Published in final edited form as:

Prog Biophys Mol Biol. 2010 December ; 103(2-3): 273–283. doi:10.1016/j.pbiomolbio.2010.09.002.

Physically-based Modeling and Simulation of Extraocular Muscles

Qi Wei^{a,b}, Shinjiro Sueda^b, and Dinesh K. Pai^{b,*}

^aFeinberg Medical School, Northwestern University, 303 E. Chicago Ave., Chicago, IL, USA

^bSensorimotor Systems Laboratory, Department of Computer Science, The University of British Columbia, 2366 Main Mall, Vancouver, BC, Canada

Abstract

Dynamic simulation of human eye movements, with realistic physical models of extraocular muscles (EOMs), may greatly advance our understanding of the complexities of the oculomotor system and aid in treatment of visuomotor disorders. In this paper we describe the first three dimensional (3D) biomechanical model which can simulate the dynamics of ocular motility at interactive rates. We represent EOMs using “strands”, which are physical primitives that can model an EOM's complex nonlinear anatomical and physiological properties. Contact between the EOMs, the globe, and orbital structures can be explicitly modeled.

Several studies were performed to assess the validity and utility of the model. EOM deformation during smooth pursuit was simulated and compared with published experimental data; the model reproduces qualitative features of the observed non-uniformity. The model is able to reproduce realistic saccadic trajectories when the lateral rectus muscle was driven by published measurements of abducens neuron discharge. Finally, acute superior oblique palsy, a pathological condition, was simulated to further evaluate the system behavior; the predicted deviation patterns agree qualitatively with experimental observations. This example also demonstrates potential clinical applications of such a model.

Keywords

physically-based modeling; extraocular muscle; interactive simulation; soft tissue motion; eye movement; biomechanics

1. Introduction

Vision is one of our most important senses. Rapid and accurate eye movements are crucial for coordinated direction of gaze. Studying human eye movement has significant implications for improving our understanding of the oculomotor system and treating visuomotor disorders. Computational simulation has played an important role in analyzing the mechanisms of ocular motility. In particular, having a realistic model of the extraocular muscles (EOMs) and simulating their biomechanics in three dimensions (3D) are necessary for investigating the function and neural control of EOMs. In this paper, we present a 3D

*Corresponding author. Tel: +1 604 822 8197; fax: +1 604 822 0848, pai@cs.ubc.ca (Dinesh K. Pai).

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biomechanical model for realistically simulating extraocular muscle motion at interactive rates.

Various computational models of the extraocular muscle and orbital mechanics have been proposed, which provide insight and scientific bases for oculomotor biomechanics, control of eye movement, and binocular misalignment. Early one-dimensional biomechanical models were based on physiological data and laid the groundwork for the subsequent studies [Clark and Stark, 1974; Collins, 1971; Collins et al., 1975; Robinson, 1964, 1981]. These models characterize the viscoelastic properties of the EOMs, based on experimental measurements such as the tension-length relationship of the EOMs [Robinson, 1964]. They focus on the realism of muscle behaviors in the horizontal direction, where the data were acquired. Extension to three dimensions is not trivial. As a matter of fact, these dynamic models have only been simulated in 1D. However, to study the kinematics and neural control of the orbital plant, it is indispensable to analyze its rotation in 3D. The above models are not easy to apply in more general situations.

The first 3D biomechanical model was developed by Robinson [1975], who simplified the formulation by only considering the elasticity of the EOMs and ignoring the dynamics. Equilibrium of the net force on the eyeball was analytically calculated, and static fixations in 3D were studied. The model incorporates anatomically realistic muscle paths and empirical EOM innervation-length-tension relationships to study normal binocular alignment, strabismus, and surgical corrections. Two models were developed that improved Robinson's model: Simonsz's model [Simonsz and Spekreijse, 1996] and the SQUINT model [Miller and Robinson, 1984]. The latter became an iterative software tool – the *Orbit*^{TM} gaze mechanics simulation [Miller et al., 1995]. Recently, Quaia et al. [2008] presented simulation of superior oblique palsy using a model refined from their previous work [Quaia and Optican, 2003]. All of the above models in 1D or 3D are lumped biomechanical models: the action of each EOM is simplified as a single force vector without taking into account the distribution of material properties and forces along the EOMs. These lumped models also do not account for muscle mass properly [Pai, 2010]. Furthermore, contact between the EOMs and the globe cannot be dealt with. Schutte et al. [2006] proposed an orbital model based on 3D finite element model (FEM). The EOMs were assigned constant stiffness and contact is handled to a limited extent by approximating the interface as a layer with low elasticity.

