# Unequal Saccades Generated by Velocity Interactions in the Peripheral Oculomotor System

ROBERT V. KENYON
*Man-Vehicle Laboratory, Massachusetts Institute of Technology, Cambridge, Massachusetts 02139*

AND

LAWRENCE STARK
*Neurology Unit, University of California, Berkeley, California 94720*

*Received 12 August 1982*

ABSTRACT

Experiments with precision eye movement recordings show binocularly unequal saccades to be present under several stimulus conditions having as a common theme ongoing low velocities at the times of the saccades. Simulations using a model of eye muscles and eyeball dynamics reproduce these unequal saccades in quantitative agreement with the experimental findings. The model uses equal innervation for the saccades, and demonstrates a peripheral interaction between the muscle forces and the eye velocities to be the cause of the large inequality of the simulated binocular saccades. Thus, the simulations provide evidence that Hering's law continues to describe the innervation patterns to corresponding muscles producing these binocularly unequal saccades found in the experimental situation.

## INTRODUCTION

The two eyes are so related to one another, that one cannot be moved independently of the other; rather, the musculature of both eyes reacts simultaneously to one and the same impulse of will. ...To the mobilizing will, it is irrelevant that this organ really consists of two separate parts because it is not necessary to move each part separately; rather, one and the same impulse of will directs both eyes simultaneously as one can direct a pair of horses with single reins [1].

These observations by Ewald Hering describe his hypothesis on the fundamental mechanism that controls the movement of the eyes as a single organ. Hering proposed that corresponding muscles in each eye receive equal innervation and thus are neurologically yoked in performing binocular eye rotations. Electromyographic recordings of the innervation pattern in corresponding muscles during horizontal eye movements have shown this relation-




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ship to dominate the neurological control of the eyes [2–4]. Consequently, this relationship, now known as Hering's law of equal innervation, has become a basic tenet in the analysis of oculomotor control by experimenter and physician.

Thus the conjugate pair of saccades that occurs when shifting gaze is expected to be synchronous and equal in amplitude and to have similar dynamic trajectories. Saccades are high velocity binocular eye movements used, among other things, to scan our visual surroundings and fixate objects of interest. Saccades can move the eye at velocities of 1000°/sec. A good review can be found in Bahill and Stark [5].

A particularly strong apparent contradiction of Hering's law is found when interpreting different saccadic amplitudes in each eye. Very often, unequal saccades have been attributed to unequal innervation patterns and taken as a sign of pathology [6–8]; however, recent reports have shown that saccades during vergence eye movements have consistently unequal amplitudes [9–11]. This inequality cannot be accounted for solely by linear summation of the measured saccadic amplitude with the calculated vergence amplitude that would have occurred had there been no saccade [9]. This report investigates the principle of Hering's law under conditions that produced significantly unequal saccadic amplitudes and shows that such inequalities could be produced in the presence of equal innervation.

## METHODS

Saccade vergence interactions were studied by having a subject change fixation between two targets along the midline, one at 25 cm and the other at 50 cm from the plane through the centers of rotation of the eyes [11]. While such a target results in mainly pure disparity vergence responses, at times, these responses also contained binocular saccades. It was these saccades that were analyzed in this report. The binocular eye position was recorded using the photocell method which tracks the nasal and temporal limbal regions of the eye; the head was stabilized by a head and chin rest [12].

## RESULTS

Saccadic eye movements occurred unpredictably during our subjects' vergence responses. These saccade vergence interaction episodes produced unequal saccades during both convergence and divergence [Figure 1(a)]. The smaller saccade of each pair was consistently associated with the eye for which the vergence opposed the conjugate saccade direction. Also, larger inequalities were correlated with larger velocities opposing the saccadic eye movements; for vergence this occurred early in the response [see velocity trace, Figure 1(a)]. The relative saccadic amplitude, defined as the ratio (in percent) of the saccadic amplitude opposing the vergence to its counterpart

