The Main Sequence, A Tool for Studying Human Eye Movements

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ABSTRACT

The astronomical term "main sequence" has been applied to the relationships between duration, peak velocity, and magnitude of human saccades over a thousandfold range of magnitude. Infrared photodiodes aimed at the iris-sclera border and a digital computer were used in experiments to derive the main sequence curves. In the pulse width modulation model, the duration of the controller signal pulse determines saccadic amplitude and peak velocity. The high-frequency burst of the oculomotoneurons needs to be only one-half the duration of the saccade, because of the "apparent inertia" of the eyeball.

INTRODUCTION

Astronomers refer to the important relationship between the brightness of a star and its temperature as the "main sequence".[1] The relationships between duration and magnitude and between peak velocity and magnitude over a very wide range of human saccades are analagously called the main sequence for normal saccades and have been used to interrelate several hypotheses concerning the generation and control of saccades.

[1]Astronomy's main sequence, or the Hertzsprung-Russel diagram, often has color, not temperature, as the independent variable. Color and surface temperature are directly proportional for blackbody radiators, however, and stars are reasonable approximations of blackbody radiators.

In the early part of this century, astronomers believed that as stars aged, they moved along the main sequence. The appearance of stars that were not on the main sequence conflicted with this theory. In seeking an explanation for this dilemma, they studied non-main sequence stars. This eventually led to a new theory for stellar evolution: stars spend over 99% of their visible lives in almost the same spot on the main sequence; however, their births and deaths follow a circuitous route in the brightness-temperature plane.

Saccadic duration, average velocity, and peak velocity all increase as the size of the saccade increases. The duration of the saccade and the average velocity are really measures of the same parameter. Dodge and Cline [17] realized this fact and showed that duration, and therefore average velocity, increase as saccadic magnitude increases. This relationship was also noted by Brockhurst and Lion [6], Ditchburn and Ginsborg [16], Yarbus [48], Hyde [25], Gurevich [22], and Mackensen [31]. The peak velocity of a saccade also increases as the saccadic magnitude increases. For small saccades this increase is linear, but approaches a soft saturation limit for large saccades, as shown by Westheimer [47], Hyde [25], Vossius [46], Zuber, Stark, and Cook [49], and Mackensen [31]. Fuchs [19], and Henn and Cohen [23] have shown that these same results hold for the monkey. Thus, many experimenters, using a variety of measurement techniques over different saccadic magnitude ranges, agree that the duration and peak velocity of saccades increase as the magnitude of the saccade increases. This paper's research extends the range to three orders of magnitude, refines the measurements, unifies the concepts, and with the aid of a homeomorphic model, clarifies certain critical points of the relationships between the magnitude of a saccade and its duration, velocity, and underlying neurophysiological control mechanisms.

The concepts embedded within the main sequence are powerful tools for studying eye movements. The peak velocity versus saccadic magnitude plot shows that a common physiological system produces both large saccades and microsaccades [49] as well as producing both voluntary saccades and the fast phase of optokinetic nystagmus [27]. Further, it defines "normalcy" of the extraocular plant, and therefore, localizes the defect to the saccadic controller in a patient with abnormally sized saccades [14]. The duration versus saccadic magnitude plot has been used to show two facts: that the resulting eye movements are saccades when the frontal eye fields [37] and the cerebellum [38] are stimulated, and that a common physiological system produces both voluntary saccades and the fast phase of rotary vestibular nystagmus [39].

With the present extension and refinement of these important concepts, the main sequence should become an even more useful tool for studying eye movements and their neurophysiological control.

EXPERIMENTAL METHODS, RESULTS, AND ANALYSIS

Two infrared photodiodes aimed at the iris-sclera border [41] and a small on-line digital computer were used to derive the main sequence curves. The accuracy of the measurement is limited by calibration techniques. For calibration, the subject fixates on a small spot of light and

suppresses his microsaccades, so that the eye will be steady within ± 3 minutes of arc. Steinman, Haddad, Skavenski and Wyman [42] have a detailed description of microsaccades and their voluntary suppression.

The peak velocity of the saccade was easily determined by digital differentiation in the computer; however, the definition of the duration of a saccade posed problems. It is sometimes difficult to determine the exact beginning or ending of a saccade by using the position versus time record. It is easiest to determine the duration of a saccade by looking at its record of eye velocity versus time. A simple definition might be the time elapsed from the point the velocity is zero at the beginning of the saccade to the point when it reaches zero at the end of the saccade. This first method runs into trouble when the saccades have dynamic undershoot or dynamic overshoot, like the saccades of Fig 1(A) and (B), respectively.

