# Dynamic Overshoot in Saccadic Eye Movements Is Caused by Neurological Control Signal Reversals

A. Terry Bahill, Michael R. Clark, and Lawrence Stark [1]

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

Three quite different types of overshoot occur in saccadic eye movements; each has unique characteristics determined by distinct neuronal control patterns. Most saccades have dynamic overshoot; it is more prevalent among, and more prominent in, small saccades. Dynamic overshoot is caused by nonrandom reversals of the neuronal control signals. It is a monocular phenomenon. The return velocities for dynamic overshoot are equal to saccadic velocities and are much larger than vergence velocities.

## INTRODUCTION

Saccades are the fast, staccato eye movements characteristically displayed by people who are reading or looking about a scene. They are the fastest of the eye movements. The eye movement system is ideal for studying neurological control, because the small constant mass of the eyeball and the high speed of the muscles make the output plant characteristics transparent and the underlying neural control signals can easily be discerned. In this paper we discuss the neurological control signals which produce variations in the shapes of normal saccades, executed by normal subjects.

To generate a saccadic eye movement, a neural control signal in the form of a pulse step (Fig. 1D) is sent to the extraocular muscles (1, 11, 16, 31). Figure 1 shows saccades with dynamic overshoot, dynamic undershoot and neither. Inferences can be made from these about the variations which come at the end of the pulse portion of the neurophysiological controller signal and also subsume the variability in the saccadic trajectory.

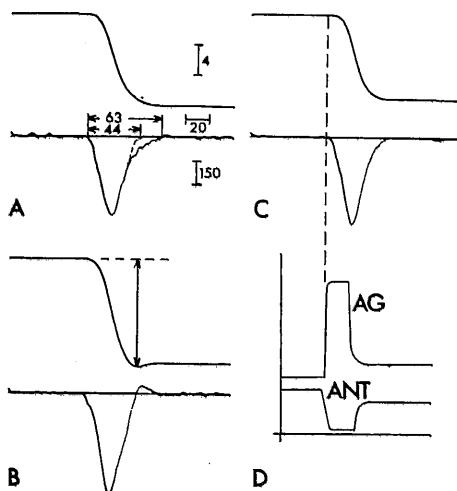The eye has overshoot when it travels beyond its final position and then

FIG. 1. Position as a function of time (top), and velocity as a function of time (bottom) for saccades with dynamic undershoot (A), dynamic overshoot (B), and neither (C); D, shows presumed motoneural activity of agonist (top) and antagonist (bottom) as functions of time. Calibrations shown in A represent 4 deg, 20 msec, and 150 deg/sec and also apply to B and C. No time delay is shown between the start of the controller signal (D) and the beginning of the saccade (C), because this delay depends upon where the controller signal is measured. A, shows the difference in duration of ideal and undershooting saccades. B, shows the definition of saccadic magnitude used for saccades with dynamic overshoot.

returns in the opposite direction. In the literature the term overshoot is used to describe three quite different types of saccadic behavior: *first,* dynamic overshoot with large return velocities on the order of 10–100 deg/sec (12, 15, 31, 33, 36, 38, 40, 42, 45); *second,* slow drifting eye movements, glissades, with return velocities of 2–20 deg/sec caused by a mismatch between the pulse and step components of the saccadic controller signal (13, 30, 32, 39, 41); and *third,* static overshoot, a common clinically observed sign in which error conditions are corrected by a secondary saccadic eye movement about 200 msec after the primordial saccade (4, 24, 41). Only static overshoot can be seen easily with the naked eye, so it is the most frequent type of overshoot described by clinicians.

This paper is concerned with dynamic overshoot, the type exhibited by the saccade of Fig. 1B, and illustrates how dynamic overshoot is caused by reversals in the neuronal controller signal.

## METHODS

A wide variety of visual targets were employed. The most common were presented on a semicircular screen 46 cm in front of the subject. The target, for saccades 25 deg and smaller, was a small spot of white light 1 mm

in diameter which was emitted from a slide projector and reflected off a mirror galvanometer. Larger eye movements used pieces of white tape for targets. The subject's head was supported with a head rest and a bite bar covered with dental impression compound.

The entire eye was diffusely illuminated with infrared light obtained by mounting a Kodak Wratten filter no. 87 on a low voltage lamp. The lamp was powered by a battery to eliminate 60 Hz noise. Infrared light was used in order to avoid distracting the subject.

