of collagen fibers. *Z. Anat. Entwick. Gesch.* **136:** 204–212.

33. Thornton, G.M., C.B. Frank & N.G. Shrive. 2001. Ligament creep behavior can be predicted from stress relaxation by incorporating fiber recruitment. *J. Rheol.* **45:** 493–507.

34. Hansen, K.A., J.A. Weiss & J.K. Barton. 2002. Recruitment of tendon crimp with applied tensile strain. *J. Biomech. Eng.* **124:** 72–77.

35. Goldstein, H.P. 1983. *The Neural Encoding of Saccades in the Rhesus Monkey.* The Johns Hopkins University. Baltimore, MD.

36. Davis-Lopez de Carrizosa, M.A., C.J. Morado-Diaz, *et al.* 2011. Dual encoding of muscle tension and eye position by abducens motoneurons. *J. Neurosci.* **31:** 2271–2279.

37. Miller, J.M. & D. Robins. 1992. Extraocular muscle forces in alert monkey. *Vis. Res.* **32:** 1099–1113.

38. Inchingolo, P. & P. Bruno. 1994. Can the phasic and tonico contributions of agonist and antagonist muscles during saccades be identified from the waveforms of the post-saccadic drift? In *Contemporary Ocular Motor and Vestibular Research: A Tribute to David A. Robinson.* A.F. Fuchs, T. Brandt, U. Buttner & D. Zee, Eds.: 200–202. Verlag. Stuttgart.

39. Optican, L.M. & F.A. Miles. 1985. Visually induced adaptive changes in primate saccadic oculomotor control signals. *J. Neurophysiol.* **54:** 940–958.

40. Kapoula, Z., L.M. Optican & D.A. Robinson. 1989. Visually induced plasticity of postsaccadic ocular drift in normal humans. *J. Neurophysiol.* **61:** 879–891.

41. Kapoula, Z., D.A. Robinson & L.M. Optican. 1993. Visually induced cross-axis postsaccadic eye drift. *J. Neurophysiol.* **69:** 1031–1043.

42. Optican, L.M., D.S. Zee & F.A. Miles. 1986. Floccular lesions abolish adaptive control of post-saccadic ocular drift in primates. *Exp. Brain Res.* **64:** 596–598.

43. Pfann, K.D., E.L. Keller & J.M. Miller. 1995. New models of the oculomotor mechanics based on data obtained with chronic muscle force transducers. *Ann. Biomed. Eng.* **23:** 346–358.

44. Pfann, K.D. 1993. *Quantitative Studies of Eye Movement Generation: Biomechanics and Neural Control.* University of California. San Francisco, CA.

45. Fung, J.C.B. 1979. Inversion of a class of nonlinear stress–strain relationships of biological soft tissues. *J. Biomed. Engin.* **101:** 23–27.

46. Lakes, R.S. & R. Vanderby. 1999. Interrelation of creep and relaxation: a modeling approach for ligaments. *J. Biomech. Eng.* **121:** 612–615.

47. Provenzano, P., R. Lakes, T. Keenan & R. Vanderby. 2001. Nonlinear ligament viscoelasticity. *Ann. Biomed. Eng.* **29:** 908–914.

48. Provenzano, P.P., R.S. Lakes, D.T. Corr & R. Vanderby. 2002. Application of nonlinear viscoelastic models to describe ligament behavior. *Biomech. Model Mechan.* **1:** 45–57.

49. Oza, A., R. Vanderby & R.S. Lakes. 2003. Interrelation of creep and relaxation for nonlinearly viscoelastic materials: application to ligament and metal. *Rheol. Acta.* **42:** 557–568.

50. Oza, A., R. Vanderby & R.S. Lakes. 2006. Generalized solution for predicting relaxation from creep in soft tissue: application to ligament. *Int. J. Mech. Sci.* **48:** 662–673.

51. Thornton, G.M., A. Oliynyk, C.B. Frank & N.G. Shrive. 1997. Ligament creep cannot be predicted from stress relaxation at low stress: a biomechanical study of the rabbit medial collateral ligament. *J. Orthop. Res.* **15:** 652–656.