HUMAN OCULOMOTOR CONTROL SIMULATION

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ABSTRACT

Techniques have been developed for monitoring human oculomotor innervation and unrestrained muscle forces during large eye movements. Results of these measurements have led to a better understanding of eye movement control strategies and an Interactive Simulation Language (ISL) model of the peripheral oculomotor plant which duplicates both healthy and pathological eye movement performance.

This model matches physiological, nonlinear innervational and viscous properties of the oculomotor plant for large excursions, performing satisfactorily over the entire + or -50° range of eye movement. The model duplicates the family of muscle length-tension curves, traces the static locus and properly generates agonist and antagonist muscle forces observed in freely moving eyes during tracking and saccadic movements. It duplicates dynamic saccadic length-tension loops (the most critical test of model performance). It shows the effect of muscle shortening on forces during a saccade, and the initial force increase in a relaxing antagonist.

The model simulates both physiological and pathological human eye movement performance. For example, the tropias, overshoot, velocity, and eye movement magnitudes are each duplicated for lateral rectus palsy patients. In addition, the effects of internuclear ophthalmoplegia are likewise duplicated. This striking identity of model performance with known pathology has important bearing on the possible clinical uses of models which can be evaluated from patient measurements.

An individual patient clinical model may eventually be able to qualitatively and quantitatively aid the ophthalmologist in his diagnosis and choice of surgical treatment plan. Operation on the model would permit the surgeon to compare various amounts and types of strabismus surgery and choose an optimum approach from many model responses before going to the operating room.

INTRODUCTION

When a person is unable to direct both eyes to the same object his visual disorder is called strabismus. The clinical correction of strabismus generally involves surgically moving the point of attachment of one or both of the extraocular muscles to realign the eyes. However, some 20 to 40 percent of all strabismus surgical patients must return for reoperation(1). Thus, there is clearly a need for a better understanding of the mechanical defects of strabismus and the surgery applied to overcome them.

METHODS

Computer simulation has become necessary for achieving a more complete and quantitative understanding of the many complex interrelationships involved in oculomotor control. Each anatomical element of the oculomotor plant must be separately represented since pathology and surgery affect each separately. Pathological ocular deviations are often significantly large, persisting to a great, but variable degree, over the entire range of ocular motility. Thus, a clinically useful model must be able to correctly simulate large eye movements embracing the performance of the grossly nonlinear oculomotor system over the complete range of plus or minus 50 degrees. The major nonlinearity is found in the relationship between the magnitude of innervation (the input control signal) and the position of the eye(2). In addition, muscle viscosity varies as muscle tension and is a nonlinear function of eye movement velocity.

The techniques for measurement of these anatomical and physiological entities have been described elsewhere(3,4) and hence are only briefly outlined here. First, the muscle innervation, or input control signal, \( g \), has been measured utilizing a specially developed multiple microelectrode technique for statistically sampling the simultaneous activity of up to 50 widely spaced, single motor units in a human ocularoratory muscle during natural eye movements(3). This is the equivalent of sampling the message on the oculomotor nerve as sent from the brain to the eye muscle. By this means we have found that the brain strategy utilizes a nonlinear (square-law) pattern of input innervation to control the position of eye fixation. Next, to measure in situ muscle tension forces, \( T \), under the natural conditions existing during unrestrained eye movements it was necessary to develop a miniature, high-performance, implantable recording strain gauge which has been separately described along with the observed patterns of human eye muscle forces(5). The elasticity, \( k \), and viscosity, \( B \), of the oculorotatory muscles and passive globe (eyeball) restraining tissues have also been measured(5). Eye position, \( \theta \), is measured conventionally, either photoelectrically(6) or by means of electro-oculography(7).

A model based on these new human physiological findings has been evolved over the past few years and an analog computer simulation(2) has now been adapted to Interactive Simulation Language (ISL) for more powerful, extensive and flexible basic and clinical applications. This model of the oculomotor plant and the differential equations describing eye movements are shown in Fig. 1. The model reflects our physiological findings that each eye muscle pair functions anatomically in a reciprocally innervated, class A, "push-pull" mode which compensates for (i.e., linearizes) the square law input innervation. This results in a linear muscle force difference acting on the
predominantly linear globe restraining elasticity. The end result is that the eye movement system appears to be linear when observed from the outside. However, you are aware of the more than linear effort required to hold your eyes at the extreme gaze position of 50° left or right.

![Diagram of the mechanical model of the human oculomotor plant and its differential equations of motion.](image)

**Fig. 1** - Diagram of the mechanical model of the human oculomotor plant and its differential equations of motion.

The physiologically measured input controls for this oculomotor plant model are represented in Fig. 2 as the static levels of innervation to the agonist and antagonist eye muscles, $\phi_M$ and $\phi_L$. This transfer function relating the voluntary or desired eye position, $\Psi$, to the level of static innervation, $\phi$, is the result of the strategy chosen by the brain to control the steady-state or fixation position of the eyes.