One major limitation of the 3D biomechanical models described above is that only static eye positions can be simulated. To study the neural control of rapid saccadic movement, models using simplified anatomical and mechanical properties of EOMs have been developed [Quaia and Optican, 1998; Raphan, 1998; Schnablock and Raphan, 1994]. These models assume that three pairs of EOMs act in three planes that are orthogonal to each other. They do not take into account the anatomical variations of different EOMs, such as muscle lengths and cross sectional areas. Another critical assumption made to simplify the analytical solution is on the nature of the EOM force – the active force is assumed to be simply proportional to the muscle innervation, whereas the actual EOM force is a complex nonlinear function of the muscle length, velocity, and innervation. Such models have the advantage of supporting 3D dynamic simulations and have been used to analyze neural controllers and the pulley hypothesis [Quaia and Optican, 1998; Raphan, 1998]. These simplifications, however, limit the models' accuracy and plausibility, as has been illustrated by [Quaia and Optican, 2003].

In this paper, we describe a novel 3D biomechanical simulator of the extraocular muscles that addresses the drawbacks of the existing models. We use the *spline strand* [Sueda et al., 2008] to model EOM mechanics. Our proposed model has the following desirable properties:

- Strand-based EOMs are associated with realistic subject-specific muscle anatomy, such as muscle paths and cross sectional areas determined from magnetic resonance images of human orbits.
- Complex nonlinear EOM physiological properties are incorporated in the computation by explicitly representing the experimental EOM constitutive models.
- Extraocular connective tissues that stabilize the EOMs in the orbit, called pulleys, can be implemented.
- The model is computationally efficient. It provides a good balance between simulation accuracy and speed. EOM motions can be simulated at interactive rates (~ 16 frames/second) on personal computers.
- Contact between EOMs and the globe is physically handled through sliding constraints, not only for visualization but also for realistically simulating force interaction and lateral sliding movement.
- Our biomechanical model can simulate extraocular muscle dynamics, which have not been supported by other 3D biomechanical models.

In a previous report [Wei et al., 2010], we have presented the structures of our biomechanical model of the orbital plant. In this paper, we focus on describing the detailed methodology and results of the extraocular muscles – the most important components of the whole orbital plant. In the following, we first briefly introduce the anatomy and biomechanics of EOMs in Section 2.1. We then present an in-depth description of our strand-based EOM model in Section 2. To assess the accuracy of our extraocular muscle model, we design several validation experiments. We analyze simulated EOM deformation during smooth pursuit and compare it to motion-encoded magnetic resonance imaging (MRI) data (see Section 3.1). We use empirical neural discharges as the control signal to reproduce saccadic movement (see Section 3.2). Finally, we present simulation of a pathological case to further evaluate the system behavior against experimental data in Section 3.3.

2. Methods

2.1. Anatomy and Biomechanics of Extraocular Muscles

The orbital plant consists of the globe (eyeball), three pairs of extraocular muscles, and connective tissues. Figure 1 shows a 3D model of the human orbit reconstructed from magnetic resonance imaging (MRI) [Wei et al., 2009]. Table 1 summarizes the acronyms used throughout the paper. The six EOMs, including four rectus muscles and two oblique muscles, are controlled by the cranial nerves to generate force, to rotate the globe to track a visual target, and to stabilize the image of the object on the retina.

The four rectus EOMs originate from the annulus of Zinn; they course anteriorly, pass through Tenon's capsule, and insert on the sclera. The lateral rectus (LR) and medial rectus (MR) muscles share a common horizontal plane. Contractions of these two muscles produce horizontal eye movement. The superior rectus (SR) and inferior rectus (IR) muscles form the vertical agonist-antagonist pair, which mainly controls vertical eye movement and also affects rotation about the line of sight (visual axis). The superior oblique (SO) muscle passes through the cartilaginous trochlea attached to the orbital wall, which reflects the SO path by 54° [von Noorden and Campos, 2001]. The inferior oblique (IO) muscle originates from the orbital wall anteroinferior to the globe center and inserts on the sclera posterior to the globe equator. The primary actions of SO and IO cause rotation of the globe around the visual axis and vertical movement. SO and IO muscles also abduct the eye as secondary actions. More details of the orbit anatomy can be found in [von Noorden and Campos, 2001].

Different from skeletal muscles, EOMs are bilaminar and consist of two layers with different fiber types [Porter et al., 1995]. This complicated and special architecture contributes to the realization of both fixations and rapid eye movements up to $900^\circ/\text{sec}$, and requires cooperative control of the EOMs involving very complex neural circuits.

Based on evidence from MRI and computerized 3D reconstruction, Miller [1989] first proposed that the rectus muscle connective tissues posterior to the globe equator, called “pulleys”, couple rectus EOMs to the orbital wall and constrain the transverse shifts of muscle paths. Pulleys are believed to have significant implications on the kinematics and neural control of the orbital plant [Demer, 2004, 2007; Haslwanter, 2002; Miller, 2007; Quaia and Optican, 1998]. In order to understand their specific functions, biomechanical simulation of the orbit incorporating the pulley connective tissues are needed [Haslwanter, 2002; Miller, 2007].

