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# PARAMETRIC SENSITIVITY ANALYSIS OF A HOMEOMORPHIC MODEL FOR SACCADIC AND VERGENCE EYE MOVEMENTS

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A non-linear sixth order homeomorphic model, fitted with parameters based on eye movements and physiological data, was tuned so that it provided good simulations for the shapes of the magnitude, velocity and acceleration trajectories. Excellent quantitative agreement was obtained in terms of the *Main Sequence* diagrams for human eye movements. Parametric sensitivity analysis was done for a saccade of ten degree amplitude, a physiologically normal magnitude at which experimental data is both abundant and relatively noise free.

Among the many useful results of this sensitivity analysis are that pulse width (PW) and pulse height (PH) were confirmed as the two controlling parameters for the human eye movement model. Output behavior was relatively insensitive to variations of the passive elements of the plant. This analysis also pointed out that more physiological data are needed to understand the role of the non-linear force-velocity relationship of the extraocular muscles.

Saccade Vergence Parametric sensitivity Main sequence Eye movements Reciprocal innervation

## 1. Introduction

Saccades are fast, dart-like, conjugate eye movements used for reading, scanning and tracking. Their function is to position the foveae of the eyes in a time optimal manner. Vergence eye movements, on the other hand, are slower, disjunctive movements that are used when looking between a near and a far point. Thus their function is to achieve binocular fixations and so aid in fusion. To simulate the dynamical features of these eye movements, we have expanded the Clark–Cook–Stark [1,2] homeomorphic sixth order non-linear model. It takes into account the reciprocal nature of the agonist-antagonist muscle pair which exerts forces on the globe and incorporates many other results of physiological experiments. There are twenty parameters in the model, all either constants or monotonic functions of saccadic amplitude. Finally, a sensitive analysis describes how each parameter affects the behavior of the system.

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## 2. The Reciprocal innervation model

### *2.1. Model description*

The human globe has three degrees of freedom and is moved by six muscles. Horizontal eye movements are controlled by the lateral and medial rectus muscles [3]. The reciprocal innervation model, shown in fig. 1 was constructed using ideal lumped parameter mechanical elements. The density of a human eyeball is approximately that of water; assuming a rigid sphere, the effective inertia developed by the globe on the muscles is $4.3\times10^{-5}$ gm tension-sec$^2$/deg (2.19 newtons-sec$^2$/meter). The orbital tissues surrounding the globe are modelled by a viscous element, a dashpot – whose force is velocity dependent – and an elastic element, a spring – whose force is position dependent. Each muscle is described by an active state tension generator, a viscous element, and both series are parallel elastic elements. We grouped the muscle parallel elasticities and the globe tissue elasticity into one spring, because springs in parallel have additive effects.

The properties of muscles have been investigated by many famous researchers like A.V. Hill and B. Katz [4,5]. They have all found that muscle shortening