![Steady-state input control signals (agonist and antagonist eye muscle innervation) as a function of eye position.](image)

**Fig. 2** - Steady-state input control signals (agonist and antagonist eye muscle innervation) as a function of eye position.

Superimposed upon this steady-state innervation are the transient control signals responsible for dynamic eye movements. The fastest eye movements are called saccades. These are quick, stepwise, coordinated movements of the eyes which are controlled by intricate, pre-programmed calculations of the central nervous system and result in remarkably accurate and rapid, precisely controlled flicking of the direction of gaze from one point to another. Figure 3 shows the magnitude and shape of the transient oculomotor control signals which produce saccades. These data have been hand averaged to remove the usual biological noise. Note a characteristic 10 msec. rise time to peak activity which is maintained for approximately one-half of the saccadic duration and falls with a time constant having a value of approximately one-fifth of the saccadic duration. The exact nature of the instantaneous value of this eye movement control signal is important in determining the transient time course of eye movements during saccades where large, inconstant and nonlinear viscous forces predominantly dictate movement.

![The time-varying pattern of the dynamic control signals of the eye muscles. These signals are superimposed on the steady-state levels to produce fast eye movements called saccades.](image)

**Fig. 3** - The time-varying pattern of the dynamic control signals of the eye muscles. These signals are superimposed on the steady-state levels to produce fast eye movements called saccades.

These physiologically measured, complex human eye movement control signals are used as inputs to drive the ISL model of the human oculomotor plant shown in the block diagram, Fig. 4. The left hand portion of the diagram performs the function of the central nervous system in generating these complex input control signals. In the upper left, $\Psi$ represents the initial desired voluntary eye position (for example, straight ahead, the so-called primary position). $\Psi$ then represents the desired new position of the eyes in degrees. The summer and multiplier in the upper and lower left portions of the diagram generate the steady-state innervation functions of Fig. 2. The left central elements of this model generate the transient components of the saccadic eye movement control signal which are added to the static components resulting in the complete eye movement control signals $\phi_M$ and $\phi_L$. 

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The upper and lower right elements of the model represent the medial and lateral rectus respectively. The contractile element consists of two dividers, a multiplier and an integrator to represent respectively the nonlinear muscle viscosity as a function of developed tension, eye movement velocity and the temporal dynamics of the contractile element. The series elastic element, \( K_s \) of each muscle is on the right of the collection of muscle elements. The muscle output consists of tendon tensions, \( T_M \) and \( T_L \).

These muscle tensions are algebraically added and applied to the globe dynamics, \( K_g \), and \( B_0 \), in the right central portion of the model. The resulting output is seen as eye position, \( \theta \), and eye movement velocity, \( \dot{\theta} \).

Figure 5 is an ISL listing of the oculomotor plant model described here.

RESULTS

Figure 6 shows the model output performance characteristics on the length-tension plane as reproduced directly from a storage oscilloscope. Figure 7 is a hand-traced rendition of these curves labeled for greater clarity. These data agree quite well with recorded physiological measurements on human oculorotary muscle patients. In fact, Fig. 7 can serve as a summary of the measured physiological output of the normal human oculomotor plant(2)showing both the steady-state and dynamic performance of this system on the length-tension plane. Muscle length increases to the right and is shown in terms of eye position.
The heavy curved line intersecting the small circles at the bottom of the diagram indicates the measured tensions utilized by the oculomotor system to hold the eye at any degree of eccentric gaze. We call this curve the static locus of fixation tensions. Tensions recorded during smooth following movements of the eyes fall on or slightly above this static locus.

Typical dynamic length-tension loops for 15° fast saccadic movements of the eyes produced by the agonist (active) muscle are shown in Figs. 6 and 7 as counterclockwise-traced force pulses extending between the open circles on the static locus. These circles represent the starting (right) and finishing (left) points of saccadic eye position. A typical dynamic length-tension loop for the antagonist (relaxing) muscle would be shown as a much smaller, flattened clockwise path moving to the right between the initial (left) and final (right) positions of the eye. The tops of the saccadic force peaks in Fig. 7 define an upper curved line which represents the maximum forces recorded during any eye movement. The area between this upper curve and the static locus we call the operational envelope. This operational envelope defines the normal ranges of tensions for horizontal eye movements as measured during the waking state. The operational envelope of normal eye movement forces measured at the tendon of the muscle is determined by neuromuscular control, the innervational strategy of the central nervous system operating on the mechanical viscoelastic characteristics of the muscles and globe. The combination of these factors restricts normal muscle activity to a mere 20 percent of the area of the length-tension diagram. All areas on the length-tension diagram outside the operational envelope are forbidden regions during normal eye movements. Only during abnormal or pathological conditions do the muscle forces leave the operational envelope area of the length-tension diagram. Thus, the operational envelope has clinical significance relating to diagnosis utilizing forced duction or other techniques.