The Hill-type muscle constitutive model [Hill, 1938] widely applied in describing the force generation mechanism of skeletal muscles has been adopted to describe extraocular muscle mechanics [Clark and Stark, 1974; Collins et al., 1975; Robinson, 1964, 1981]. It consists of an active contractile element (CE), a series elastic element (SE), and a parallel elastic element (PE). See Figure 2 for a representative model. CE, the active muscle force, depends on the muscle activation dynamics (a), the active force-length (FL) relationship f_l , and the active force-velocity (FV) relationship f_v . PE models the passive muscle force as a function of muscle length, characterized by the passive FL curve. The active and passive FL relationships have been measured experimentally [Robinson et al., 1969]. Models of f_v have been proposed based on Hill's FV curve of skeletal muscles, experimental data on rat EOMs, and maximum saccadic velocity of human eyes [Robinson, 1981]. SE represents the passive muscle force from elastic elements in series with CE, contributed by tendons and connective tissues within muscles.

2.2. Strand-based EOM Model

As mentioned in Section 1, previous 3D biomechanical EOM models do not simulate dynamics. In addition, the *Orbit*^{TM} simulator does not consider the local deformation of EOMs, which is nonuniform caused by the varying cross sectional areas along the muscle path and fast dynamic motions. The finite element orbital model [Schutte et al., 2006], similar to many other FEM studies, faces numerical difficulties while modeling thin structures like the EOM tendons. The range of deformation that can be simulated is limited and model parameters are difficult to choose. Furthermore, these are expensive to simulate, which restricts their interactive analysis and applications.

To simulate both static and dynamic eye movements realistically and interactively, we use the strand model [Sueda et al., 2008] to represent EOMs. A *strand* is a musculotendon modeling primitive based on a spline with inertia. Each strand represents a part of the musculotendon aligned with the fibers; depending on the level of detail needed, a strand can be as fine as a single fascicle or as coarse as an entire muscle. We model EOMs, consisting of parallel fibers, as a collection of strands. EOM orbital layer (OL) and global layer (GL) are explicitly modeled. An EOM strand incorporates the curved muscle path, which provides geometric realism and handles contact. Compared to other biomechanical models that treat each EOM as a single unit, the strand-based EOM model computes the force and motion continuously distributed along the EOM path. In other words, it models EOM mechanical properties locally and possesses a higher level of complexity. Our EOM model is efficient to compute, benefiting from the parametric representation.

In the following, we first review the strand simulator in Section 2.2.1 and then describe the strand-based EOM model in Section 2.2.2.

2.2.1. Strand Musculoskeletal Simulator—The simulator consists of two dynamic primitives: rigid bodies for the globe and orbit, and strands for the EOMs.

The path of a strand is described by a cubic B-spline curve, whose control points are the generalized coordinates, (position, $q_i(t)$ and velocity, $\dot{q}_i(t)$), which define the strand dynamics. Given the generalized coordinates, a point on a strand at a parameter s is

computed as $p(s, t) = \sum_{i=0}^3 b_i(s) q_i(t)$, where $b_i(s)$ are the B-spline blending functions. With these generalized coordinates of the strand, we derive the kinetic and potential energies, and then the Euler-Lagrange equations of motions. Although a strand can have an arbitrary number of control points, a point on a strand only depends on four control points, due to the local support of the cubic B-spline basis, making the summation a function of only four terms ($i = 0 \dots 3$). Because of this, the mass matrix of a strand [Qin and Terzopoulos, 1996] also depends on only four generalized coordinates, making the system mass matrix sparse. The B-spline based strand gives C^2 continuity on the geometry and the dynamics.

The infinitesimal strain can be computed at any point along the strand.

$\varepsilon(s, t) = \left(\sum_{i=0}^3 b'_i(s) \dot{q}_i(t) \right) / L(s) - 1$, where $b'_i(s)$ are the spline derivatives of the blending functions, and $L(s)$ is the initial strain. Based on these quantities, the passive (f_p) and active (f_a) forces acting on the strand control points can be computed, to get the equations of motion of a single strand:

$$M\dot{q}^{(k+1)} = M\dot{q}^{(k)} + h(f_p + a f_a), \quad (1)$$

where M is the generalized mass matrix of the strand, h is the step size, and a is the activation level.

The equations of motion for the rigid body (globe) are based on the standard body-local formulation with Coriolis term [Murray et al., 1994]. The sliding surface, on which the EOM strands slide, is modeled as a bicubic B-spline patch that is kinematically attached to the globe.

Constraints are required for modeling EOM origins/insertions and for dealing with EOM contact with the globe. Velocity-level constraints on the relative velocities of the globe and the EOM strands are used. Origins/insertions are modeled as a constraint that allows no relative velocity between a point on a strand and a point on the globe. For sliding constraints, the relative velocity of a point on a strand and a point on the sliding surface on the globe is constrained to have no normal component. This way, only tangential relative velocities are permitted, allowing the strand to slide freely on the surface patch. Since strands are based on parametric curves, it is computationally inexpensive to keep track of the contact points. At each time step, the closest points on another strand or a rigid body B-spline surface are updated using Newton-Raphson search. The combined velocity vector of rigid body and strands is denoted by Φ . The equality constraints with a stabilization term is formulated as $G\Phi = \mu g$. G is the constraint Jacobian for the fixed and sliding constraints outlined above; g is the vector of position constraint functions; and μ is the stabilizer weight [Baumgarte, 1972].