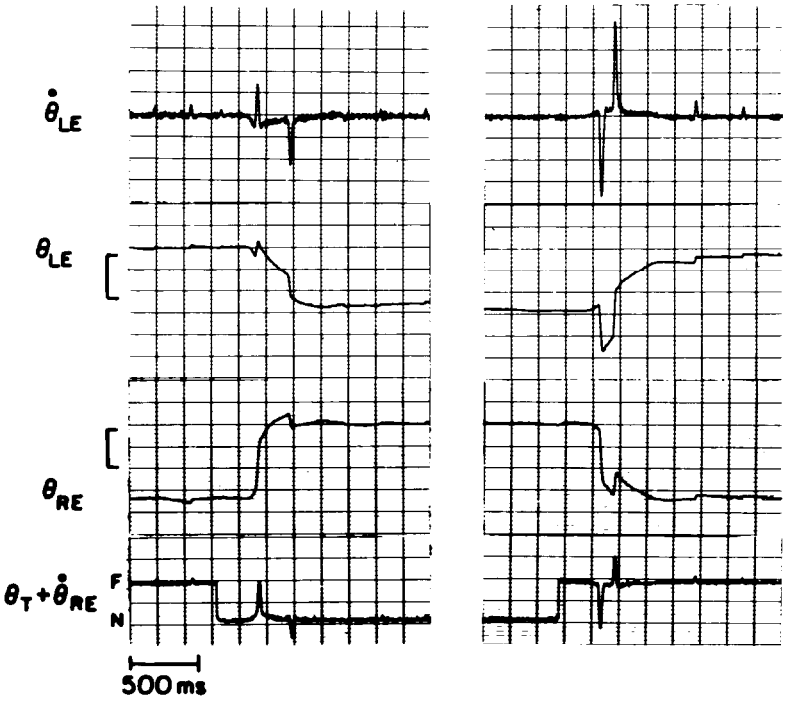


FIG. 1. (a) Binocular eye movements showing vergence-saccade interaction. Saccades during early portions of vergence movement show larger inequalities than those occurring later in vergence. Amplitude of saccade opposing vergence movement is less than that of fellow eye saccade. The occurrence of saccades during symmetric vergence responses were unpredictable in time and amplitude. These saccade-vergence responses were intermingled with smooth symmetric vergence responses as well. $\theta_{RE}$ and $\theta_{LE}$ represent right and left eye position; $\dot{\theta}_{RE}$ and $\dot{\theta}_{LE}$ represent right and left eye velocity; $\theta_t + \dot{\theta}_{RE}$ represents linear summation of target position marker with right eye velocity signal: $F$ = far target illuminated, $N$ = near target illuminated. Calibration bars are 2°; pen deflections up are movements left.

in the fellow eye, was plotted as a function of time after the start of vergence responses [Figure 2(a)]. The largest difference in saccadic amplitudes, as much as 60% attenuation, occurred early in the vergence movement. Then, with a roughly exponential rise, the ratio returned to the equal amplitude value at 700 ms.

The general exponential character of the data in Figure 2(a) suggested that the vergence velocity, which decreased exponentially with time, might have been modulating saccadic amplitudes. Portions of the data from Figure 2(a) were replotted [Figure 2(b), open circles] to investigate this possible relationship between relative saccadic amplitude and vergence velocity prior to the