Our method for measuring eye movements employs a pair of photodiodes (Texas Instrument, LS400) aimed at the iris–sclera border, one on each side of the iris, as indicated in Fig. 2. The photodiodes are incorporated into a bridge circuit (Fig. 3) where the photocurrents are converted into voltages (35). These voltages are differentially amplified with d-c amplifiers, and recorded on a f-m tape recorder or a computer disk memory unit. When the eye turns toward the nose, the nasal photodiode will be exposed to more of the dark iris and less of the white sclera: therefore, its photocurrent will decrease. Simultaneously, the photocurrent of the temporal photodiode will increase, due to its greater exposure to the white sclera. The difference of the two photocurrents is a measure of the eye position. The differential nature of the system eliminates noise common to both photodiodes. Although it seems at first glance that the polarity of the battery *and* photodiodes could be reversed, this is not always the case. The
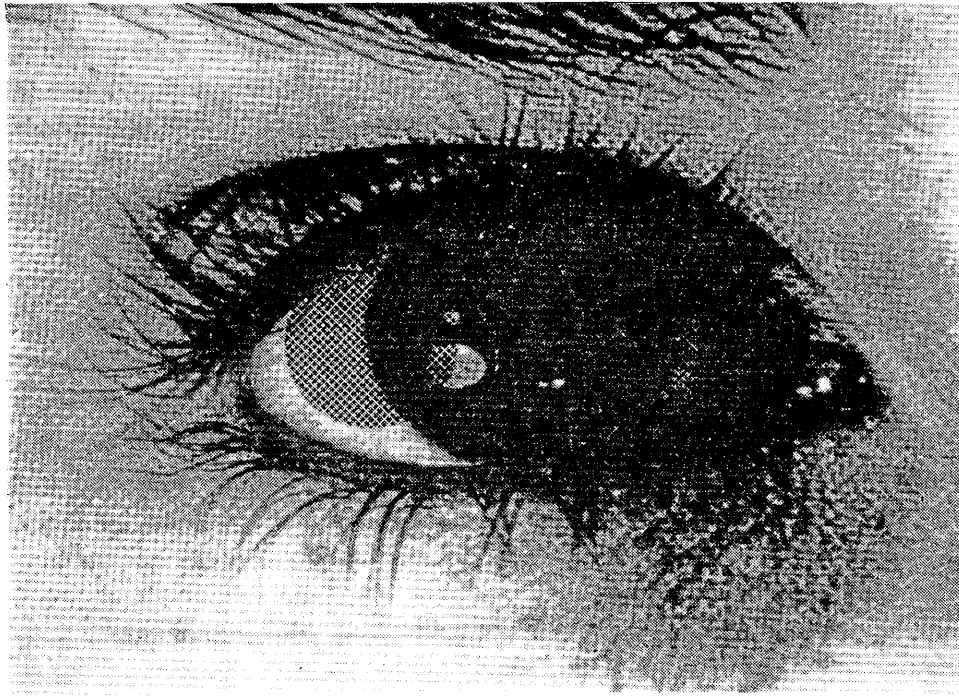


FIG. 2. To measure horizontal eye movements, photodiodes are aimed to receive light from stippled areas.
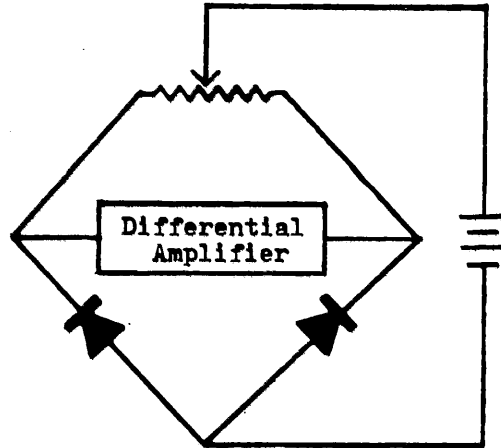
Fɪɢ. 3. Bridge circuit for converting photocurrent into voltage and differentially amplifying this voltage. The potentiometer is 500 kohm; the battery is 12 v.

positive terminal of the battery should be connected to the potentiometer, as shown in Fig. 3, so that the amplifier input bias current can be supplied through the potentiometer, since the photodiode dark current may not be sufficient. The vertical components of the eye movements were measured by adding a second pair of photodiodes aimed at the lower limbus.

*Linearity.* A calibration check was made at the beginning and end of each of our 5 min experimental runs. The results of a typical session (Fig. 4) clearly demonstrate that the system is linear for more than 20 deg of eye rotation. This limit on linearity is prescribed by the size of the iris and the covering of the iris by the eyelids. The output of this exceptionally simple instrument is nonlinear for very large eye movements. We do not wish to add more photodiodes, or a computer linearization program to the instrument to compensate for this nonlinearity, because the present instrument compresses the extremes of the range and expands the central region, producing linearity and a signal to noise ratio greater than 1,000 near the center, where the saccades achieve peak velocity. Yet, it still allows measurement of saccades up to 50 deg, which includes most naturally occurring human saccades. For very large saccades the dynamic overshoot occurred in this nonlinear region; the velocity computations were correspondingly adjusted.

Definitions of peak velocity, saccadic magnitude and duration are clear from the figures and are elaborated upon by Bahill, Clark, and Stark (1). Noise and drift of the instrumentation were less than 1 mv and were, therefore, smaller than the signals produced by eye movements of one minute of arc, which were on the order of 30 mv.