Overall, a linear Karush-Kuhn-Tucker (KKT) system [Boyd and Vandenberghe, 2004], is solved at each time step to obtain the generalized velocities of the rigid body and the strands at the next step.

$$\begin{pmatrix} M & G^T \\ G & 0 \end{pmatrix} \begin{pmatrix} \Phi^{(k+1)} \\ \lambda \end{pmatrix} = \begin{pmatrix} M\Phi^{(k)} + hf \\ -\mu g \end{pmatrix}, \quad (2)$$

where M is the generalized mass matrix of the globe and the EOM strands, and λ is the vector of Lagrange multipliers for the constraints. A direct method based on Gaussian Elimination is used to solve this matrix. Once the new velocities, $\Phi^{(k+1)}$, are computed, the new positions are computed trivially (using Rodrigues' formula [Murray et al., 1994] for the rigid body.)

2.2.2. EOM Strands—We have two types of strand elements, *contractile elastic strands* and *non-contractile elastic strands*. A contractile elastic strand models muscle fibers; its total force is simplified to be the sum of the active force (F_{CE}) and the passive force (F_{PE}):

$$F_m = F_{CE} + F_{PE} = a \cdot f_l \cdot f_v + F_{PE}. \quad (3)$$

The muscle activation a is assumed to be a unitless variable between 0 and 1, with $a = 1$ meaning the muscle is fully innervated. The active (passive) force-length function f_l (F_{PE}) models the generated (passive) muscle force as a function of muscle length. The force-velocity function f_v relates the generated muscle force to the muscle velocity. Both f_l and f_v have complex nonlinear characteristics, as does F_{PE} . The SE element of the Hill muscle model illustrated in Figure 2 is not included in our EOM constitutive model. As Robinson [1981] pointed out, the force mechanism of the EOM series element has not been adequately defined due to the lack of physiological data. Therefore, it is difficult to derive a reasonable model for the SE element. None of the existing 3D biomechanical models of the orbit incorporates SE because of its indeterminacy, and we follow this common choice.

We adopt the the active force-length curve of a fully innervated EOM and the passive FL curve used in the Orbit™ 1.8 simulator, which best approximate the published empirical data. Cubic B-spline curves are fitted to the original FL data to conveniently evaluate F_m at any given muscle length. Figure 3(a) shows the force-length relationships of a LR muscle. In our simulation, other EOMs' strengths are scaled by their cross sectional areas. We use the force-velocity model in [Robinson, 1981] for shortened EOMs, and function $f_v = 2 - (0.18 + 0.5V)/(V + 0.18)$ for lengthened EOMs, where V is the EOM lengthening rate. Figure 3(b) shows the FV curve used in simulating dynamics.

Non-contractile elastic strands are used to model EOM tendons and the intermuscular coupling between IR and IO. These strands can be passively stretched because of the attachments; they do not actively shorten to generate force. EOM tendons are very stiff and thus we use a high stiffness to model them. The IR-IO strand stiffness is chosen such that the anteroposterior shift of IO is half the movement that the IR insertion travels, which has been observed from MRI studies [Demer et al., 2003].

Each rectus muscle has two contractile elastic strands in parallel modeling the orbital and global layers of the muscle segment, and a tendon strand in series. The SO muscle has a muscle strand and a long tendon strand (30mm). The IO muscle has one muscle strand, assuming its short tendon is negligible. Subject-specific EOM anatomy, including the EOM path and strength determined by cross section, is incorporated based on 3D geometric models of the EOMs reconstructed from MRI [Wei et al., 2009]. Figure 4(a) is a representative LR muscle mesh. The medial axis depicting the average longitudinal muscle path is represented by a B-spline curve in blue; the control points are shown as green

crosses. We partition the medial spline into two connected curves and fit two strands to model the muscle (in blue) and tendon (in gray) segments respectively (see Figure 4(b)). The cross sectional area A is a variable along the strand, which allows simulation of realistic EOM local deformation. We use the radius $r = \sqrt{A/\pi}$ associated with each control point to approximate the area A . Figure 4(c) renders the strand as a cylinder to demonstrate the nonuniform cross sections.

The globe is modeled as a spherical rigid body with a ball-and-socket joint allowing eye rotation in 3D. EOM origins and insertions are implemented as attachment constraints (cyan spheres in Figure 4(d)), which couple their positions to the attachment sites. Contact between the extraocular tendon and the globe surface is detected on the fly to prevent penetration. Surface sliding constraints (red violet spheres) are pre-specified on the EOM tendon strands such that tendons always slide on the globe. 3-5 evenly spaced sliding points are sufficient because of the parametric representation of strands.