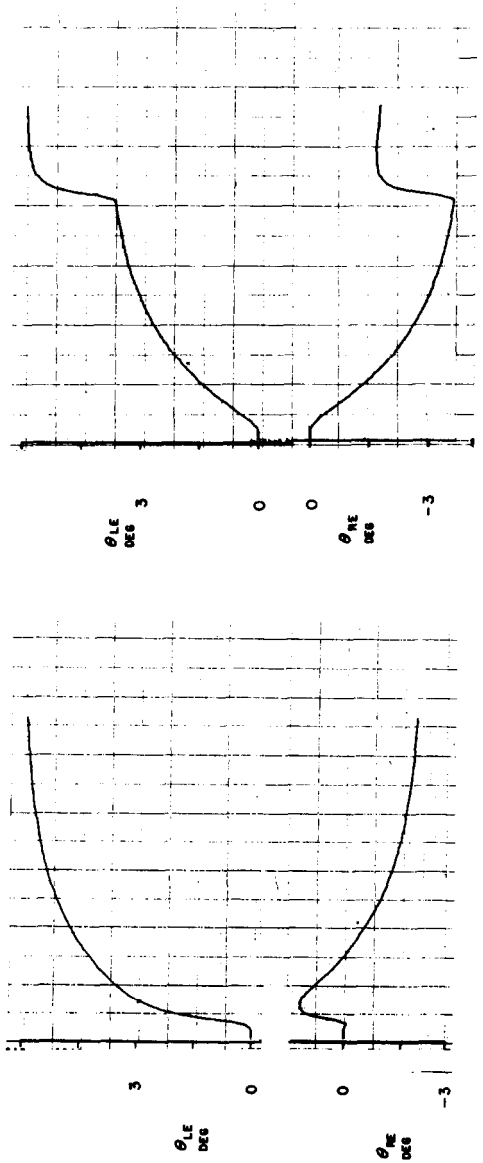


FIG. 1. (b) Model response to a programmed 4° vergence and 2.1° saccade delayed by 25 and 300 ms after the start of the vergence. Saccadic inequalities at 25 ms are larger than those at 300 ms. Saccades opposing vergence are smaller than fellow eye saccades. The shapes of the control signals represent frequency-of-firing envelopes of neural activity used to activate the extraocular muscles during eye movements. Our modeling results also indicate that initial eye velocity affects saccadic amplitudes asymmetrically, reducing opposing saccades much more than increasing concurrent saccades. Time scale is 40 ms/div; pen deflections up are movements left.

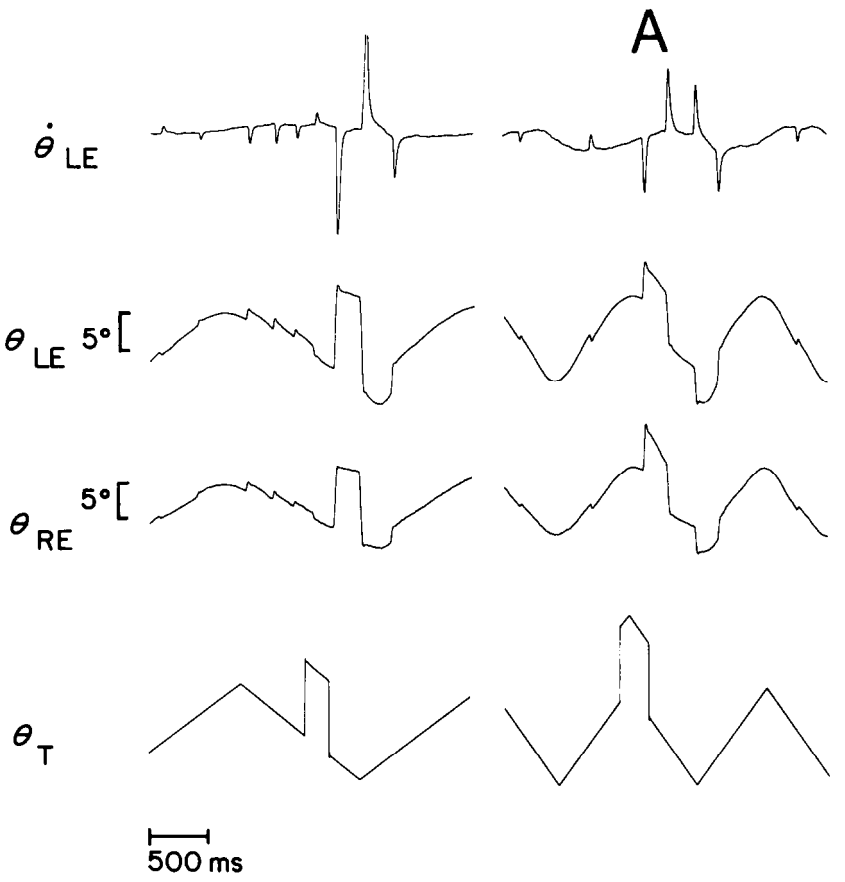


FIG. 1. (c) Binocular eye movements while tracking a constant velocity target with unpredictable pulse changes in position. On the right side (marked A), the first two successive saccades have different amplitudes despite equal displacement of the target. Since a reference saccade is not present for comparison as in vergence, it might be that these saccades were programmed to be different in amplitude. Notation as in (a). Stimulus velocity 2.5°/sec (left) and 8.0°/sec (right); target displacement 10°.

saccade. These data revealed that saccadic inequalities were greatest when the velocity was greatest, demonstrating that the ongoing velocity is an important condition for the occurrence of saccadic inequalities. The systematic changes in saccadic inequalities as functions of time and vergence velocity led us to hypothesize that these saccades were not produced by unequal saccadic innervations to each eye, i.e. violations of Hering's law of equal innervation. Instead, we reasoned that Hering's law was observed but that the eye velocity prior to the saccade influenced the saccade size through interactions within the "peripheral" (i.e., muscle globe) oculomotor system.