In the model of Figs. 1 and 4, the contractile element force consists of three components: the developed force, F, due to innervation, φ; the force-velocity relationship of the muscle, or viscosity, B; and the force due to the parallel elastic element, k_2, contributing to the length-tension characteristic of muscle. The sum of these three forces is equal to the force measured at the muscle tendon. However, any one component of this contractile element force cannot be uniquely determined at the tendon because of the isolating series elastic element, k_1.

Figure 8 is a time based record of the performance of the model left eye in response to a zero to 20-degree right saccadic input innervation. φ_M of the medial rectus muscle shown on the top channel of the oscillographic record. The next lower record shows the innervation, φ_L of the antagonist lateral rectus muscle which was reciprocally innervated, in this case turned completely off during the saccade in accord with the quantitative results of physiological measurements of innervation.

The next two records are those of model-derived muscle tensions, T_M and T_L, of the medial and lateral rectus muscles respectively. The agonist medial rectus muscle force is seen to be greater, peaking in about 50 msec and then slowly decaying to its steady-state fixation or holding value. Just below it, the lateral rectus muscle tension is also seen to increase before decreasing to its final steady-state value. This increase was unexpected since the lateral rectus muscle innervation drops to zero during the saccade, and separate isometric measurements of this antagonist muscle force have shown that the muscle force drops below baseline tensions. The reason for the muscle force increase is that this antagonist muscle is stretched by the agonist muscle at a rate which causes the antagonist muscle viscous forces to increase more rapidly and with greater magnitude than the force decrease due to the drop in innervation. Both the agonist and antagonist model muscle forces faithfully reproduce the patterns and magnitude of forces as measured by our "C" gauges implanted in human oculorotary muscles during strabismus surgery (4).

The next lower record represents the resultant eye movement, θ, which again truly represents the pattern of eye movement seen in normal human subjects. Finally, the lowest record shows the eye movement velocity, θ, which again is a sensitive test of model performance. This velocity profile agrees quite well with measured human eye movement data and again illustrates the realistic performance of this oculomotor plant simulation.
Fig. 9A - Oculomotor model simulation of a normal left eye.
9B - Simulation of a pathological eye movement with a paralyzed antagonist left lateral rectus muscle.
9C - Simulated pathological eye movement with a paralyzed agonist left medial rectus muscle.
9D - Actual patient records of a lateral rectus palsy patient. Note how closely these are duplicated by the model.

Figure 9 compares pathological oculomotor performance with the normal performance record of Fig. 8. Figure 9A is an oculomotor simulation of a normal left eye during a 0 to 15-degree right saccade. At the top are the unrestricted medial and lateral muscle tensions, \( T_m \) and \( T_l \), shown on the same scale to illustrate comparative performance. The next lower trace shows eye position, \( \theta \). The bottom trace indicates velocity, \( \dot{\theta} \).

Figure 9B shows the model simulation of a pathological left eye during a 15° right saccade with a paralyzed antagonist left lateral rectus muscle. Note the 10° right esotropia (inward misalignment), 3° saccadic overshoot, 150° per second velocity (about half normal), and 8° final eye movement instead of the normal 15°.

Figure 9C shows the model simulation of a pathological left eye movement during a 15° right saccade with a paralyzed agonist left medial rectus muscle. Note the 10° left exotropia (outward misalignment), 500 msec saccadic duration, 60° per second velocity, and 7° final eye movement.

Figure 9D presents eye movement records of a lateral rectus palsy patient. The top record with a paralyzed antagonist shows the tropia, overshoot, velocity, and size of eye movement which are duplicated by the model in Fig. 9B. The bottom trace is a patient record of movement with a palsied muscle acting as agonist. Note the model duplicates this pathological eye movement in Fig. 9C.

**DISCUSSION**

It can be seen that the model simulates both physiological and pathological human eye movement performance. This striking identity of model performance with known pathology has important bearing on the possible clinical uses of models which can be evaluated from patient measurements. By making simple clinical office measurements with a strain gauge forceps it appears to be possible to evaluate the various model elements of the patient's oculomotor plant in the surgeon's office. Calibrated forceps measurements (8) of isometric forces during contralateral eye fixations can determine neuromuscular functional imbalances; and contractures or mechanical restrictions may be quantitatively determined in the office with position monitored forceps measurements of ocular length-tension characteristics.

An individual patient clinical model thus resulting may be able to quantitatively and qualitatively aid the ophthalmologist in his diagnosis and choice of surgical treatment plan. An interactive graphic CRT display now under development in our laboratories would provide immediate feedback indicating the results of various combinations of simple surgical techniques on oculomotor performance and alignment. For example, the effects of surgery on comitance (relative alignment of the eyes) could be instantly and directly displayed in graphical form. Simulated surgery on the model would permit an ophthalmologist to explore various types and amounts of corrective surgery. He would then have the choice of pursuing a conventional plan or of considering a different surgical approach suggested by newly derived computer data based on quantitative measurements of the patient before going to surgery.

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**REFERENCES**