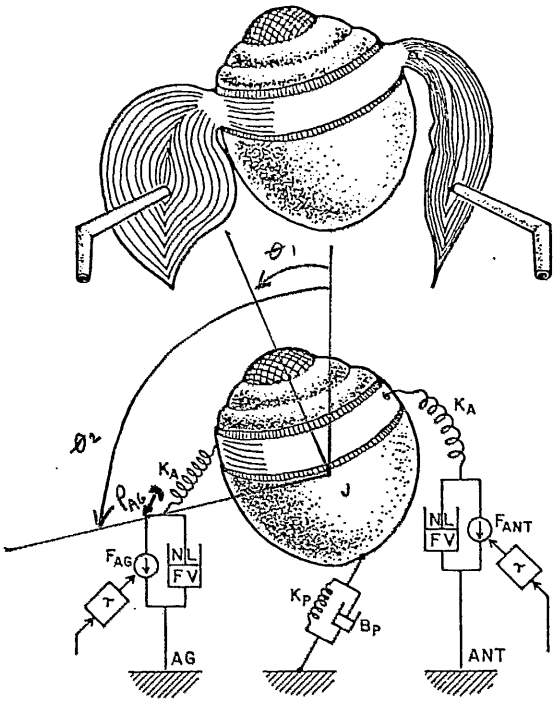



Fig. 1. Reciprocal Innervation Model. The top figure shows Descartes' basic concept of reciprocal action of extraocular muscles: Descartes thought that muscles were like balloons; when inflated they would be short and fat, but when deflated they would be tall and skinny; the pipes were to pump fluid in and out of the muscles. The shortening of the agonist together with the lengthening of antagonist produces eye movements. The bottom figure shows the ideal mechanical elements used for modelling the plant. The globe and surrounding tissues were modelled by the effective inertia ($J$), a viscous element ($B_p$) and a passive elasticity ($K_p$). Each muscle was modelled by an active state tension generator, a non-linear dashpot ($NL-FV$) representing the non-linear force–velocity relationship, a series elasticity ($K_A$) and a parallel elasticity which was combined with the passive elasticity of the globe to form ($K_p$). The active state generator converts oculomotoneuronal firing into force through a first order activation–deactivation process.

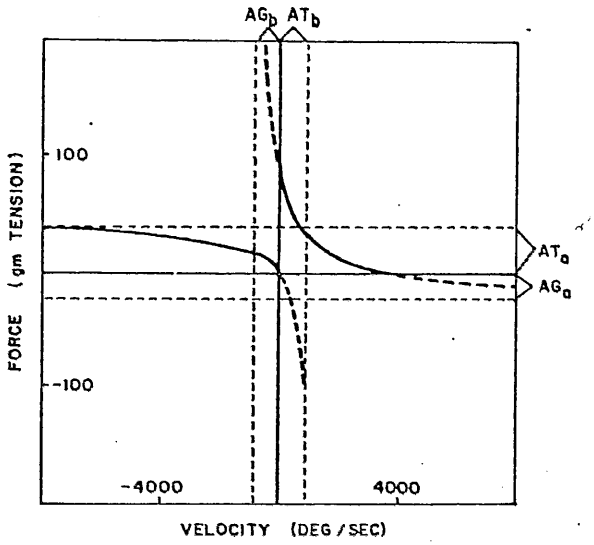



Fig. 2. The force-velocity relationship of active human muscles is hyperbolic. The constants a and b of the Hill equation shift the asymptotes away from the origin. There are, of course, a family of hyperbolic curves for different activation states. The forces of the shortening muscle, $P_{AG}$, and lengthening muscle, $P_{AT}$, are functions of active state tensions $F_{AG}$, $F_{AT}$ respectively. They are given by:

$$P_{AG_1} = F_{AG} - B_{AG}\dot{\theta}_2 \quad \text{where} \quad B_{AG} = \frac{F_{AG} + AG_a}{\dot{\theta}_2 + AG_b},$$

$$P_{AT} = F_{AT} - B_{AT}\dot{\theta}_3 \quad \text{where} \quad B_{AT} = \frac{F_{AT} - AT_a}{\dot{\theta}_3 - AT_b}$$

for muscle lengthening. These curves show the force–velocity relationship for agonist active state tension of 87 gm and antagonist active state tension of 0.79 gm – the values used in the model for a ten degree saccade.

obeys roughly a hyperbolic relationship, the force-velocity curve, demonstrating the non-linearity of the viscous element (fig. 2). The force-velocity relationship of lengthening of the antagonist muscle has a steep slope for low velocities and saturates at some maximum force at high velocities [5, 6, 7, 8, 9, 10]. For our simullations, hyperbolic relationships were used for both agonist and antagonist dashpot coefficients. Series elasticities have also been studied in detail and their effects are best demonstrated by the quick release experiments [11]. We assumed that during saccades, muscles have constant series elasticities.

The high frequency bursts of motoneuronal firing which drive the eyes rapidly to their new positions last about half the duration of the saccades [12, 13]. The tonic changes in motor-neuronal activity hold the eyes in their new positions. These phasic and tonic changes can be thought of as pulse-step envelopes of neural activity. These pulse-steps of neural activity are acted upon by several physiological facets to produce the active state tensions of the muscles. Both innervation frequency and active state tension generated at the

muscles seem to follow first-order processes with the activation-deactivation processes being rate limiting.

### 2.2. Model equations

The system equations for the model are as follows:

$$F_{AG} = K_{AG}(\theta_2 - \theta_1) + B_{AG}\dot{\theta}_2 ,$$

$$F_{AT} = K_{AT}(\theta_1 - \theta_3) - B_{AT}\dot{\theta}_3 ,$$

$$K_{AG}(\theta_2 - \theta_1) - K_{AT}(\theta_1 - \theta_3) = K_P\theta_1 + B_P\dot{\theta}_1 + J_G\ddot{\theta}_1 .$$

Where $F_{AG}$ is the actual force exerted by the agonist; $F_{AT}$ is the actual force exerted by the antagonist; $\theta_1$ is eye position; $\theta_2$ and $\theta_3$ are separated from $\theta_1$ by the series elasticities of agonist muscle and antagonist muscle respectively. $\dot{\theta}$ and $\ddot{\theta}$ indicates velocity and acceleration.