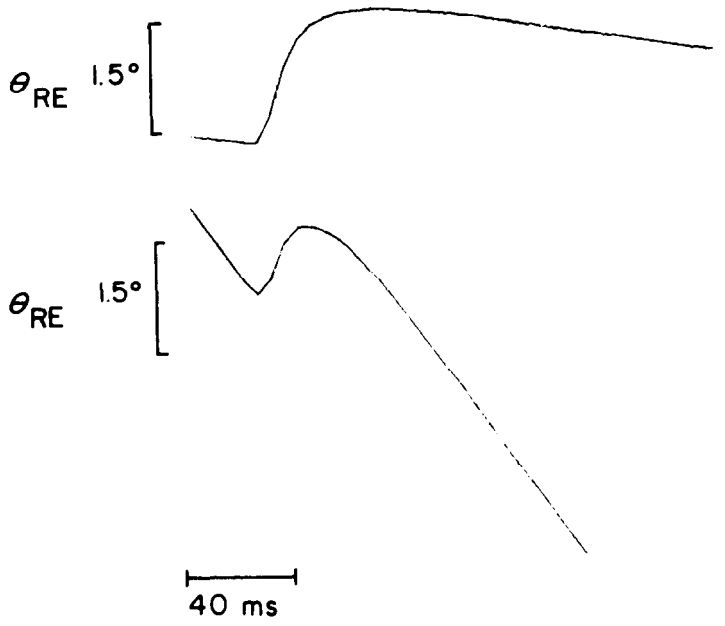


FIG. 1. (d) Model responses of one eye to a programmed 2.1° saccade during a 5°/sec and a 25°/sec smooth pursuit eye movement. Saccades injected 400 ms after start of smooth pursuit to insure constant velocity. Saccade opposing 25°/sec velocity measures 1°, instead of 2.1° with no smooth pursuit velocity; saccade opposing 5°/sec velocity is much closer to the reference 2.1° saccade.

We investigated the validity of this hypothesis by simulating saccade vergence interactions on a minicomputer using a sixth order nonlinear model of the eye and orbit. The model incorporated reciprocal innervation and a nonlinear force-velocity relationship [13–16]. Control signals used to drive the model were linearly summated vergence (step) and saccadic (pulse-step) control signals, each innervating its own corresponding muscle pair according to Hering's law. The model responses for a 4° vergence with a 2.1° saccade occurring 50 ms [Figure 1(b), left] and 300 ms [Figure 1(b), right] after the start of the vergence, displayed two important characteristics found in our experimental data. First, the saccade opposing the vergence was smaller than the saccade moving with the vergence; in fact, the opposing saccade was smaller than saccades simulated with no initial eye velocity. Second, as the saccades occurred earlier in the vergence, the saccadic inequalities increased. To demonstrate the general applicability of our model responses to experimental data, relative saccadic amplitudes from a series of simulations with saccades occurring at 50 to 400 ms after the start of a 4° and a 6° vergence were plotted with the experimental data [Figure 2(a) and (b)]. Model data

versus time [Figure 2(a)] showed the same general exponential shape as the experimental data; these same data plotted against vergence velocity prior to the saccade [Figure 2(b)] showed a similar close agreement with experimental results.

The model responses showing saccadic inequalities despite adherence to Hering's law suggest that peripheral oculomotor mechanisms alone are capable of producing saccadic inequalities. Moreover, this peripheral interaction predicts the same quantitative effects on saccadic amplitude when an initial eye velocity is produced by other types of slow eye movements. Documenting these effects, however, is more difficult for conjugate slow eye movements than for vergence, since only vergence supplies both opposing and concurrent ongoing velocities with each saccadic pair; conjugate eye movements affect the binocular saccadic pair identically in each eye. Nevertheless, systematic differences in saccadic amplitudes have been reported between saccades opposing smooth pursuit velocity and those moving concurrently with these velocities [17]. Experimental data [Figure 1(c)], showing two successive saccades with different amplitudes, were obtained by having subjects track a constant velocity target with randomly occurring equal amplitude pulsed target displacements. For example, the data marked "A" in this figure show the smaller saccade in opposition to the smooth pursuit velocity. Simulation of smooth pursuit and saccade interactions [Figure 1(d)] showed a similar result, with the saccades opposing the initial condition velocity having smaller amplitudes as higher initial velocities were used. Relative saccadic amplitudes for a series of such simulations spanning velocities from 0° to 80°/sec also accurately modeled the entire range of saccade vergence experimental data [Figure 2(b)]. These results reinforce the importance of the ongoing velocity to a peripheral oculomotor effect on saccadic amplitudes.