The bandwidth of the complete system was measured by exciting the photodiodes with a light emitting diode and was found to be in excess of 1 kHz under all circumstances. This large bandwidth was necessary for observing dynamic overshoot. Fourier analyses of saccadic eye movements have shown that the amplitude of the Fourier components decreased with frequency. Zuber, Semmlow and Stark (43) showed that the mangitude of the 50 Hz component of a critically damped saccade was 10% of the magnitude of the low frequency components. Thomas (39), using a saccade with dynamic overshoot, computed the magnitude of the 80 Hz component to be 45% of the magnitude of the low frequency components. Therefore, the basic shape of the saccade is delineated by the low frequency components, but the dynamic overshoot is dominated by the higher frequency components. To illustrate the effect of eliminating the high frequency components of a saccade, the position and velocity records are shown in Fig. 5 for a saccade that has been low-pass filtered (Kron-Hite model 3750) at 1,000, 80, 40, and 20 Hz. Low-pass filtering made the peak velocity seem much smaller, and obscured even the large amount of dynamic overshoot exhibited by this saccade. It was difficult, but not impossible, to observe dynamic overshoot using Electro-oculography (EOG), because low-pass
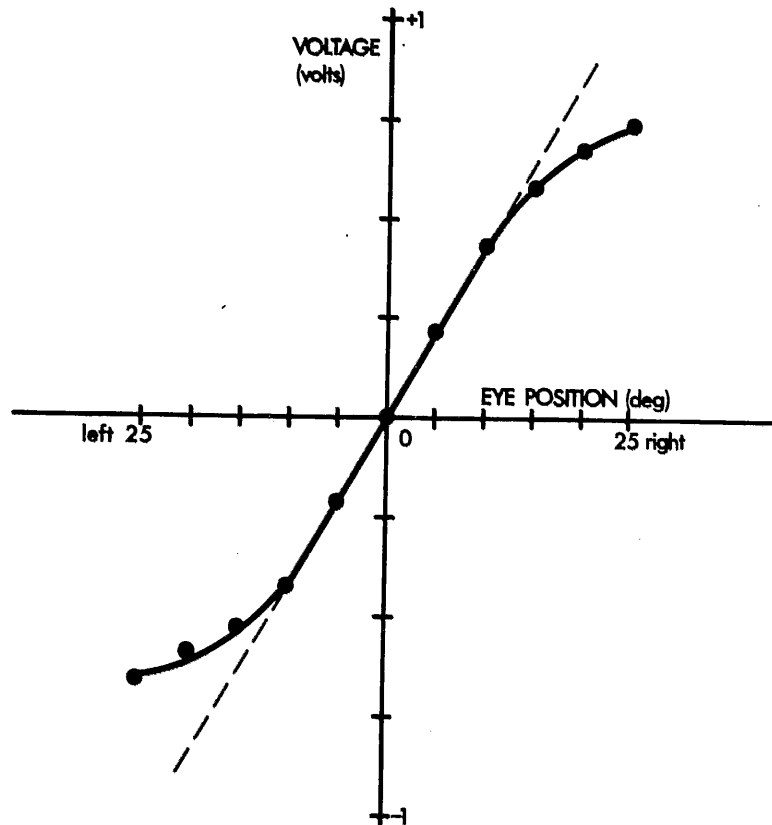


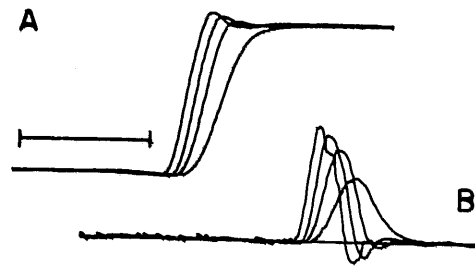Fig. 4. Typical output voltage vs eye position relationship.

Fig. 5. Effect of low-pass filtering data. A, position as a function of time for a 5.6 deg saccade passed through a low-pass filter with a cutoff frequency of (from top to bottom) 1000, 80, 40, and 20 Hz. B, velocity as a function of time for this same saccade and filter parameters. Peak velocity of this saccade is 300 deg/sec. Note the irregularities often seen with minimal low-pass filtering. The time calibration represents 100 msec.

filtering removed the overshoot; and without low-pass filtering, the noise inherent in the EOG technique tended to obscure the overshoot.

A small digital computer was used to gather the saccadic eye movement responses and generate the velocity records. A computerized slow down routine was employed for plotting the saccadic responses. This enabled us to maintain the large bandwidth that would not be available with conventional recorders operated in a real time mode.

Preliminary data were taken from about 160 subjects; extensive data were taken and analyzed in detail for seven normal subjects.

To obtain quantitative records to compare with the physiological data, simulations of the model were performed on a digital computer coupled to an IBM 2250 interactive graphics unit with a CRT display.

## RESULTS

The existence of dynamic overshoot is quite capricious. On one day most of a subject's saccades will have dynamic overshoot, and on another day very few will. In one record of uniform sized saccades we found consecutively: six saccades with dynamic overshoot, ten without dynamic overshoot, one with, three without, 15 with, and 13 without. Dynamic overshoot may simultaneously be present in one eye and absent in the other. We have recorded both microsaccades and 50 deg saccades both with and without dynamic overshoot. A few of these dynamically overshooting saccades are shown in Fig. 6. Care was taken to insure that the recording devices were not saturated by the eye movements, for if any part of the recording system saturates, no overshoot will be seen. It may not be obvious that the device has saturated, however, because it may be a soft saturation, which will still permit the records to look smooth and normal.