The newtonian equation is:

$$F_{AG} = B_{AG}\dot{\theta}_2 + K_P\theta_1 + B_P\dot{\theta}_1 + J_G\ddot{\theta}_1 + F_{AT} + B_{AT}\dot{\theta}_3 .$$

Note: $\theta$ in the above equations are angular positions.

The six state equations are:

| | Initial conditions |
|---|---|
| $\dot{\theta}_1 = \theta_4$ , | $\theta_1 = 0$ , |
| $\dot{\theta}_2 = \dfrac{AG_b(K_{AG}(\theta_1 - \theta_2) + \theta_5)}{AG_a\theta_5 + K_{AG}(\theta_2 - \theta_1)}$ | $\theta_2 = 16/K_{AG}$ , |
| $\dot{\theta}_3 = \dfrac{AT_b(K_{AT}(\theta_1 - \theta_3) - \theta_6)}{AT_a - K_{AT}(\theta_1 - \theta_3)}$ | $\theta_3 = -16/K_{AT}$ , |
| $\dot{\theta}_4 = \dfrac{K_{AG}\theta_2 + K_{AT}\theta_3 - \theta_1(K_{AG} + K_{AT} + K_P) - B_P\theta_4}{J_G}$ | $\theta_4 = 0°/s$ , |
| $\dot{\theta}_5 = 1/\tau_{AG}(-\theta_5 + N_{AG})$ | $\theta_5 = 16$ gm tension , |
| $\dot{\theta}_6 = 1/\tau_{AT}(-\theta_6 + N_{AT})$ | $\theta_6 = 16$ gm tension . |

Note: $\theta$ in the above six equations are *state variables*.

To simulate saccades and vergence eye movements from 0.1 to 50 degrees, the following parametric equations were used:

$K_{AG}$ = $K_{AT}$ = 1.8 gm tension/deg = 91.9 newtons/meter;
$K_P$ = 0.86 gm tension/deg = 43.9 n/m
$B_P$ = 0.018 gm tension-sec/deg = 0.919 n-s/m
$J_G$ = $4.3\times10^{-5}$ gm tension-sec$^2$/deg = $2.192\times10^{-3}$ n–s$^2$/m
$N_{AG}$ = see table 1
$N_{AT}$ = $(0.5+16e^{-\Delta\theta/2.5})$ gm tension
$N_{AG}$-tonic = $(16+0.8\,\Delta\theta)$ gm tension
$N_{AT}$-tonic = $(16-0.06\,\Delta\theta)$ gm tension
$AG_{act}$ = $(13-0.1\,\Delta\theta)$ ms
$AG_{deact}$ = 2 ms
$AT_{act}$ = 3 ms
$AT_{deact}$ = 11 ms
$PW$ = see table 1 (antagonist pulse always circumscribe agonist pulse by 3 ms on each side)

$$B_{AG} = \frac{F_{AG} + AG_a}{\dot{\theta}_2 + AG_b} \qquad AG_a = 0.25\,F_{AG} \quad AG_b = 900°/s$$

$$B_{AT} = \frac{-F_{AT} + AT_a}{\dot{\theta}_3 + AT_b} \qquad AT_a = 40 \text{ gm tension} \quad AT_b = 900°/s$$

Note:
1 gm tension = $9.806\times10^{-3}$ newtons ,
1 degree = $1.92\times10^{-4}$ meters.

The development of the model and physiological justification of the parameters are the subjects of other papers in preparation.