Experimental data on saccades during coordinated head-eye movements provided another instance in which a peripheral interaction could account for a reduction in saccadic amplitudes. To review briefly, changing gaze via a head-eye coordinated movement results in a vestibularly induced compensatory eye movement that opposes the trajectory of a fixation saccade. Reports in the literature have shown that these fixation saccades have smaller amplitudes when the head rotates than when the head is stationary [18]. Investigators have hypothesized that the vestibular system modulates the generation of saccadic innervation to produce smaller saccades during head movements [18]. Nevertheless, this is also a situation in which the saccades oppose an initial eye velocity, for the head rotation case, or no velocity, for the head stationary case. We therefore simulated a 35° saccade and a 40° compensatory vestibular eye movement similar in amplitude to those published by Morasso et al. [18] to investigate whether a peripheral interaction rather than an internal neural modulation could account for these changes in

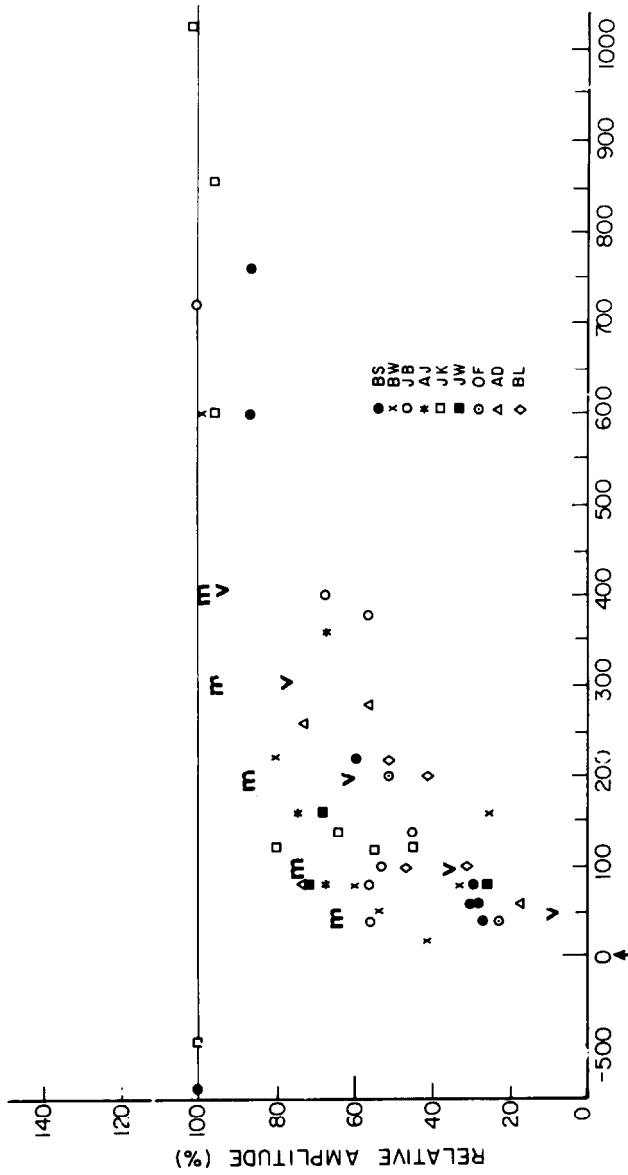


FIG. 2. (a) Saccadic inequality as a function of time after vergence. Data from nine subjects defined as symbols to right; model simulations represent a 2.1° saccade interacting with a 4° vergence (*m*) and a 6° vergence (*v*). Plot shows larger inequalities near start of vergence with saccade opposing velocity smaller than fellow eye's saccade. Since the data were plotted with the saccade opposing vergence as the numerator of that ratio, any counterexample to the opposing saccade being the smaller would have resulted in points above the 100% line. This did not occur in this plot in the region of large saccadic attentuation (0–300 ms).