A detailed analysis of over 3,000 saccades executed by seven subjects revealed that about 70% of the saccades had dynamic overshoot and 5%
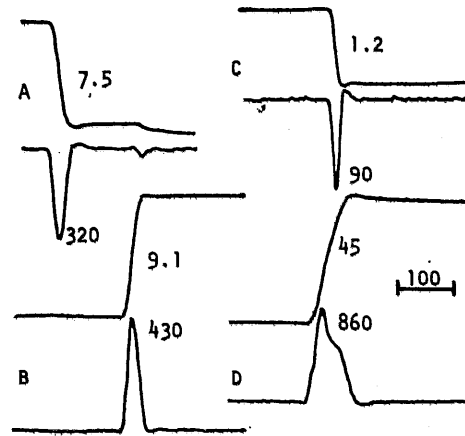
FIG. 6. Position (top) and velocity (bottom) records for several saccades with dynamic overshoot. Saccades of A and B were recorded with the eye abducted 35 deg from primary position. Saccades of C and D were recorded with the eye moving through primary position. Right, nasal, is upward in these records.

had dynamic undershoot. These percentages varied from day to day, from subject to subject, and with the size of the saccade. The percentages were influenced by fatigue; as the subject fatigued, undershoot became more prevalent (2).

Small saccades are more likely to have dynamic overshoot than large saccades (Fig. 7). Dynamic overshoot is also more prominent in small saccades. Figure 8 shows the size of dynamic overshoot for 457 saccades. The size of the overshoot gradually increases with saccadic magnitude, until it levels off at about 1 deg for saccades 30 deg in amplitude. The percentage of overshoot is defined as the maximum saccadic deviation minus the final eye position, all divided by the final eye position, or

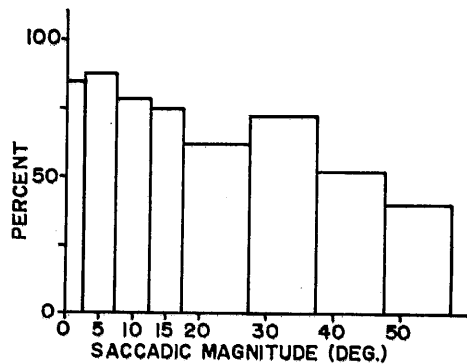$$\text{percentage of overshoot} = \frac{\theta_{max} - \theta_{final}}{\theta_{final}} \times 100.$$



FIG. 7. The percentage of saccades that have dynamic overshoot as a function of saccadic magnitude.
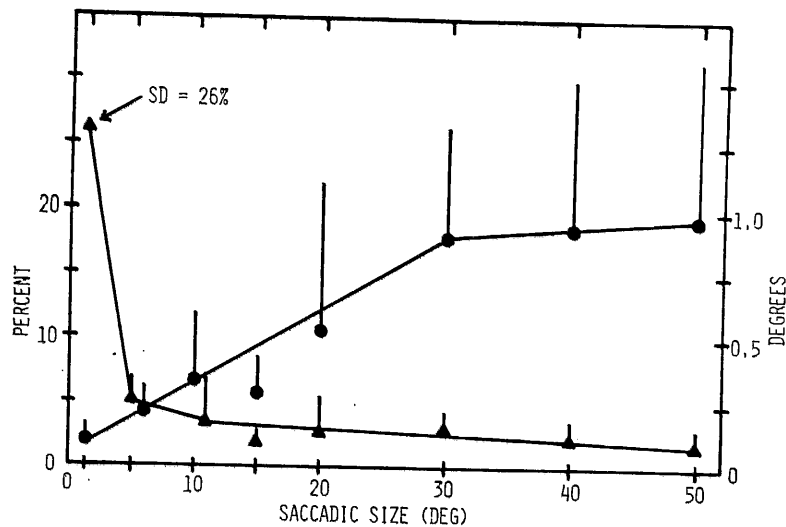
Fig. 8. Average size of dynamic overshoot in degrees (●) and in percentage of overshoot (▲) both as functions of saccadic size. The bars represent one standard deviation.

This percentage of overshoot is greatest for small saccades. Sometimes it exceeds 100%. It is about 2 or 3% for all large saccades.

The use of standard deviation (Fig. 8) is not meant to imply that the size of the dynamic overshoot is normally distributed. It is not, primarily, because of the rare saccades with excessively large values of dynamic overshoot. Figure 9 is a frequency distribution histogram for the size of dynamic overshoot for 54 saccades with magnitudes between 9 and 11 deg. The mean size of the dynamic overshoot for these saccades is 0.34 deg, and the standard deviation is 0.27 deg. If the one saccade with excessive dynamic overshoot is eliminated, then the mean and standard deviation become 0.31 and 0.11 respectively. Without this one saccade the new distribution is approximately Gaussian.