## 3. Simulation method

The model was simulated on a PDP8 computer using mixed FORTRAN II and SABR assembly code. Displays of the behavior of model and parameters were presented graphically on a Tektronix 611 storage scope and quantitatively on a teletype machine. Copies of

Table 1
Values of PW and PH used for simulation.

| Magnitude degrees | Pulse width (PW) ms | Pulse height (PH) GMS-tension |
|---|---|---|
| 0.1 | 10 | 17.6 |
| 0.5 | 10 | 20 |
| 1 | 11 | 22 |
| 5 | 15 | 53 |
| 10 | 20 | 87 |
| 20 | 31 | 124 |
| 30 | 40 | 155 |
| 40 | 54 | 160 |
| 50 | 70 | 160 |

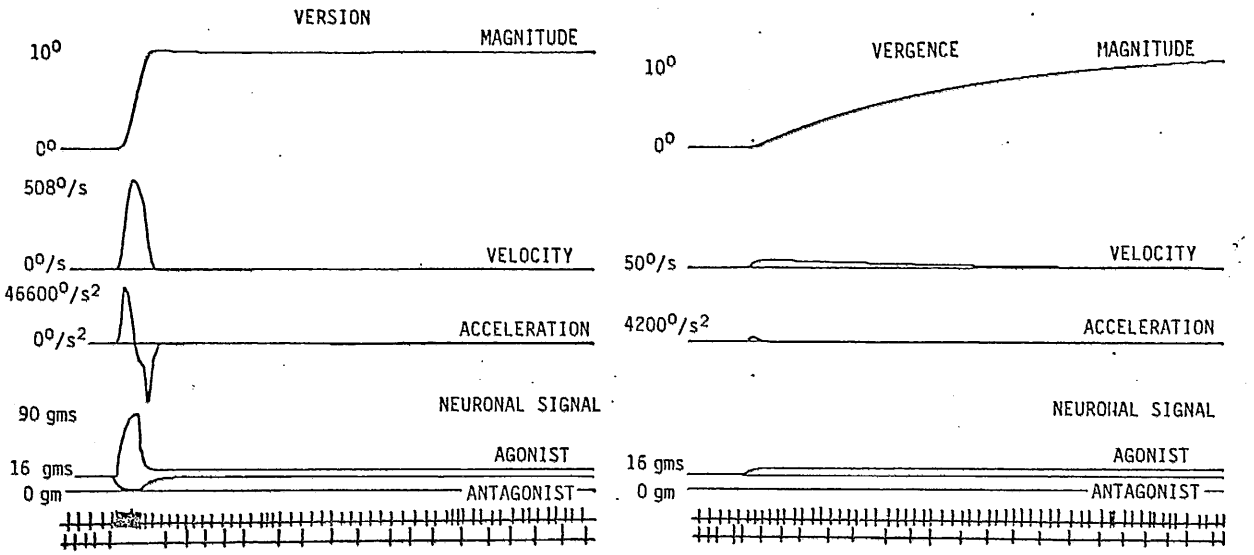



Fig. 3. Simulation of ten degree saccadic and vergence eye movements. Traces show from top to bottom magnitude, velocity and acceleration trajectories, and the active state tension with their corresponding neuronal firing patterns. Note that saccades are driven by a high frequency oculomotoneuronal burst which settles to tonic activity. This activity resembles a pulse step envelope for saccades. Vergence eye movements are driven by an increase in tonic level firing, the envelope of firing resembling a step increase of activity. These responses agree well with physiological data both qualitatively and quantitatively.

simulations were made either by a Tetronix hard copy unit or an X-Y plotter. A fourth order Runge–Kutta integration routine was used in solving the six differential equations. To determine the duration of saccades smaller than thirty degrees, the saccade was said to be finished when the velocity reached 5°/s; for larger movements, 15°/s was used. Since it was a digital simulation, internal parameters were varied easily and model responses were noise free. A simulation bandwidth of 500 Hz was used.

Throughout the process of tuning values of parameters, available physiological data were checked to assure the numbers picked were within physiological range. The behavior of the model output was compared both qualitatively by matching shapes of magnitude, velocity and acceleration trajectories of physiological data and quantitatively by comparing with the Main Sequence diagrams.

## 4. The Main Sequence for human eye movements

Raymond Dodge was perhaps the first to have studied eye movements quantitatively [15]. His data fit on the Main Sequence diagrams of Bahill et al. [16] which summarize the fine structure of eye movement trajectories. When the peak velocity and duration of eye movements were plotted against their magnitudes on a log-log scale (due to three orders of magnitude involved), they were found to cluster around a straight line with saturation at higher velocities. The greatest upper bound of the Peak Velocity Main Sequence shows the maximum peak velocity (PV) for various sized eye movements of normal unfatigued humans while the least lower bound of the Duration Main Sequence shows the shortest duration (DUR) for various magnitudes of human saccadic movements. These bounds are the most important because they summarize the optimal performance of this biological system. Equations that fit the two bounds are:

$$PV = 850\,(1 - e^{-\Delta\theta/10.6})\ \text{deg/sec}\,,$$

$$DUR = (1.7\,\Delta\theta + 20)\ \text{ms}\,.$$

Fig. 3 shows simulations of ten degree saccades and vergences. To show how well the model fits real eye movement data, model output values were superimposed on the Main Sequence diagrams (fig. 4).