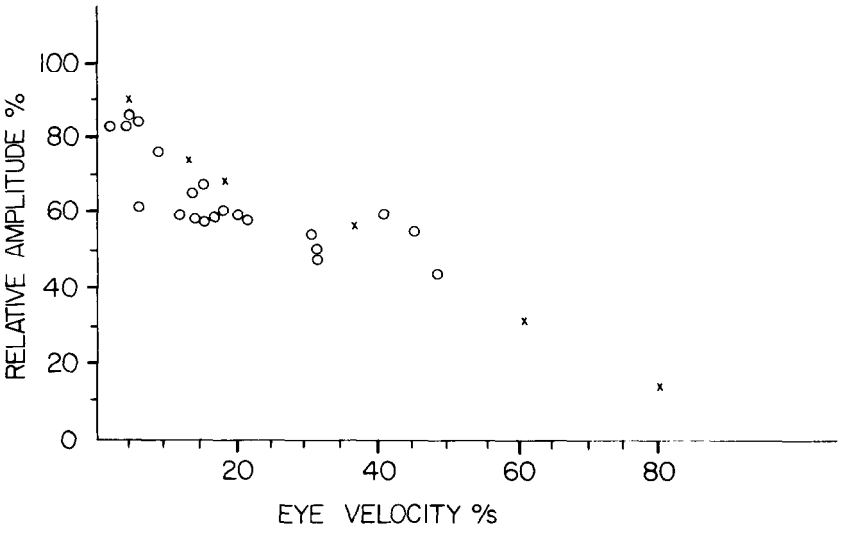


FIG. 2. (b) Saccadic inequality plotted against the velocity of the eye just prior to saccade initiation. Open circles represent subject data, and points plotted with an $x$ are from modeling data using both the 4° and 6° vergence data to span a range of velocities.

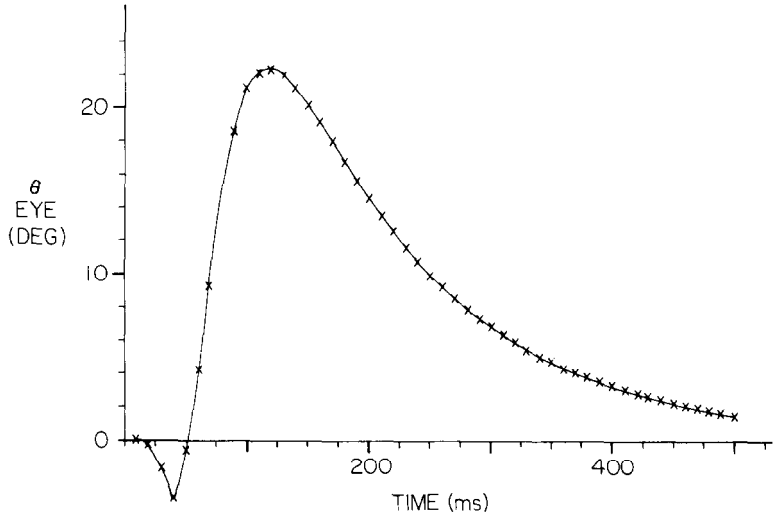


FIG. 2. (c) Simulation of a vestibular compensatory movement of 40° interacting with a fixating saccade of 35°. Measured saccadic amplitude equals 26° when the saccade occurs 25 ms after start of vestibular compensatory movement.

saccadic amplitude. Our simulations [Figure 2(c)] demonstrated a 25% reduction in the measured amplitude of the saccade during vestibular compensation compared to the case in which there is no ongoing compensatory eye movement. Comparing this result with published eye movement records of Morasso et al. [18] revealed a saccadic amplitude difference of 5% between our simulated and their experimental results.

