The High-Frequency Burst of Motoneuronal Activity Lasts About Half the Duration of Saccadic Eye Movements

A. TERRY BAHILL

AND

LAWRENCE STARK

*Department of Electrical Engineering and Computer Science and Department of Physiological Optics, University of California, Berkeley, California 94720**

ABSTRACT

Modeling studies, EMG studies, and single-unit recordings from neurons in the pontine reticular formation all seem to indicate that the pulse portion of the motoneuronal saccadic controller signal, the high-frequency burst of motoneuronal activity, should last for about half the duration of normal-sized saccades, namely, those 15 degrees or less in magnitude. This is in harmony with a postulated apparent inertia that must be decelerated during the last half of the saccadic eye movement.

INTRODUCTION

In order to produce a saccade, the extraocular muscles must receive a pulse-step controller signal as shown in Fig. 1(a). The duration of the pulse, the high-frequency burst of motoneuronal activity, lasts only half the duration of the saccade for saccades 15 degrees and smaller. Approximately 86 percent of all naturally occurring human saccades are 15 degrees or smaller [1]. Human electromyographic (EMG) studies [5, 6] and modeling studies [2, 7, 11, 15] all show the pulse duration to be about one-half the saccadic duration for normal-sized saccades. Neurophysiological data taken from alert, behaving monkeys show that the high-frequency burst of neuronal firing is definitely less than the saccadic duration and approaches one-half the duration of the saccade. There are three such pools of data available for neurons located in the pontine reticular formation (PRF) and associated with saccadic eye movements: Luschei and Fuchs' study [10],

*Address correspondence to: Terry Bahill, 226 Minor Hall, University of California, Berkeley, CA 94720.

Keller's study [9], and the present one.

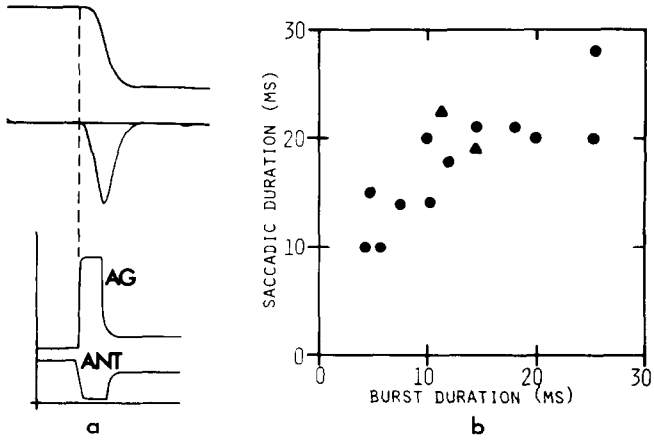

FIG. 1. (a) Shown from top to bottom, as functions of time: eye position, eye velocity, neuronal activity of agonist motoneuronal pool, and motoneuronal activity of antagonist motoneuronal pool. (b) Saccadic duration versus PRF burst-unit duration. The ratio of burst duration to saccadic duration was 0.56 for the 8 smallest saccades. The triangles are individual saccades from another data pool, as explained in the text.

RESULTS

In Fig. 1 we show unpublished additional data from the previously reported experiments of Keller [9]. There is no significant difference in the neuronal response between the fast phase of vestibular nystagmus and saccadic eye movements; they are pooled together in this figure. The data show that for 14 saccades with burst durations of 35 ms or less, the ratio of burst duration to saccadic duration was 0.73 with a standard deviation of 0.25. The 8 saccades with burst durations less than 18 ms had a ratio of burst duration to saccadic duration of 0.56 with a standard deviation of 0.15. Comparatively, the ratio of burst duration to saccadic duration was 0.75 for Luschei and Fuchs' 54 saccades with bursts of less than 22 ms. The two points indicated by triangles in Figure 1(b) were derived by Bahill, Clark, and Stark [3] from the data of Luschei and Fuchs [10]. All of these data which show the burst duration to be 50 to 75 percent of the duration of the saccade are significantly different than the data for large saccades. Our data for 39 saccades with burst durations greater than 30 ms were repetitious of Luschei and Fuchs' data. Linear regression analysis of the data indicated that the ratio of burst duration to saccadic duration approached unity.

Thus, this neurophysiological data from monkeys corroborates the behavioral data and EMG studies from man: the high-frequency burst of neuronal activity approaches one-half the duration of the saccade for saccades 15 degrees and smaller, and the burst is therefore much shorter than the saccade itself.

DISCUSSION

Because this controller signal pulse, the motoneuronal burst, ends near the middle of the saccade, the maximum velocity of the saccade should also occur near the middle of the saccade. Human eye movement records for normal-sized saccades show this to be true [Fig. 1(a)]. 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. The time necessary to decelerate the eye is not due to the mass of the eyeball, because this mass is so small that it has a negligible effect on the dynamics of the saccade [12]. Therefore, the "apparent inertia" is produced by the energy stored in the series elasticity of the muscles during the initial stretch, the apparent muscle viscosity that limits rates of change of position, the activation and deactivation time constants of the neurons and muscle fibers, and—only slightly—the inertia of the eyeball.