In an overshooting saccade, the eye passes its final position and must return in the opposite direction. The maximum velocity of this return phase has been plotted (Fig. 10) along with the maximum velocities for
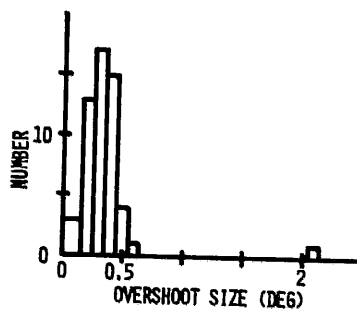


ιG. 9. Frequency distribution histogram for the size of the dynamic overshoot of deg saccades.
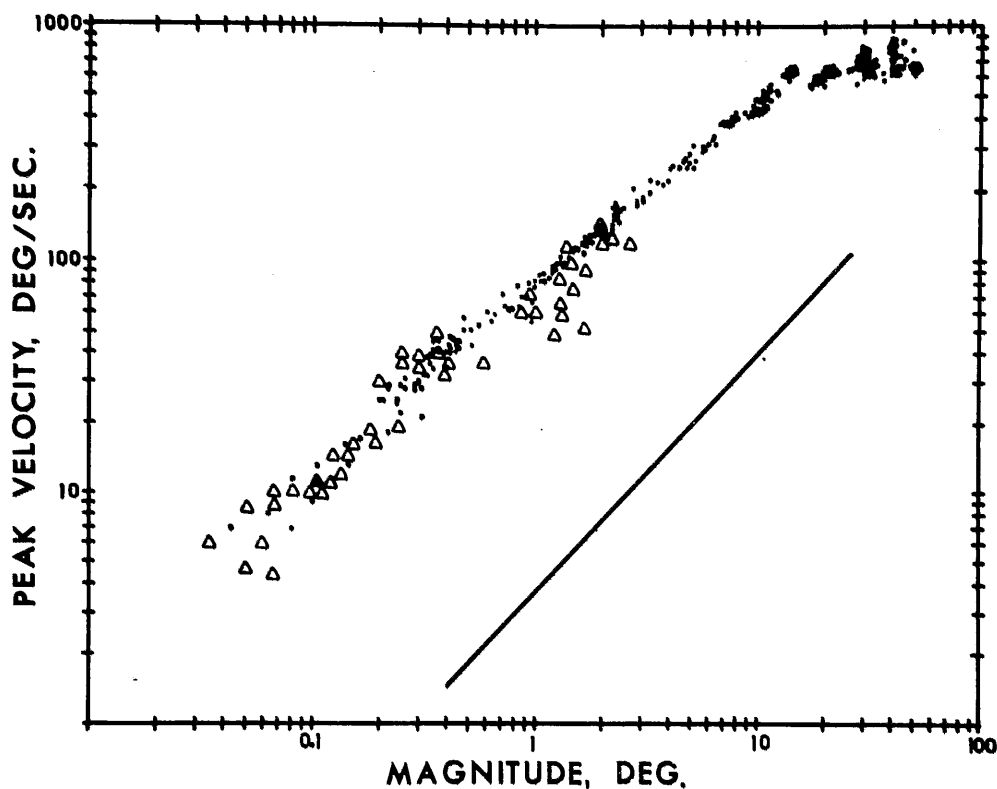
Fig. 10. Peak velocity versus magnitude for saccades (●), the return phase of dynamic overshoot (▲), and fusional convergence (–).

saccades (from Ref. 1), and fusional convergence eye movements (from Refs. 27, 29, 44). It can be seen that the return velocities are large: They are equal, or nearly equal to the velocities of saccades of the same small amplitude.

To investigate the cause of dynamic overshoot, the Clark-Cook-Stark model of the extraocular plant was simulated (6, 11). The extraocular plant—the extraocular muscles, the eyeball, and the surrounding tissues— is overdamped (11, 31); therefore, the underdamped nature of the eye response for saccades with dynamic overshoot is of neural origin. Figure 11 shows, from top to bottom: the position, the velocity, the simulated active state tension of the agonist muscle, the simulated active state tension of the antagonist muscle, and the presumed motoneuronal signals required for the agonist and antagonist in order to produce such responses. The nervous activity reverses at the end of the main pulse. For instance, the firing rate of the pool of motoneurons supplying the agonist muscle rises from the original tonic level to the high-frequency level, remains there until about the middle of the saccade, drops to a very low value, and then rises again up to its new tonic level. The pool of motoneurons innervating the antagonist muscle perform an analogous, but opposite task. Due to this moto-

neuronal innervation, the active state tensions of the muscles reverse. The active state tension of the agonist first becomes larger, then smaller, then equal to all of its opposing forces. These active state tensions are not the muscle forces measured at the tendon of a muscle (21). The changes in force at the tendon will be slower than the changes in active state tension shown in this figure.

All of the previous data were for horizontal saccadic eye movements. However, dynamic overshoot also occurs in vertical and oblique saccadic eye movements. It is less frequent in the vertical channel. Purely vertical saccadic eye movements had dynamic overshoot for 36% of the upward, and 15% of the downward movements. Figure 12B shows an oblique saccadic eye movement that has dynamic overshoot in both the horizontal and the vertical components, and Fig. 12A shows an oblique saccadic eye movement with dynamic overshoot only in the horizontal component.
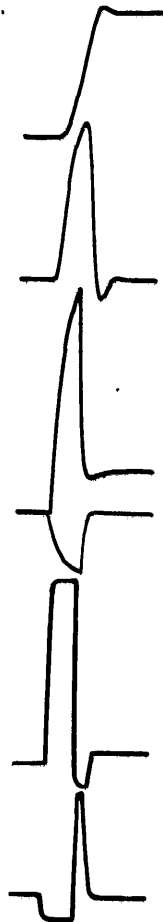