Rectus pulleys are approximated by their discrete functional locations in 3D that have been estimated from MRI [Clark et al., 1997; Kono et al., 2002]. Our goal is to develop a model that captures the primary characteristics of pulleys while introducing a minimal number of undetermined parameters. We use prismatic joints to abstract the mechanical functions of the rectus pulleys. These joints allow the pulleys to slide along the one-dimensional joint axis with zero movement in the transverse direction (see the orange double ended arrows in Figure 4(d)). In other words, by using prismatic joints, we apply zero longitudinal stiffness and infinitely large transverse stiffness on the pulleys to stabilize the rectus muscle paths. Small ($< 1\text{mm}$) transverse sideslips observed from normal subjects under physiological conditions [Kono et al., 2002] are neglected. Because of its simplicity, the strand-based orbital plant model with pulleys is particularly suitable for studying the dynamics and neural control of ocular motility.

Our simulator is sufficiently general to support other implementations of pulleys, for instance using elastic suspensions to incorporate transverse shifts (see simulation of coordinated pulleys in [Wei, 2010]).

2.3. Innervation Solver

As pointed out in [Robinson, 1975], simulating orbital mechanics involves two parts. *Forward simulation* computes EOM forces and eye movement, given the EOM innervations (activation levels). *Inverse control simulation* solves innervations of the six EOMs to hold the eye in fixation or to move the eye to target positions. Previous 3D biomechanical models of the orbit deal with static eye movement only. In steady states, the net force of the six EOMs balances the passive force of the orbital connective tissues. Utilizing this force equilibrium relationship, innervations required to stabilize the eye at any position can be found [Miller et al., 1995; Robinson, 1975]. To address the redundancy problem (three degrees of freedom of the eye rotation vs. six EOMs), most existing models apply Sherrington's Law of Reciprocal Innervation by using a symmetric hyperbola function to explicitly define the innervation relationship of an agonist and antagonist EOM pair. The deficiency of this symmetry assumption has been noted recently [Quaia et al., 2008].

To simulate dynamic eye movement, we cannot use equilibrium equations but need a more complicated solver taking into account eye velocities. We extend the activation solver in [Sueda et al., 2008] to compute EOM innervations. At any time step, given the desired velocity of the globe v_x , the activation levels $a \in [0, 1]$ of the six EOMs are solved by optimizing a constrained quadratic objective function:

$$\begin{aligned} \min_a \quad & w_x \| (H_x a + v_f) - v_x \|^2 + w_a \| a \|^2 \\ \text{s.t.} \quad & 1 \geq a_i > a_i^0 c_v(v_{eye}) c_p(p_{eye}). \end{aligned} \quad (4)$$

The first term minimizes the difference between the simulated globe velocity and the target motion. The matrix H_x can be thought of as the effective inverse inertia experienced by the muscle activation levels in order to produce the target motion. $H_x a$ determines the velocity contributed by the active EOM forces, and is derived by re-ordering the KKT system (Equation 2) to solve for the activation levels, a . The v_f vector is the globe velocity due to the non-active forces. The second term in the objective function minimizes the total activation from all EOMs such that among the numerous possible solutions, the set of innervations with the minimal summed activation energy are preferred. It also serves as a regularization of the quadratic problem. w_x and w_a are scalar weights blending the two terms.

In order to incorporate the characteristics of ocular motor neuron activities [Collins et al., 1975; Sylvestre and Cullen, 1999], we add the constraint terms in the optimization in Equation 4. Each inequality specifies the lower innervation bound of an extraocular muscle EOM_i as a function of the minimal activation level a_i^0 , the velocity factor c_v , and the position factor c_p . We define

$$c_v(v_{eye}) = \max \left(0, \frac{v_{max} - |v_{eye}|}{v_{max}} \right), \quad (5)$$

$$c_p(p_{eye}) = \max \left(0, \frac{p_{max} - |p_{eye}|}{p_{max}} \right). \quad (6)$$

p_{eye} and v_{eye} are the position and velocity of the globe in EOM_i 's action dimension: horizontal for LR and MR, and vertical for the other EOMs. v_{max} is the velocity defining the onset and offset of a saccade, which is set to $20^\circ/s$ [Sylvestre and Cullen, 1999]. The position factor c_p linearly decreases the lower bound with eccentric eye position. p_{max} is the maximum OFF direction (contralaterally directed) eye position, at which an antagonist EOM's innervation is completely off; we assume $p_{max} = 30^\circ$ in our experiments.

Our constraints model the following properties of ocular motoneuron activities that have been observed experimentally. All EOMs are innervated at primary position [Collins et al., 1975]. In fixations, ocular motor neuronal discharges decrease with contralateral eye position [Collins et al., 1975]. Many of the antagonist motoneurons cease firing during contralaterally directed saccades [Sylvestre and Cullen, 1999].