The zero velocity to zero velocity definition is not used on saccades with a dynamic undershoot, because this makes the duration inconsistently long.

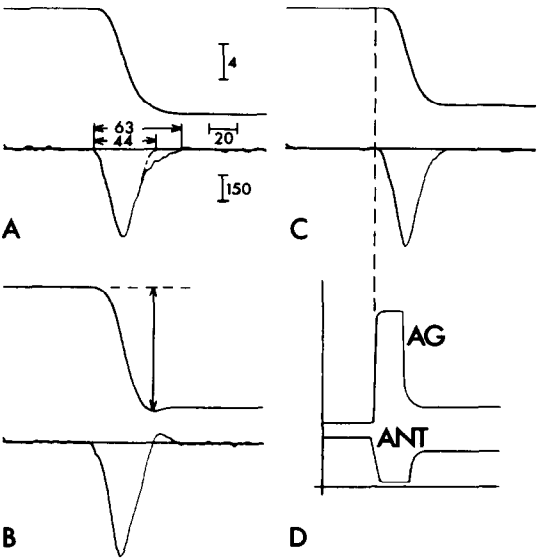

FIG. 1. Position (upper) and velocity (lower) records for a saccade with dynamic undershoot (a), dynamic overshoot (b), and a critically damped saccade (c), and the proposed neuronal controller signal (d). The magnitude, velocity, and duration calibrations shown in (a) apply to all of the saccades. These data are for the same subject during the same experimental run (i.e., within 8 minutes). Each of these three types of saccades can be found, for most subjects, for any magnitude between 3 minutes of arc and 50 degrees, and for any direction.

This paper is concerned with the saccadic system, not the fixation system or the interaction of the two systems, so the prolonged endings of these undershooting saccades are not of immediate interest here. Yet all saccades with overshoot or undershoot should not be discarded. To avoid difficulties in defining the duration of saccades with dynamic undershoot, a second method is used. Find a critically damped saccade of the same magnitude and peak velocity, as shown in Fig. 1(c). Trace the ending of this new saccade onto the velocity record of the undershooting saccade. This corrected ending is shown as a dashed line in Fig. 1(a). This new velocity record is used to determine the duration of the saccade. The duration is defined as the time elapsed between zero velocity at the start and zero velocity at the end. Less then 2% of the points in Figs. 2 and 3 were obtained in this manner, because few saccades have dynamic undershoot.

Unfortunately, this same second technique is not as easily utilized for saccades with dynamic overshoot, which comprised 65% of our records; for these saccades, the velocity records were not altered. The first method, computing the time difference between the zero velocity point at the start of the saccade and the first zero velocity point at the end, was used for the duration. Still other methods—measuring zero velocity to the second zero velocity at the ending, measuring zero velocity to peak negative velocity, tracing in an "ideal" velocity ending, and measuring the 10% to 90% rise time—were tried, but none of these methods was felt to be superior to the one specified. The magnitude of saccades with dynamic overshoot was defined as the angle traversed from the original starting position to the peak of the overshoot, as shown in Fig. 1(b).

Fig. 2 shows that the duration of human saccadic eye movements is related in a nonlinear manner to the amplitude of these movements over a

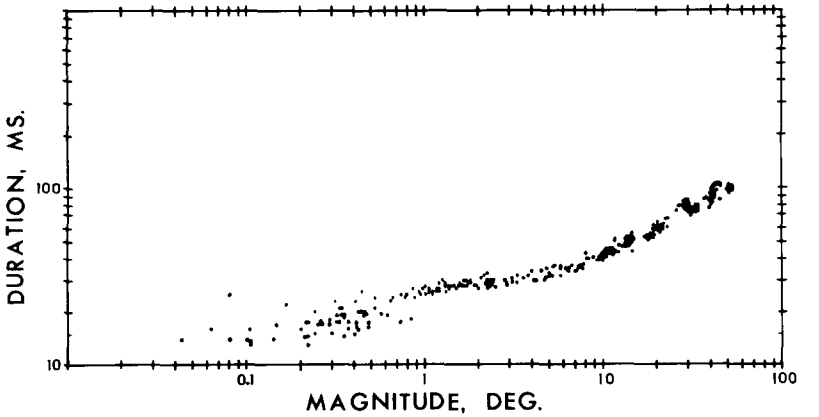

FIG. 2. Duration versus magnitude of human saccadic eye movements.

thousandfold range (from 3 minutes of arc to 50 degrees); data scatter is extremely small. It is important to realize that a linear system would have constant duration, and thus even a straight line relationship is indicative of a strongly nonlinear controller policy.