The curve of peak velocity versus saccadic magnitude has an inflection point at about 15 degrees [3]. Up until this point, as the saccades get larger, the controller signal pulse width, the number of neurons recruited, and their maximum firing frequencies may all 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. 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 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 the movement.

LIMITATIONS

It cannot be proven that these PRF units produce the controller signal pulse; however, they do seem to be very closely associated with its production. There are known direct connections from this area to the oculomotor neurons [13]. An important difficulty in relating the burst of these units to

the controller signal pulse is how to define the burst duration. The firing frequency of neurons does not change instantaneously: it has a nonzero rise and fall time. The gradual turn-on and turn-off only slightly modifies the total force developed, whereas the high-frequency burst is of prime importance in the control of the extraocular muscles. Accordingly, Barmack [4] 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 [14]). Luschei and Fuchs [10] defined the burst duration as the time between the first and last neuronal spikes, but this allows the burst duration to be larger than the saccadic duration in some instances.

Care must also be taken with the definitions of saccadic duration and saccadic magnitude, especially for saccades with overshoot. Bahill, Clark, and Stark [2, 3] have found it most expedient to define *duration* as the time between zero velocity at the start of the saccade and the first zero velocity at the end, and *magnitude* as the angle between the initial position and the largest excursion during the overshoot phase. With these definitions the burst durations will exceed half the duration of the saccades with overshoot. We are aware of other problems in trying to relate data from single unit recordings in the PRF to the saccadic controller signal. There is apprehension that single unit recordings may not be representative of the neuronal population being studied, because of their sampling nature. Furthermore, uncertainty in comparing data between man and monkey exists. There is also the confounding effect that it is easiest to study large saccades in laboratory monkeys, while most human saccades have magnitudes of 15 degrees or less. The PRF burst-unit durations are approximately equal to the saccadic duration for large saccades. Finally, the variability in saccadic amplitude-duration relationships has not been systematically studied. The calibration data from Luschei and Fuchs indicate that 15 degree saccades had saccadic durations of about 20 ms, while Fuchs [8] used similar monkeys and reported that 15 degree saccades take about 30 ms.

In conclusion, the neurophysiological data presented here show that for saccades less than 15 degrees the high-frequency burst of neural activity is much shorter than the saccadic duration, approaching one-half the saccadic duration.

We thank Dr. Edward L. Keller for allowing us to use his data and for his helpful discussions. We note, however, that he does not necessarily agree with all aspects of this paper.

REFERENCES

1 A. T. Bahill, D. Adler, and L. Stark, Most naturally occurring human saccades have magnitudes of 15 degrees or less, *Invest. Opthalmol.* **14** (6), 468–469 (1975).

2 A. T. Bahill, M. R. Clark, and L. Stark, Dynamic overshoot in the saccadic eye

movements is caused by neurological control signal reversals. *Exp. Neurol.*, **48**, 107–122 (1975).

3 A. T. Bahill, M. R. Clark, and L. Stark, The main sequence, a tool for studying human eye movements. *Math. Biosci.*, **24**, 191–204 (1975).

4 N. H. Barmack, Saccadic discharges evoked by intracellular stimulation of extraocular motoneurons, *J. Neurophysiol.* **37** (3), 395–412 (1974).

5 C. C. Collins, The human oculomotor control system, in *Basic Mechanisms of Ocular Motility and Their Clinical Implications*, (G. Lennerstrand and P. Bach-Y-Rita, Eds.), Pergamon, New York, 1975.

6 C. C. Collins and A. B. Scott, The eye movement control signal, in Proceedings of the Second Bioengineering Conference, Milan, Italy, 1973.

7 G. Cook and L. Stark, Dynamic behavior of human eye-positioning mechanism, *Commun. Behav. Biol.*, Part A, **1** (3), 197–204 (1968).

8 A. F. Fuchs, Saccadic and smooth pursuit eye movements in the monkey, *J. Physiol.* **191**, 609–631 (1967).

9 E. L. Keller, Participation of medial pontine reticular formation in eye movement generation in monkey, *J. Neurophysiol.* **37** (2), 316–332 (1974).

10 E. S. Luschei and A. F. Fuchs, Activity of brain stem neurons during eye movements of alert monkeys, *J. Neurophysiol.* **35** (4), 445–461 (1972).

11 R. J. Reinhart and B. L. Zuber, Parameters of the control signals for saccadic eye movement: electrical stimulation and modeling, *Exp. Neurol.* **30**, 148–161 (1971).

12 D. A. Robinson, The mechanics of human saccadic eye movement, *J. Physiol.* **174**, 245–264 (1964).

13 M. E. Scheibel and A. B. Scheibel, Structural substrates for integrative patterns in the brain stem reticular core, in *Reticular Formation of the Brain* (H. H. Jasper, Ed.), Little, Brown, Boston, 1958.

14 L. Stark, Proposed new unit of frequency, *Nature*, **225**, 394–395 (1970).

15 J. G. Thomas, The dynamics of small saccadic eye movements, *J. Physiol.* **200**, 109–127 (1969).