FIG. 11. Model responses for a saccade with dynamic overshoot, (from top to bottom) as functions of time, position, velocity, active state tension of agonist muscle, active state tension of antagonist muscle, agonist motoneuronal activity, and antagonist motoneuronal activity.
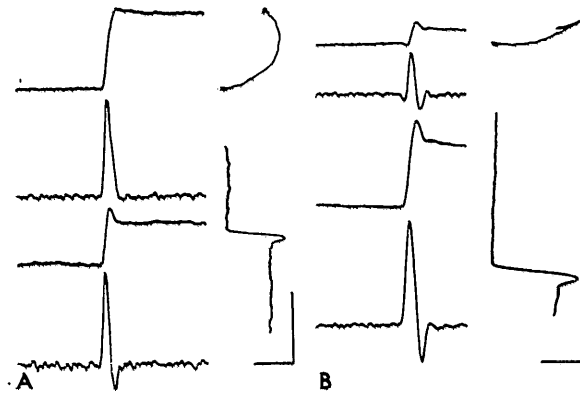
FIG. 12. Oblique saccadic eye movements with dynamic overshoot in both the horizontal and vertical components (B), and with dynamic overshoot in only the horizontal component (A). In the left column, from top to bottom, are shown; vertical eye position, vertical eye velocity, horizontal eye position, and horizontal eye velocity, all as functions of time. The right column shows (top) the eye position in space, or the X-Y trajectory, and (bottom) the horizontal position record rotated 90 deg and aligned with the X-Y trajectory above. The calibrations represent 4 deg, 150 deg/sec, and 100 msec. In the left column rightward (temporal) and upward eye movements are represented by upward deflections.

No systematic differences in dynamic overshoot were found when the saccades were: accurate or inaccurate, temporally or nasally directed; to or from primary position; at a rate of 0.3 or 3 saccades/sec; in primary position or 35 deg abducted; in light or darkness; of short or long latency; tracking a target or shifting the position of regard; executed by myopic or nonmyopic subjects; executed by subjects with or without corrective lenses; executed by subjects wearing or not wearing contact lenses; for predictable tracking; and for red, white or green targets.

Dynamic overshoot is a monocular phenomenon. That is, the size of the overshoot is usually different in the two eyes. In fact, often one eye has dynamic overshoot and the other has dynamic undershoot.

## DISCUSSION

The return velocities of dynamic overshoot fit on the Main Sequence diagrams for saccadic eye movements (Fig. 10). However, occasionally the peak velocity of the return phase of the dynamic overshoot is slightly smaller than for equivalent sized saccades. This may be due to the unusual initial conditions imposed upon the dynamic overshoot. For example, at the commencement of the primary saccade the initial eye velocity is zero, and the antagonist motoneurons are switched from a nominal firing rate to zero; whereas, at the start of the return phase of the dynamic overshoot the eye velocity is nonzero, and one of the motoneuronal pools must be switched from its maximum firing rate to zero (Fig. 11).

The antagonist activity (Figs. 1D and 11) circumscribes the agonist activity. Modelling studies (11) have predicted, and electromygraphic (EMG) studies (9, 10) have confirmed that the anatagonist resumes its activity after the agonist ceases its burst of activity. It has been reported that the pause of the antagonist motoneuron pool starts before the agonist motoneuron pool begins its high-frequency burst of firing in the abducens motoneurons (16, 25), the trochlear motoneurons (3, 17), the pausing and bursting units associated with saccades in the reticular formation (22), the arm muscles (14, 23), and the jaw muscles (8).

*Teleology of Dynamic Overshoot.* This model for the extraocular plant plant and saccadic controller suggests that dynamic overshoot must be caused by neural control signal reversals. These control signal reversals cannot be due to random noise, however, because often as many as a dozen consecutive saccades will have identical overshoot. The reversal at the end of this controller signal is similar to the signal required for time optimal control of an inertial object (37, p. 631), and also the signal used in optimal responses when the forearm moves a load (34). Clark and Stark (7) averaged the responses of many saccades, thereby smoothing out the dynamic overshoot, and suggested that the first order time optimal controller signal was used by the saccadic system. It can now be seen, however, that most saccadic eye movements have dynamic overshoot. The neural control signal reversal which underlies the dynamic overshoot is precisely that second order time optimal controller signal predicted by the evaluation of the Hamiltonian functional (7). This controller signal stratagem enables the eye to arrive and stay within one central foveal radius (0.5 deg) of the target in the least possible time.

It is unlikely that dynamic overshoot is a corrective saccade produced by sensory feedback, because small saccades, as shown in Fig. 6C, have durations of 15 to 20 msec, and if the sensory system sampled the eye position near the middle of the saccade, there would be only ten msec between sensory stimuli and motor response. Normal reflexes are not this fast. For example, the H-reflex, which does not include a sensory organ and is, therefore, only a portion of a reflex arc, has a 30 msec latency (18).