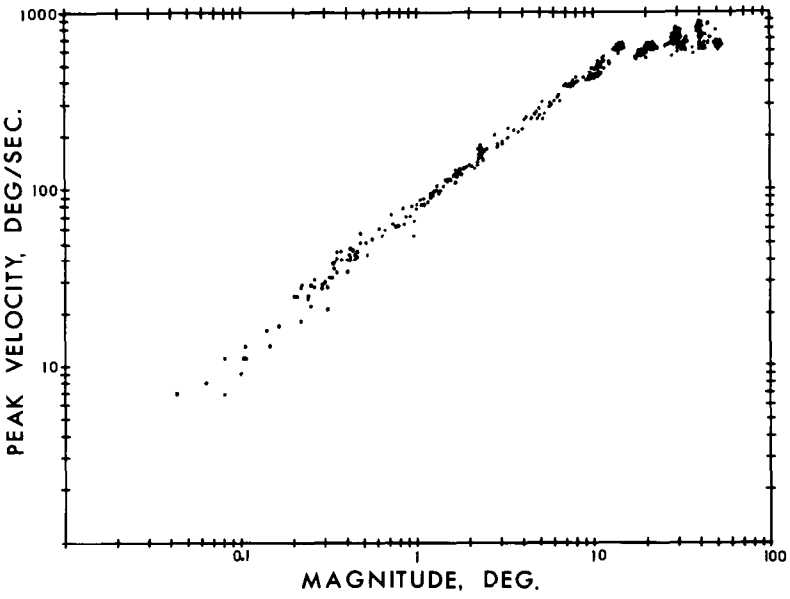

FIG. 3. Peak velocity versus magnitude of human saccadic eye movements.

The peak velocity is similarly related in a quasi-linear manner to saccadic amplitude up to about 15 or 20 degrees, as shown in Fig. 3, where it reaches a soft saturation limit and thereafter does not increase as rapidly.

The points shown in Figs. 2 and 3 exhibit the extreme values for duration and peak velocity for each saccadic magnitude shown. They are not of selected saccades except when, due to the subject being fatigued, the saccades changed in form and showed widely differing parameters and were, therefore, rejected. Upon questioning, the subject was aware of his fatigue. These changes, beyond the scope of the present paper, will be reported in a subsequent paper. The data are from one particular subject on more than a dozen different days, over a period of one year; however, intensive data were taken on five subjects and much less careful measurements on about 160 subjects. The general shapes of the curves are the same, although the exact values and the amount of data scatter varied.

The infrared photodiode method of measuring eye movements yields low noise, linear records for the smallest possible microsaccades[2] and for saccades as large as 25 degrees. The size of the iris and the covering of the iris by the eyelids imposes this upper limit on linearity. Records of saccades between 25 and 50 degrees will be nonlinear at the beginning and at the end of the saccade, because the measurement system goes into soft saturation. The voltage continues to vary, but nonlinearly. The beginning and end of the saccades, and therefore the duration, can still be easily determined. The system is linear in the central region, where the peak saccadic velocity occurs, so the peak saccadic velocity can still be determined.

Main sequence curves were also obtained using electro-oculography (EOG) for the eye movement measurements. The results were similar, except that the velocities were smaller, due to the limited EOG bandwidth, and the data scatter was greater, due to the large noise inherent in the EOG measurement technique. These curves are in accordance with the EOG derived curves of Becker and Fuchs [4].

The main sequence data are for saccades around primary position. No significant systematic dependence was noted upon initial position or saccadic direction, in agreement with the findings of Mackensen (31), Zuber, Semmlow, and Stark [50], Leushina [29], and Troost, Boghen, Daroff, and Birkett [45]. However, adducting saccades larger than 30 degrees often had longer durations and, paradoxically, larger peak velocities than abducting saccades of the same magnitude. These differences, however, were smaller than those caused by daily variations.

THEORY—THE PULSE WIDTH MODULATION MODEL

To produce a saccade, the agonist motoneurons must produce a high frequency burst or pulse of activity and then revert to some lower tonic activity [35]. According to Descartes's law of reciprocal innervation [15,24], the antagonist must also be inhibited and then must revert to a new tonic level, as demonstrated by Cook and Stark [13], Fuchs and Luschei [21], and Bahill and Stark [1] and shown in Fig. 1(d).