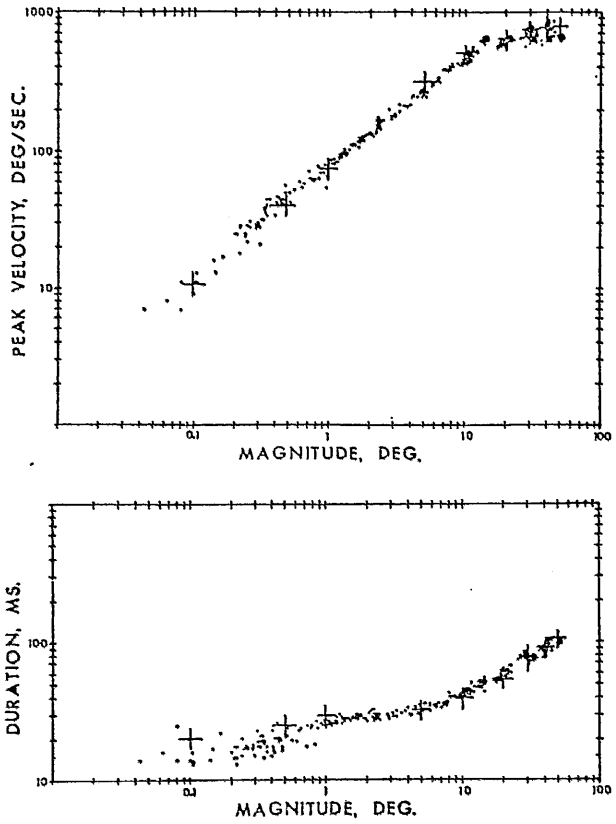



Fig. 4. The Main Sequence for human eye movements. When duration and peak velocity were plotted versus saccadic magnitude, data points for normal humans cluster together as shown. Crosses show model response for various magnitude saccades.

## 5. Sensitivity study

Sensitivity analysis has been used for some years in computer aided simulation studies and problems in operations research. Essentially, the relative effect of changes in various model parameters on system performance is determined. For system design purposes, values of critical parameters can be refined while parameters with little effect can be ignored or eliminated. For understanding physiological systems, sensitivity analysis provide a means of examining the overall validity of a model by comparing inferences from analysis to conclusions drawn from physiological data. With a valid homeomorphic model, sensitivity analysis can also help in diagnosis of disorders caused by disease and suggest logical starting points for developing bio-engineering approaches to treatment.

There is a dilemma that if a model is completely validated, a sensitivity analysis will not be very fruitful but if a model is not valid, one does not know how much confidence can be placed in the sensitivity study. Miller [17] in a study of pollutants in the Ottawa River showed that meaningful information can be provided with a sensitivity study even on an uncertain model. For the eye movement plant, not all of the physiology of underlying processes are well understood. Although the model accurately reproduces available data, the sensitivity analysis provided us with direct information concerning the importance of each parameter and suggested new experiments for further understanding of the system and updating the model.

A parametric sensitivity analysis was done for a ten degree saccade because this is a normal physiological magnitude and data are both abundant and relatively noise free. Each parameter was varied from 20–200% of the value used for producing a 'good' ten degree saccade while the other nineteen parameters were held constant. Fig. 5 shows examples of trajectory