Dynamic overshoot cannot be produced merely by a mismatch in the size of the pulse and step components of the controller signal, for then the return phase would be powered by the difference in the two tonic levels of innervation of the agonist and antagonist muscles. This would produce velocities similar to vergence velocities (2, 13). This is clearly not the case for dynamic overshoot, because as shown in Fig. 10, the return velocities equal saccadic velocities and are much larger than vergence velocities.

It has been hypothecated (Merton Flom, personal communication) that the CNS may first command a saccade which only approximates the desired magnitude. Then, based upon refined analysis of both the retinal

position error and the size of the impending saccade, it produces a second saccade—the return phase of dynamic overshoot. This hypothesis, however, does not explain either the preponderance of saccades with dynamic overshoot (70%), or the paucity of saccades with dynamic undershoot (5%), and it also requires a violation of Hering's law in order to produce monocular dynamic overshoot.

The center of rotation of the eye is not stationary during saccadic eye movements. Its movement could affect the shape of the measured saccade (Meredith Morgan, personal communication). For identical eye movements, however, the center of rotation follows the same trajectory. Whereas saccadic eye movements between two points show tremendous variability—one may have dynamic overshoot, and the next, dynamic undershoot. Therefore, movement of the center of rotation of the eye is not likely to be the primary cause of dynamic overshoot.

*Affect of Dynamic Overshoot on Motoneuronal Firing.* The phenomenon of dynamic overshoot should be reflected in the motoneuron firing patterns. Perhaps some of the apparently random pauses and straggling spikes noted in single cell recordings of oculomotoneurons are actually manifestations of this control signal reversal. The Main Sequence velocities of dynamic overshoot (Fig. 10) require great synchronism between the motor nuclei. For example, for a typical 10 deg saccade with dynamic overshoot most of the motoneurons in each nucleus must either pause (for the primary saccade agonist) or burst (for the primary saccade antagonist) during the same 5 to 10 msec interval. Typical oculomotoneurons have tonic frequencies on the order of 100 pulses/sec, and reach frequencies of about 500 pulses/sec during the saccadic burst (16, 33). Therefore, for the return phase of dynamic overshoot, the primary saccade antagonist motoneurons should have bursts containing about two to four spikes, and the primary saccade agonists should drop out about one spike.

It is feasible that dynamic overshoot, being a monocular phenomenon, is caused by membrane properties of the neurons. For instance, many neurons will fire a burst of spikes when released from inhibition (19, 26, 28) : A phenomenon called postinhibitory rebound or anodal break bursting. The primary saccade antagonist motoneurons are inhibited during the agonist saccadic burst. If these neurons exhibited postinhibitory rebound firing, then they would produce the short burst of spikes required for generating the return phase of dynamic overshoot. Depending upon the neuronal level at which reciprocal innervation is implemented, this burst of spikes could be sufficient to produce the pause in the neuronal firing of the primary saccade agonist which occurs immediately before it resumes its tonic activity. Alternatively, this pause could also be due to membrane phenomenon, such as summation of after hyperpolarization (5, 20). If the return phase of dynamic overshoot is produced by membrane phenomenon, as described

above, then the CNS must know in advance when dynamic overshoot will or will not occur, so that it can make the primary saccade appropriately larger. This will insure that the eye will be on target even after the return phase of the dynamic overshoot.

*Dynamic Undershoot.* Although this paper has dealt primarily with dynamic overshoot, dynamic undershoot has also been illustrated. Dynamic undershoot would also be caused by oscillations at the end of the controller signal pulse. The initial pulse would be too small and would be followed by a second very short burst of motoneuronal activity. However, there would be no role reversal between the agonist and antagonist, for the two pulses of activity would drive the eye in the same direction.

There are three distinct types of overshoot in saccadic eye movements— dynamic, glissadic, and static. The first, dynamic overshoot, is generated by reversals in the neuronal control signals sent to the extraocular muscles. The second, glissadic, is caused by a mismatch of the pulse and step components of the saccadic controller signal. The third, static overshoot, is produced by an incorrect saccadic magnitude which is feedback visually so that the consequent error is reduced by a corrective saccade. Dynamic and glissadic overshoot are open loop phenomena, whereas, static overshoot is corrected by a closed loop system.

## REFERENCES

1. BAHILL, A. T., M. R. CLARK, and L. STARK. 1975. The main sequence, a tool for studying eye movements. *Math. Biosci.* (In Press).
2. BAHILL, A. T., and L. STARK. 1975. Overlapping saccades and glissades are produced by fatigue in the saccadic eye movement system. *Exp. Neurol.* **48**: 95–106.
3. BAKER, R., and A. BERTHOZ. 1974. Organization of vestibular nystagmus in oblique oculomotor system. *J. Neurophysiol.* **37**: 195–217.
4. BECKER, W. 1972. The control of eye movements in the saccadic system. *Bibl. Ophthal.* **82**: 233–243.
5. CALVIN, W. H., and P. C. SCHWINDT. 1972. Steps in production of motoneuron spikes during rhythmic firing. *J. Neurophysiol.* **35**: 297–310.
6. CLARK, M. R., and L. STARK. 1974. Control of human eye movements. *Math. Biosci.* **20**: 191–265.
7. CLARK, M. R., and L. STARK. 1975. Time optimal behavior of human saccadic eye movements. *IEEE Trans. Auto. Control,* **AC20**, in press.
8. CLARK, R. W., and E. S. LUSCHEI. 1974. Short latency jaw movement produced by low intensity intracortical microstimulation of the precentral face area in monkeys. *Brain Res.* **70**: 144–147.
9. COLLINS, C. C. 1975. The human oculomotor control system. *In* "Basic Mechanisms of Ocular Motility and Their Clinical Implications." Pergamon Press, Ltd., New York. (In Press.)
10. COLLINS, C. C., and A. B. SCOTT. 1973. The eye movement control signal. Proceedings of the Second Bioengineering Conference, Milan, Italy, November 1973.
11. COOK, G., and L. STARK. 1968. Dynamic behavior of human eye-positioning mechanism. *Commun. Behav. Biol.* Part A, **1**: 197–204.