In the pulse width modulation model, the width of the controller signal determines the saccadic amplitude and also, because of the main sequence relationships, the duration and peak velocity of the saccade. Knowledge of these three parameters determines the average velocity and even characterizes the general shape of the velocity versus time curves.

The width of the controller signal pulse for saccades of less than 15 or

[2]Steinman et al. [42] have reported microsaccades of less than 3 minutes of arc. We have recorded microsaccades where the difference between the initial and final positions was less than 2 minutes of arc (unpublished data).

20 degrees should be about half the duration of the saccade. The peak velocity is almost always in the middle of these saccades. This is reasonable, because tautologically, as long as the muscles are applying accelerating forces, the velocity will increase. Only after the agonist force starts to drop from its maximum to the tonic level will the velocity decrease. Therefore, the peak velocity should be reached around the end of the controller signal pulse, which should occur about in the middle of the saccade.[3] This argument seems to be based on the time necessary to decelerate the mass of the eyeball; yet, we know that the mass of the eyeball is so small that it has a negligible effect on the dynamics of the saccade [35]. Therefore, we must look for an "apparent inertia" produced by the energy stored in the series elasticity of the muscle during the initial stretch, the apparent muscle viscosity that limits rates of change of position, the activation-deactivation time constants, and—only slightly—the inertia of the eyeball. The effect of each of these elements can be readily visualized by using the Clark-Cook-Stark model for the extraocular muscles [13, 8–10] and solving for the acceleration of the eye to yield the following equation:

$$J\ddot{\theta}_{\text{eye}} = F_{\text{ag}} - F_{\text{ant}} - K_P\theta_{\text{eye}} - B_P\dot{\theta}_{\text{eye}} - B_{\text{ag}}\dot{\theta}_{\text{ag}} - B_{\text{ant}}\dot{\theta}_{\text{ant}}$$

This equation shows explicitly each of the terms mentioned above that effect the acceleration of the eye. When working in concert, they produce the "apparent inertia" that makes the duration of the acceleration and the deceleration phases approximately equal for usual-sized saccades.

The peak velocity versus saccadic magnitude curve of Fig. 3 has an inflection point at about 15 or 20 degrees. Up until this point, as the saccades get larger, the controller signal pulse width, the number of neurons recruited, and their maximum firing frequencies increase. After this point, almost all motoneurons are firing near their maximum rates during the saccadic burst. Therefore, only the first of these three parameters can increase, so the slope of the curve decreases.

Interpretation of the data from the modeling studies of Cook and Stark [13], Thomas [44], Reinhart and Zuber [34], and Clark [7] shows that for saccades less than 15 or 20 degrees, the peak velocity does indeed coincide with the end of the controller signal pulse, which occurs in all studies long before the end of the saccade and, in most studies, about in the middle of the saccade.

It is probable, that for saccades greater than 15 degrees, the controller signal pulse width becomes larger than one-half the saccadic duration, because the resisting viscous and elastic forces of the muscles and orbital

[3]For very small saccades the duration of the controller signal may even become less than one-half the duration of the saccades.

tissues become equal to the net muscle forces, even while the forces are at their extremes. However, the controller signal duration still remains substantially less than the saccadic duration, in order to allow an appropriate time for the velocity to drop to zero at the end of movement. Reinhart and Zuber [34] have pointed out that there is a "knee" in the velocity traces obtained for very large saccades from the Cook-Stark model. This "knee" occurs after the peak velocity and shows when the controller signal terminates, giving a good indication of the pulse width. For large saccades the pulse width is between the values for saccadic duration and one-half the saccadic duration.

Early electromyographic (EMG) studies of Tamler, Marg, Jampolsky, and Nawratzki [43] indicated that the muscle fibers fire at high frequencies during the whole saccade; however, more recent studies indicate that the EMG estimate of pulse duration may be equal to one-half the saccadic duration. Bizzi, Kalil, and Tagliasco [5] showed an EMG burst that is about one-half the duration of the saccade. Collins and Scott [12], who used very fine electrodes that would sample at most four or five motoneurons simultaneously, state, "The time course of the oculomotor saccadic control signal is not constant as we had previously thought. The saccadic burst of activity is held at its constant maximum value for only the first half of the saccadic duration, thereafter falling exponentially to its new...value."