Figure 5 shows computed innervations of LR and MR as a function of horizontal and vertical eye positions. The EOM innervations are interpolated and plotted as iso-innervation curves. The LR and MR activation levels depend on the horizontal eye positions, and are nearly independent of the vertical eye positions, as is consistent with the physiology of the horizontal EOMs [von Noorden and Campos, 2001]. Note that our innervation solver solves for the innervations of all six EOMs simultaneously without enforcing the reciprocal relationship. However, the reciprocal innervation characteristics is obtained automatically

which can be seen from Figure 5, due to the fact that we have a realistic orbital plant with nonlinear geometry and mechanics.

3. Results

Our simulator is programmed in Java. Experiments were performed on a PC with an Intel Core2 Duo 2.4GHz CPU and 3GB of RAM. Interactive simulation of the extraocular muscle dynamics and the orbital plant is achieved at about 16 frames per second.

Figure 6 shows simulated horizontal eye movements. The influence of the pulleys on the rectus muscle paths is clearly observed. As the eye rotates horizontally, the LR and MR pulleys move along their respective joint axis anteroposteriorly while the SR and IR pulleys remain stable in the mediolateral direction. The SR and LR pulleys cause sharp muscle path inflections – the SR and IR muscle bellies posterior to the pulleys are fixed relative to the orbit and their anterior paths follow rotation of the globe in eccentric gazes.

3.1. EOM Deformation During Smooth Pursuit

Extraocular muscle deformation as a function of gaze and time is an important parameter in studying extraocular mechanics [Miller et al., 2006]. We validate the dynamic EOM deformation of our model by comparing simulation to the *in vivo* EOM motion data. Motion-encoded MRI was recently proposed to assess EOM motion during smooth pursuit eye movement [Piccirelli et al., 2007]. Sparse tissue points on the transverse imaging planes of tagged MRI were tracked as the subject was instructed to visually pursue a slowly moving stimulus. EOM deformation was inferred by analyzing the recorded positions of the tissue points. Stretch ratio, defined as the ratio between the deformed muscle length and its initial length, was used to quantify the deformation. Along the longitudinal axis in the primary eye position, the EOM path was divided into three segments of equal length. Stretch ratios of the segments in the two horizontal rectus muscles were analyzed.

We simulate the same smooth pursuit movement as in [Piccirelli et al., 2007]. The eye moves horizontally from 20 degrees in abduction to 20 degrees in adduction with zero vertical movement. The velocity is a sinusoidal function with a 2 second period. The maximum speed is 6.4°/sec. Applying the same analysis scheme, we present the simulated EOM strains in solid curves in Figure 7. For comparison, simulated EOM strains are overlaid on top of the *in vivo* EOM motion data reported in [Piccirelli et al., 2007]. Simulation of this smooth pursuit movement is demonstrated in the supplementary video (MoviePursuit).

Our simulated deformation trends of the LR and MR muscles follow the published MRI data. All EOM segments have sinusoidal deformation pattern. Nonuniform shortening and elongation are observed in the two horizontal EOMs. The middle segment, which has larger cross sectional areas, deforms more than the posterior segment. The anterior part contains more tendinous tissue and deforms the least. Simulated strains of all segments except for the middle LR fall in the 95% confidence interval. The mean percentage difference (MPD) between the simulated strain S_s and the average experimental strain S_e is computed as the

percentage difference $\%Diff = \frac{|S_s - S_e|}{S_e} \times 100$ averaged over all the time frames in which tagged MR images were acquired. MPDs of LR and MR are summarized in Table 2. The main disparity occurs at the middle segments – simulated LR and MR show larger deformation than the empirical data by less than 10%. It is known that due to low contrast, accurate identification of EOM insertions from MR images is difficult. The tracking curves manually placed on the images [see Piccirelli et al., 2007, Fig. 2] show more posterior

insertional ends than the actual insertions. We conjecture that this variation in delineating muscle paths may partially contribute to the discrepancy.

3.2. Simulation of Saccades

In order to test the plausibility of our biomechanical model, we simulated saccadic movement using experimental EOM neural activity data as the neural input of the LR muscle to drive the orbital plant. We then compared the simulated motion to the desired motion to evaluate the system behavior.

The neural drive of a saccadic eye movement can be characterized by a *pulse* component to overcome the viscoelasticity of the orbital plant, a *step* component to stabilize the eye in the new position, and a *slide* component that models the gradual transition between the pulse and step [Robinson, 1964]. The neural control of saccades has been studied extensively [Fuchs and Luschei, 1970; Horn and Cullen, 2009; Robinson, 1964; Sylvestre and Cullen, 1999]. The activities of neurons are characterized by the discharge rates recorded at the neuronal sites. The movement of the LR muscle is controlled by abducens neurons (ABNs) whose discharges during saccades can be expressed as a first order equation [Sylvestre and Cullen, 1999]:

$$FR = b + kE + r\dot{E}. \quad (7)$$

The model approximates the ABN firing rates as a linear function of the eye position E and velocity $\dot{E}$. b is a bias constant, which is the neural firing rate at the stationary central eye position. k and r are constants and their optimal values have been estimated by fitting the above model to the actual neuron discharge recordings from monkeys [Sylvestre and Cullen, 1999].