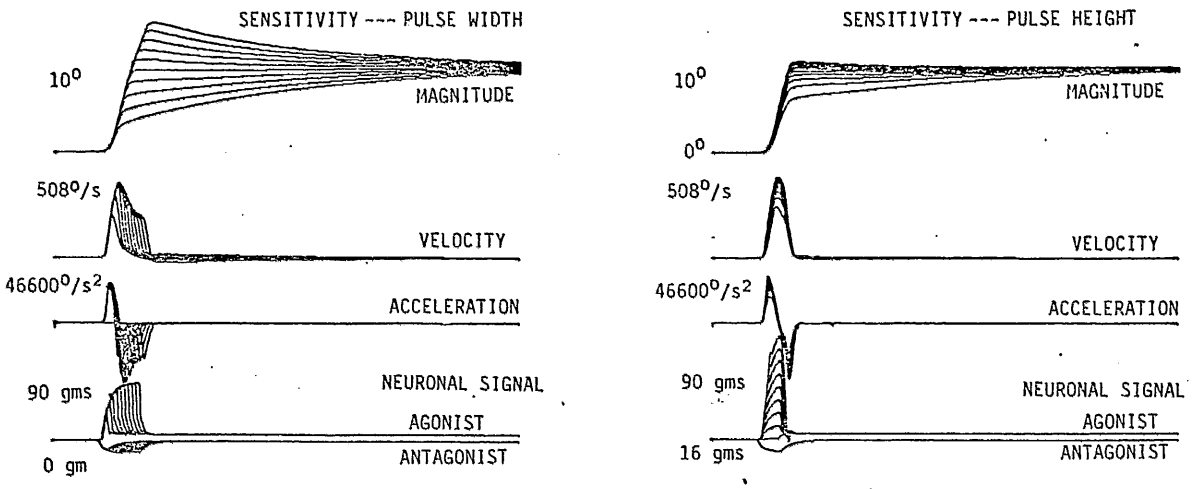
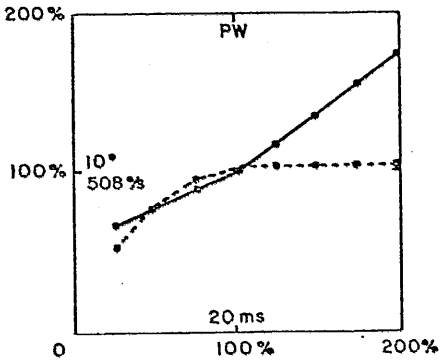
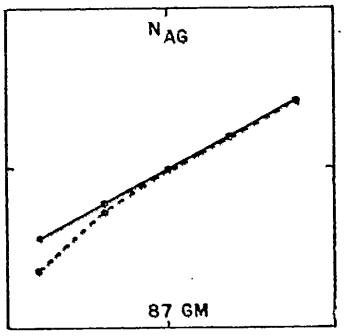



Fig. 5. Trajectories of position, velocity and acceleration for the eye movement plant as pulse width (PW) – left, and pulse height (PH) – right, are varied from 20–200% of the value used for producing a 'good' ten degree saccade. Varying pulse width had a large effect on the magnitude of saccade but a small effect on peak velocity. The shapes of the velocity and acceleration trajectories are drastically changed nevertheless. Varying pulse height affects all trajectories as shown.




variations when pulse width (PW) and pulse height (PH) were varied. Resultant trajectories were noted and both normalized magnitude and normalized peak velocity were plotted versus the normalized value of each parameter (figs. 6,7). The slopes of these sensitivity curves (sensitivity coefficients) give a emasure of the relative importance of each parameter. The steeper the slope, the more eye movement behavioral changes are produced by standard variation of that parameter. For instance, when the pulse width was 20 ms, the saccadic magnitude was 10 degrees. When pulse width was doubled to 40 ms, the saccadic magnitude was 17.3 degrees, increasing by 73%. However, doubling the series elasticity of agonist from 1.8 gm tension/deg to 3.6 gm tension/deg only produced an increase of 17% in saccadic magnitude. The slope of the magnitude sensitivity curve for pulse width was 0.54 compared to 0.1 for series elasticity of the agonist.

Fig. 6. Sensitivity curves for the two controlling parameters for the human eye movement plant: pulse width (PW) and pulse height (PH). Magnitude sensitivity coefficients are 0.54 for PW and 0.519 for PH. Among the twenty internal parameters, the model outputs are most sensitive to these two parameters. Note that doubling the value of pulse width has relatively little effect on peak velocity. Solid line: magnitude sensitivity. Dotted line: peak velocity sensitivity.