Most of these arguments are for saccades of less than 15 or 20 degrees in magnitude. This is physiologically the most important saccadic magnitude region, because most naturally occurring saccades fall in this region. Over 70 years ago Dodge and Cline [17] noted that all saccades invoked in a normal reading task are less than 13 degrees; when looking at pictures, normal "scanpath" patterns are characterized by a number of saccades similar in amplitude to the reading saccades [33]. Lancaster [28] stated that 99% of all eye movements are within 15 degrees of primary position. Many experimenters use EOG, which is most suitable for studying saccades larger than 15 or 20 degrees because of its large noise signal and limited bandwidth. The EOG signal could be highly filtered to remove the noise, but that might, of course, destroy information about the saccadic dynamics.

DISCUSSION

Modeling studies, EMG recordings, and intracellular responses of individual neurons indicate that the saccadic controller signal duration is about one-half the duration of the saccade. This was not fully realized in the past. A decade ago Robinson [35] postulated a model in which his controller signal, the active state tension, was a pulse step. The duration of his pulse, however, was the same as the duration of the saccade, as shown in

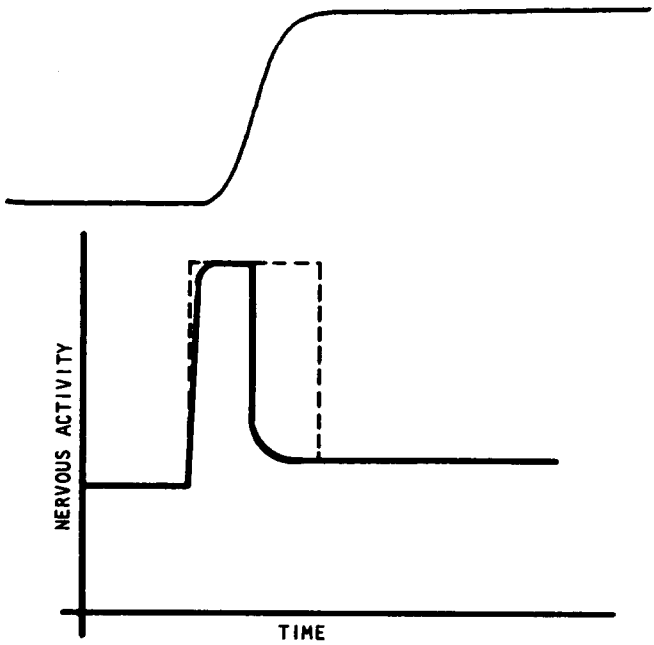

FIG. 4. A controller signal for a 10 degree saccade whose duration is one-half the saccadic duration is shown with solid lines. The controller signal shown with dashed lines is based on Robinson's Fig. 11B [35]. It starts and ends a few milliseconds before the saccade does, and therefore its duration is clearly intended to be equal to the saccadic duration.

Fig. 4. This is probably one of the reasons that his model does not produce realistic velocity records, a fact shown by Cook and Stark [13] and by Thomas [44].[4]

Luschei and Fuchs [30], in an elegant experiment, recorded from neurons associated with eye movements in the brain stems of monkeys and found that for large eye movements the data fits their expected "...ideal 1 : 1 relationship between saccadic duration and burst duration." Below saccadic durations of about 20 ms, which seem to correspond to saccadic magnitudes of about 15 degrees in their data, the burst duration is less than

[4]Robinson was able to get reasonable position versus time records from his linear, symmetrical model by neglecting the activation and deactivation time constants, lumping both the antagonist and the agonist muscles into one unit, using only the much smaller viscosity of a shortening muscle, and estimating a small value for the series elasticities.

the saccadic duration. For their 54 saccades with bursts of less than 22 ms, the ratio of burst duration to saccadic duration was 0.75. This result was unexpected, for their burst durations are longer than the controller signal pulse, a direct result of their defining the burst duration as the time between the first and last spikes. The firing frequency of neurons does not change instantaneously: it has a non-zero rise and fall time. The high frequency burst is of prime importance to the extraocular muscles. The gradual turn-on and turn-off probably adds very little to the total force developed. Thus, it is the width of the high frequency burst that is important; therefore, aberrant, straggling spikes may be disregarded. Accordingly, Barmack [3] defined the saccadic pulse duration as the interval during which the instantaneous frequency of the motoneurons exceeded 300 pitts ("pitts" stands for pulses per second [40]). We have plotted the data of Luschei and Fuchs [30, Fig. 3] in our Fig. 5. Two possible definitions are shown for the burst duration. The time between the first and last spikes t_{LF}, yields durations with a large amount of scatter, where some burst durations can even be larger than the saccadic duration. Disregarding the straggling spikes and using only the duration of the high frequency burst, t_{BCS}, produces burst durations that approach one-half the saccadic duration for saccades less than 15 degrees.