Figure 8 shows the analysis of a 20 degree abduction saccade while the eye is elevated by 20 degrees. This movement is generated by contraction of the LR muscle, relaxation of the MR muscle, and co-contraction of other EOMs. Here we experiment with a saccade from a secondary position to a tertiary position, which is more interesting and challenging than a purely horizontal saccade. The torsional component in the tertiary gaze is a standard test to evaluate the biomechanics of eye movement.

Figure 8(a) is a snapshot of the simulated gaze trajectory and the orbit configuration when saccade is completed. Muscle activation levels are shown in red on the EOM strands. Obviously, the LR and SR muscles are the two active EOMs. Figure 8(b) shows the approximated ABN firing rate profile for a 20 degree saccade based on the model in Equation 7. The model coefficient values are $b = 156$, $k = 4.2$, and $r = 0.42$. Figure 8(c) plots our estimated innervations of the LR and MR muscles – the primary rectus muscles contributing to horizontal saccades. The other EOMs maintain the vertical elevation of the eye throughout this movement. Note that the computed LR innervation has the pulse-slide-step characteristics of saccadic neural control. Also note that the computed MR innervation realistically models the behavior of antagonist motoneurons during OFF direction saccades – most of them completely cease firing after saccade onsets [Sylvestre and Cullen, 1999]. The model-based firing rate profile in Figure 8(b) is linearly scaled such that the starting and ending values match the computed innervation. The scaled profile is plotted in Figure 8(c), overlaying on the estimated innervation for comparison. We observe that the computed LR innervation dynamics (in blue) agrees with the experimental ABNs discharge dynamics (in red).

We use the scaled empirical neural discharge profile as the neural drive of LR and re-simulate the 20 degree abduction saccade. The blue curves in Figure 8(d) are the desired position and velocity profiles, based on saccade traces recorded from monkeys [Sylvestre and Cullen, 1999]. The superimposed red curves are the simulation results. The simulated saccade reasonably follows the desired saccadic trajectory. The root mean square errors (RMSEs) are analyzed quantitatively in Table 3. The associated torsion is very small, which further demonstrates the simulation accuracy and realism of the nonlinear EOM model in reproducing saccades. We also simulate abduction saccades with the amplitudes of 10 and 30 degrees. Simulation results are shown in Figure 9 and Figure 10. Agreement on the eye position and velocity trajectories is also achieved.

3.3. Simulation of Superior Oblique Palsy

We simulated the pathological case in which the superior oblique (SO) muscle action is weakened due to paralysis of the trochlear nerve. Because the contractibility is diminished, a paretic EOM's generated force is inadequate for moving the eye by the desired amount. The change of the oculomotor plant mechanics caused by paretic SO muscle leads to ocular misalignment, or strabismus. Biomechanical simulation is useful for systematically understanding the mechanical influences of the paretic EOMs.

To simulate superior oblique palsy (SOP), we first computed the muscle innervations that stabilize a normal eye in various eye positions. To mimic an eye with paretic SO muscle, we then set the SO innervation to zero while keeping the values of the other EOMs unchanged. The new fixation locations are the predicted eye positions of a pathological eye with SOP.

We compare simulation results with published data from a monkey with acute SOP [Shan et al., 2007]. We choose this data for comparison because the controlled experiment ruled out other abnormalities possibly affecting alignment. Our goal is to evaluate whether our model is able to predict deviation patterns that are qualitatively consistent with observed pathology.

The predicted eye positions of a SOP eye are plotted in Figure 11. Monkey alignment data reproduced from [Quaia et al., 2008] as well as simulated data are superimposed for comparison. Incomitant deviations dependent on eye positions are observed. Our simulation shows a vertical deviation for every fixation; the deviation is greatest with the eye adducted and down. These results are consistent with the role of SO in depressing the globe. Extorsional deviation, presented as the angle between the vertical bar of the cross and the vertical axis, is also observed. We conclude that simulated SOP alignment shows static deviation patterns consistent with experimental acute SOP data.

The mean absolute difference between the simulation and the data shown in Figure 11 is 0.98° in the horizontal direction and 2.50° in the vertical direction. Again we would like to emphasize that our main objective is to validate the model qualitatively rather than quantitatively based on these numbers, because the simulated model is a human orbit and not a monkey orbit, and the degree of deviation due to SOP varies significantly due to anatomical variations [Quaia et al., 2008].

4. Conclusions

This paper presents a biomechanical model that simulates 3D motion of the extraocular muscles interactively. Our objective is to lay the foundation of a biomechanical and computational framework that possesses fewer limitations than previous models and is sufficiently general for both scientific research and clinical applications.

Our model is the first 3D biomechanical model that simulates dynamic eye movements. The strand-based EOM model is associated with nonlinear anatomical and constitutive properties. Realistic EOM paths and cross sectional areas of these EOM strands are based on geometric models reconstructed from MR images of human orbits. The model implements pulleys with infinite transverse stiffness; it generates reasonable EOM paths, gaze positions and trajectories given EOM innervations.