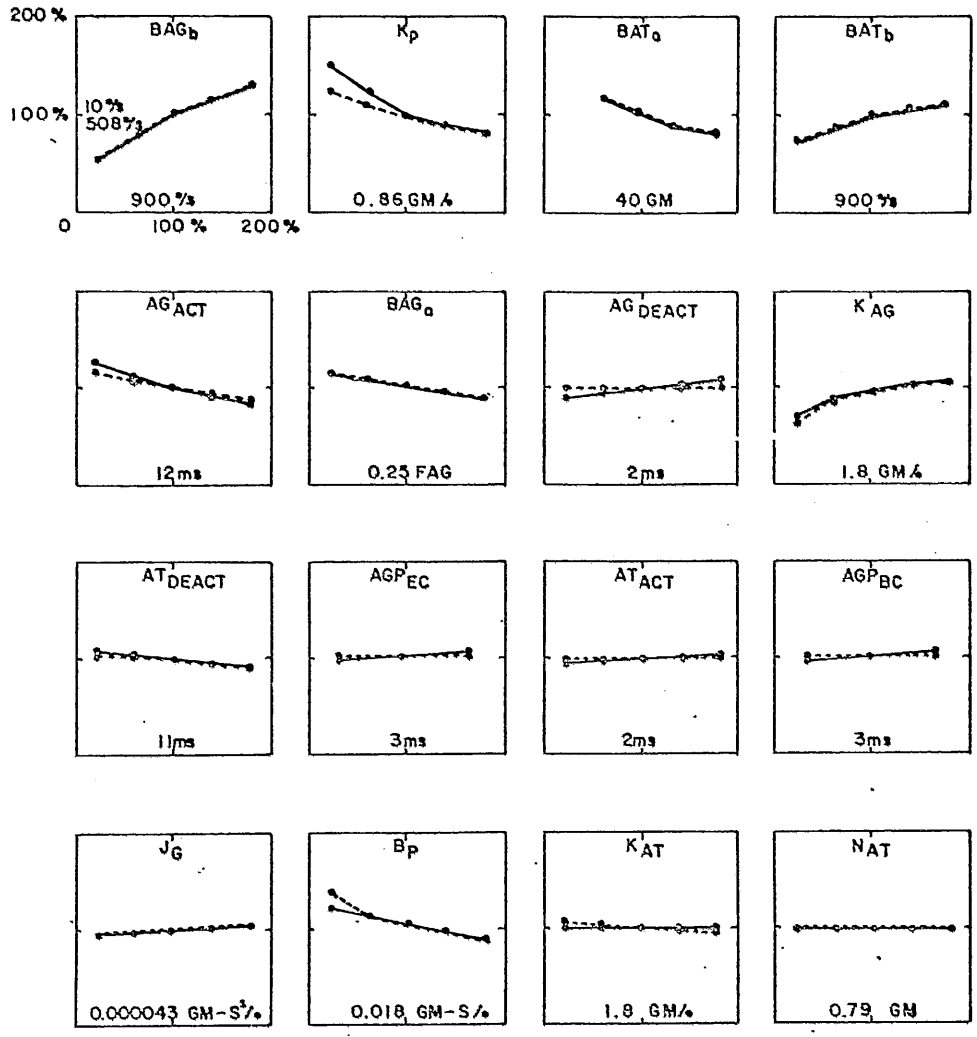



Fig. 7. Rank ordered sensitivity curves for sixteen of the internal parameters. The actual values of each parameter used for simulating a ten degree saccade is given along the X-axis and normal output values along the Y-axis. Output behavior was studied while each internal parameter was varied from 20–200% of its value used for producing a 'good' ten degree saccade. Note that the sensitivity curves for agonist and antagonist tonic levels are not presented since they only have effect on duration (they have glissades at the end portion of their trajectory). Solid line: magnitude sensitivity. Dotted line: peak velocity sensitivity.

## 7. Results and discussion of sensitivity analysis

Fig. 5 shows trajectory variations of saccadic eye movements due to variations in PW and PH. Fig. 6 and 7 show the sensitivity curves of the internal parameters. Table 2 summarizes the magnitude sensitivity coefficients of the twenty internal parameters.

The model is most sensitive to four parameters (table 2) – PW, PH, $AG_b$, $K_p$ – all of whose sensitivity coefficients exceeded 0.4. From the Main Sequence diagrams, we can roughly divide the magnitude range into three regions judging from the slopes of the curves: the first region for saccades of smaller than one degree, the second for saccades greater than ten degrees and the third for saccades in between one and ten degrees. The duration of a saccade is strongly correlated to the duration of the high-frequency burst of oculomotoneuronal firing (PW). We see that large amplitude sac-

Table 2
Sensitivity coefficients: slopes of magnitude sensitivity curves.