Keller [26] has also recorded from the burst units in the pontine reticular formation of alert monkeys. Although these cells do not represent the pulse

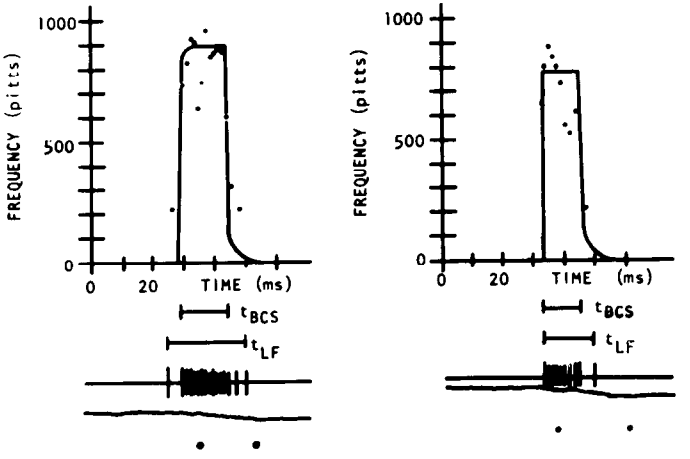

FIG. 5. Neuronal frequency (top), voltage spikes of the oculomotor neurons (middle), and eye position (bottom) versus time for monkey saccadic eye movements; based on Luschei and Fuchs [30]. The dots below the eye position records represent their estimation of the beginning and end of the saccade.

portion of the pulse step controller signal, they are closely associated with its production and may be the direct cause of the pulse. To avoid the artificial lengthening of the burst resulting from straggling spikes, his data are based on the neuron's average burst duration for ten saccades of the same size. Fuchs [19] used similar monkeys and reported that 15 degree saccades take about 30 ms. Keller (personal communication) has found that for 110 saccades with duration of 28 ms or less the ratio of burst duration to saccadic duration was 0.7. The 60 saccades with durations less than 20 ms had a ratio of burst duration to saccadic duration of 0.54. For saccades larger than 15 degrees, the burst duration was approximately equal to the saccadic duration.

Monkey saccades are different from human saccades [19], but the controller signal pulse seems to be about half the duration of the saccade for both species for saccades less than about 15 degrees.

RECIPROCAL INNERVATION AND THE ANTAGONIST

The antagonist activity is seen to circumscribe the agonist activity. The models of Cook and Stark [13], and Clark [7] clearly demonstrate that the antagonist resumes its activity after the agonist ceases its burst of activity. The fact that the antagonist motoneuron pool ceases activity before the agonist motoneuron pool begins its high frequency burst of firing has been reported for the abducens motoneurons [32, 20], the trochlear motoneurons [21, 2], the pausing and bursting units associated with saccades in the reticular formation [26], the biceps and triceps muscles [18], and the jaw muscles [11]. Thus, the duration of the antagonist inhibition is probably somewhat longer than the duration of the agonist burst, somewhere between the duration and half the duration of the saccade for saccades less than 15 degrees. Evidence of this can be found in the neurophysiological literature: Robinson [36, p. 401] commented, "During the last half of the saccade many antagonist motor units had resumed activity."

Quantitative studies which show the pause as one-half the saccadic duration are rare, because up to one interspike interval may inadvertently be added at each end to the real pause. For a firing frequency of 50 pitts, this interspike interval is 20 ms. Adding one interspike interval to each end of the true pause would make the pause seem 40 ms longer than it really is and would obscure the true pause duration.

CONCLUSION

The relationships of saccadic magnitude to saccadic duration and peak velocity have been called the *main sequence*, a term borrowed from our astronomer friends. Because the saccadic duration is about twice the con-

troller signal duration, the saccadic magnitude is a strong function of controller signal duration. This illustrates the nonlinear nature of the saccadic system, for in a linear second order system the duration of the movement is constant over the entire magnitude range.

Modeling studies, EMG recordings, and intracellular responses of individual neurons indicate that the controller signal duration for saccadic eye movements is always less than the saccadic duration. For saccades less than 15 degrees—the range which encompasses most naturally occurring saccades—the controller signal pulse width is about *one-half* the duration of the saccade. This concept should help clarify the neurophysiological literature concerning the neurological substrates underlying the generation of saccadic eye movements.

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