Before using the biomechanical model to answer scientific questions, we need to assess the plausibility and accuracy of the simulator. We performed several validations from different aspects, which not only evaluate the accuracy of the EOM model but also provide guidance for choosing model parameters. EOM deformation during smooth pursuit was examined and compared to experimental motion-encoded MRI data from human subjects and consistent deformation patterns were observed. We simulated saccadic eye movements that abduct the eye at different amplitudes. Abducens motoneuron discharges based on empirical data were used to drive the lateral rectus muscle, the primary EOM that is active in these eye movements. We found that the simulated saccadic trajectories reasonably follow the desired trajectories, which shows the realism of the EOM dynamics. We also simulated strabismus caused by superior oblique muscle palsy. The predicted deviations in various directions were qualitatively consistent with experimental data from controlled subjects. Based on the results, we conclude that our EOM model with nonlinear constitutive characteristics and MRI-based anatomical properties is able to generate realistic static and dynamic eye movements.

In future work, we plan to further investigate the force and motion of the simulated EOMs by comprehensively analyzing various eye movements. Efforts on speeding up simulation to enable real-time applications will be made. We would also like to understand the roles of pulleys in coordinating EOMs and test pulley hypotheses that have been proposed.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

We would like to thank Dr. Joel Miller and Dr. Joseph Demer for very helpful discussions. We also thank Dr. Kathleen Cullen for the monkey saccade data and Dr. Christian Quaia for the monkey SOP data. This research was supported in part by NIH grants R01 NS050942 and R01 AR053608, Canada Research Chairs Program, Peter Wall Institute for Advanced Studies, NSERC, CIHR, Canada Foundation for Innovation, and BC KDF.

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Figure 1.
3D model of a human orbit reconstructed from MRI [Wei et al., 2009]. The six extraocular muscles are: LR-lateral rectus, MR-medial rectus, SR-superior rectus, IR-inferior rectus, SO-superior oblique, IO-inferior oblique.

Figure 2.

Three-element Hill-type muscle constitutive model. CE: contractile element; PE: parallel elastic element; SE: series elastic element.

(a) Force-length relationship

(b) Force-velocity relationship

Figure 3.

(a) Isometric force-length curves of the LR muscle, based on Orbit 1.8^{TM}. (b) Force-velocity relationship.

(a) Fitted spline to 3D mesh

(b) Strands of LR

(c) Radius of LR strand

(d) Mechanical constraints of LR strand

Figure 4.
Strand-based modeling of an EOM.

Figure 5. Inversely computed innervations of LR and MR plotted as a function of horizontal (ABD – abduction, ADD – adduction) and vertical (DOWN – downward, UP – upward) eye positions between -30 degree and 30 degree.

Figure 6. Simulated horizontal eye movements. Rectus muscle pulleys are represented as prismatic joints, shown as green arrows. Note the inflection of the SR and IR muscle paths and the anteroposterior movement of the LR and MR pulleys.

Figure 7.

Comparison of the lateral rectus and medial rectus muscle strains from simulation (in solid curves) to published MRI data. The shaded regions show the average stretch ratios (mean $\pm 1S.D.$) from 7 subjects over time (reproduced from [Piccirelli et al., 2007]). Values larger than one indicate muscle lengthening; values smaller than one indicate muscle shortening.

Figure 8. Simulated 20 degree abduction saccade in 20 degree elevation. (See supplementary demonstration file MovieSaccade.

Figure 9.
Simulated 10 degree abduction saccade in 20 degree elevation.

Figure 10.
Simulated 30 degree abduction saccade in 20 degree elevation.

Figure 11.

Simulated eye positions of a SOP eye shown as black crosses. Grey crosses are monkey data, reproduced from [Quaia et al., 2008]. Cyclorotation, multiplied by 1.5 for better visualization, is shown as the vertical tilt of the crosses. Courtesy of Dr. Christian Quaia for the monkey data.

Table 1

Acronym table.

Acronym	Term
EOM	extraocular muscle
LR	lateral rectus
MR	medial rectus
SR	superior rectus
IR	inferior rectus
SO	superior oblique
IO	inferior oblique
CE	contractile element
SE	series elastic
PE	passive elastic
FL	force-length
FV	force-velocity
ABN	abducens nucleus
SOP	superior oblique palsy

Table 2

Computed mean percentage difference (MPD) between the simulated strain and the average experimental strain of the LR and MR muscle segments.

EOM	Anterior	Middle	Posterior
LR	0.84%	9.50%	1.46%
MR	1.71%	8.48%	4.29%

Table 3

Root mean squared errors (RMSEs) of the simulated saccadic positions and velocities, and maximum torsional errors.

Saccade Amplitude (°)	Position RMSE (°)	Velocity RMSE (°/sec)	Max Velocity Difference (°/sec)	Max Torsional Error (°)
10	1.27	34.31	6.31	0.16
20	1.27	32.93	24.00	0.10
30	1.78	41.27	12.22	0.09