| | | |
|---|---|---|
| 1 Pulse width | PW | 0.540 |
| 2 Pulse height-agonist | PH-NAG | 0.519 |
| 3 Agonist dashpot-*b* | $BAG_b$ | 0.505 |
| 4 Parallel elasticity | $K_p$ | 0.432 |
| 5 Agonist dashpot-*a* | $BAT_a$ | 0.316 |
| 6 Antagonist dashpot-*b* | $BAT_b$ | 0.263 |
| 7 Agonist activation time constant | $AG_{act}$ | 0.250 |
| 8 Agonist dashpot-*a* | $BAG_a$ | 0.160 |
| 9 Agonist deactivation time constant | $AG_{deact}$ | 0.113 |
| 10 Agonist series elasticity | $K_{AG}$ | 0.100 |
| 11 Antagonist deactivation time constant | $AT_{deact}$ | 0.094 |
| 12 Pulse width-end circumscription | $AGP_{EC}$ | 0.045 |
| 13 Antagonist activation constant | $AT_{act}$ | 0.038 |
| 14 Pulse width-start circumscription | $AGP_{SC}$ | 0.030 |
| 15 Effective inertia | $J_G$ | 0.030 |
| 16 Globe viscosity | $B_p$ | 0.020 |
| 17 Antagonist series elasticity | $K_{AT}$ | 0.013 |
| 18 Pulse height-antagonist | *PH-NAT* | 0.013 |
| 19 Tonic level-agonist | $NAG_{tonic}$ | glissades |
| 20 Tonic level-antagonist | $NAT_{tonic}$ | glissades |

cades show a strong dependence on PW since the Main Sequence has the greatest slope for large saccades. Small magnitude saccades seem to be controlled only by PH since the Main Sequence duration plot shows a scattergram rather than a tight cluster of points. The region in between one and ten degrees saccades seem to be controlled by both PW and PH. This is confirmed by the sensitivity analysis in that PW and PH are the two most sensitive parameters of the model. The fact that parallel elasticity $K_p$ is fourth on the list is not surprising since it is a lumped parameter representing three separate elasticities – the parallel elasticity of both muscles and the passive elasticity of the globe. $K_p$ is a passive element of the plant and is not a control parameter. The sensitivity of the model to $AG_b$ is surprising. $AG_b$ is a constant in the Hill equation for the force-velocity relationship of muscles. This parameter had been extensively studied by Hill and he estimated that $AG_b$ should be approximately $1/4\,V_{max}$. But $V_{max}$ must be evaluated for each muscle studied. Cook and Stark estimated $V_{max}$ to be 3600°/s. Because this model is so sensitive to $AG_b$, we have started experiments to investigate it in greater detail. This unexpected result has stimulated new experiments and thus illustrates the value of a sensitivity analysis for models.

From table 2, we see that the other three variables of the two muscle dashpot coefficients $AT_a$, $AT_b$, $AG_a$ rank 5, 6 and 8, respectively, in their sensitivity coefficients. This shows that viscosity has a relatively strong effect on output behavior. Although skeletal muscle behavior has been studied extensively, in vivo measurements of extraocular muscles have only been done by relatively few experimenters [18] due to the difficulties in doing research on human subjects. However, the sensitivity analysis points out that data on viscosity of the extraocular muscles are crucial to improving our understanding of the eye movement plant.

The physiology of activation-deactivation processes of active state tension of muscles is not well understood. Their time constants have a secondary effect on behavior [19] as confirmed by their ranks in table.2. Another interesting fact is that series elasticity of agonist has a sensitivity coefficient of 0.1 as compared to 0.013 of series elasticity of antagonist. This is probably due to the greater force exerted by the agonist than the antagonist.

## 8. Conclusion

Our reciprocal innervation model reproduced physiological data accurately for both saccadic and vergence eye movements over three orders of magnitude. A sensitivity analysis on ten degree saccades was performed on the twenty internal parameters. These results showed the most sensitive parameters to be pulse width and pulse height, the two controlling parameters representing neuronal firing for saccadic control. Passive elements representing the eyeball, surrounding muscles and tissues had low sensitivity coefficients and have little effect on the dynamic behavior of the model. This contrast is in concurrence with the fact that most saccadic trajectory variability is of neural, not muscular origin [20,21,22]. Further studies will be needed to understand why the model is so sensitive to viscosity of the muscles, especially to $AG_b$ of the Hill equation. The usefulness of a sensitivity analysis for simulation studies is illustrated by the fact that old ideas were confirmed, new insights gained, and direction for further research was provided.

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