## DISCUSSION

While our model responses do not eliminate the possibility of non-Hering's-law innervations or vestibular neural interactions, they present strong evidence that changes in saccadic amplitudes need not be explained exclusively by neural effects. The model's faithful reproduction of subjects' saccadic interactions with vergence, smooth pursuit, and vestibular compensatory eye movements, without changes in saccadic control signals, establishes a peripheral oculomotor interaction as a serious alternative explanation for these changes in saccadic amplitudes. Peripheral oculomotor mechanisms that are components of our model and appear to be logical mechanisms for these effects include the nonlinear force-velocity characteristics of muscle [19–21] and its asymmetric activation-deactivation time constants [22]. Clearly, eye movement analysis of saccadic amplitudes when an ongoing slow eye movement is present must now address the role of peripheral oculomotor effects in explaining changes in saccadic amplitude and cannot relegate such inequalities to changes in neural control signals exclusively.

## REFERENCES

1 E. Hering (1868), in *Theory of Binocular Vision* (B. Bridgeman and L. Stark, Eds.), Plenum, New York, 1977, pp. 17,19.

2 E. Marg, A. Jampolsky, and E. Tamler, Elements of human extraocular electromyography, *Arch. Ophth.* 61:258–269 (1959).

3 G. M. Breinin and J. Moldaver, Electromyography of the human extraocular muscles, *Arch. Ophth.* 54:200–210 (1955).

4 G. M. Breinin and J. Moldaver, Electromyography of the human extraocular muscles, *Arch. Ophth.* 54:407–411 (1955).

5 A. T. Bahill and L. Stark, Trajectories of saccadic eye movements, *Sci. Amer.* 240:506–516 (1979).

6 H. Davson, *The Physiology of the Eye*, Academic, New York, 1972, p. 331.

7 A. T. Bahill, K. J. Ciuffreda, R. V. Kenyon, and L. Stark, Dynamic and static violations of Hering's law of equal innervation, *Amer. J. Opt. Phys. Opt.* 53:786–796 (1976).

8 D. Cogan, *Neurology of the Ocular Muscles*, Charles C. Thomas, Springfield, Ill., 1948, pp. 10.32, 57.60, 92.

9 R. V. Kenyon, K. J. Ciuffreda, and L. Stark, Binocular eye movements during accommodative vergence, *Vis. Res.* 18:546–555 (1978).

10 H. Ono, S. Nakamizo, and M. Steinbach, Non-additivity of vergence and saccadic eye movement, *Vis. Res.* 18:735–739 (1978).

11 R. V. Kenyon, K. J. Ciuffreda, and L. Stark, Dynamic vergence eye movements in strabismus and amblyopia: symmetric vergence, *Invest. Ophth. and Vis. Sci.* 19:60–74 (1980).

12 L. Stark, G. Vossius, and L. Young, Predictive control of eye tracking movements, *IRE Trans. Human Fac. Electr.* HFE-3:52–57 (1962).

13 G. Cook and L. Stark, The human eye-movement mechanism: experiments, modeling and model testing, *Arch. Ophth.* 79:428–436 (1968).

14 M. Clark and L. Stark, Time optimal behavior of human saccadic eye movement, *IEEE Trans. Automat. Control* AC-20:345–348 (1975).

15 F. K. Hsu, T. A. Bahill, and L. Stark, Parametric sensitivity analysis of a homeomorphic model for saccadic and vergence eye movements, *Comput. Prog. Biomed.* 6:108–116 (1976).

16 S. Lehman and L. Stark, Simulation of linear and nonlinear eye movement models: sensitivity analyses and enumeration studies of time optimal control, *J. Cybernet. and Inform. Sci.* 4:21–43 (1979).

17 R. Schilling, Saccadic trajectory superimposed on the smooth pursuit movements, Master's Thesis, Univ. of California, 1970.

18 P. Morasso, E. Bizzi, and J. Dichgans, Adjustment of saccade characteristics during head movements, *Exp. Brain Res.* 16:492–500 (1973).

19 W. O. Fenn and B. S. Marsh, Muscular force at different speeds of shortening, *J. Physiol. (London)* 85:277–297 (1935).

20 A. V. Hill, The heat of shortening and the dynamic constants of muscle, *Proc. Roy. Soc. Ser. B* 96:136–195 (1938).

21 B. Katz, The relation between force and speed in muscular contradiction, *J. Physiol. (London)* 96:45–64 (1939).

22 S. Ebashi and M. Endo, Calcium ions and muscle contraction, *Progr. Biophys. Mol. Biol.* 18:123–134 (1968).