12. DODGE, R. 1907. An experimental study of visual fixation. *Psy Rev.* mono. supp. Vol. **8**, No. 4, whole No. 35: 1–95.

13. EASTER, S. S. 1973. A comment on the "glissade." *Vision Res.* **13**: 881–882.

14. EVARTS, E. V. 1974. Precentral and postcentral cortical activity in association with visual triggered movement. *J. Neurophysiol.* **37**: 373–381.

15. FUCHS, A. F. 1967. Saccadic and smooth pursuit movements in the monkey. *J. Physiol.* **191**: 609–631.

16. FUCHS, A. F., and E. S. LUSCHEL. 1970. Firing patterns of abducens neurons of alert monkeys in relationship to horizontal eye movement. *J. Neurophysiol.* **33**: 382–392.

17. FUCHS, A. F., and E. S. LUSCHEI. 1971. Development of isometric tension in simian extraocular muscle. *J. Physiol.* **219**: 155–166.

18. GOTTLIEB, G. L., G. C. AGARWAL, and L. STARK. 1970. Interactions between voluntary and postural mechanisms of the human motor system. *J. Neurophysiol.* **33**: 365–381.

19. KATER, S. B. 1974. Feeding in Helisoma trivolvis: The morphological and physiological bases of a fixed action pattern. *Amer. Zool.* **14**: 1017–1036.

20. KATER, S. B., and C. R. S. KANEKO. 1972. An endogenously bursting neuron in the gastropod mollusc, Helisoma trivolvis. *J. Comp. Physiol.* **79**: 1–14.

21. HILL, A. V. 1951. The transition from rest to full activity in muscle: the velocity of shortening. *Proc. Roy. Soc. London Ser B*, **138**: 329–338.

22. KELLER, E. L. 1974. Participation of medial pontine reticular formation in eye movement generation in monkey. *J. Neurophysiol.* **37**: 316–332.

23. KIRSNER, R. L. G. 1973. Visual control of human muscular movement. *J. Physiol.* **230**: 28P.

24. KOMMERELL, G., and W. F. HOYT. 1973. Laterpulsion of saccadic eye movements. *Arch. Neurol.* **28**: 313–318.

25. MAEDA, M., H. SHIMAZU, and Y. SHINODA. 1971. Inhibitory postsynaptic potentials in the abducens motoneurons associated with the quick relaxation phase of vestibular nystagmus. *Brain Res.* **26**: 420–424.

26. MAYNARD, D. M. 1969. Excitatory and inhibitory processes, pp. 56–70. *In* "The Interneuron." Mary Brazier [Ed.]. UCLA Forum Med. Sci. No. 11, Univ. Calif. Press, Los Angeles.

27. MILLER, P. J. 1973. "Dynamics of voluntary vergence in intermittent exotropia." Thesis for Master of Science in Mechanical Engineering, Univ. of California, Berkeley.

28. PERKEL, D. H., and B. MULLONEY. 1974. Motor pattern production in reciprocally inhibitory neurons exhibiting postinhibitory rebound. *Science* **185**: 181–183.

29. RASHBASS, C., and G. WESTHEIMER. 1961. Disjunctive eye movements. *J. Physiol.* **159**: 339–360.

30. REINHART, R. J., and B. L. ZUBER. 1971. Parameters of the control signals for saccadic eye movement: Electrical stimulation and modelling. *Exp. Neurol.* **30**: 148–161.

31. ROBINSON, D. A. 1964. The mechanics of human saccadic eye movement. *J. Physiol.* **174**: 245–264.

32. ROBINSON, D. A. 1973. Models of the saccadic eye movement control system. *Kybernetik* **14**: 71–83.

33. SCHILLER, P. 1970. The discharge characteristics of single units in the oculomotor and abducens nuclei of the unanesthetized monkey. *Exp. Brain Res.* **10**: 347–362.

34. SMITH, O. J. M. 1962. Nonlinear computations in the human controller. *IRE Trans. on Bio-Med. Electron.* **BME-9**: 125–128.

35. STARK, L., G. VOSSIUS, and L. R. YOUNG. 1962. Predictive control of eye tracking movements. *IRE Trans. on Human Factors in Electronics* **HFE-3**: 52–57.

36. STEINMAN, R. M., G. M. HADDAD, A. A. SKAVENSKI, and D. WYMAN. 1973. Miniature eye movement. *Science* **181**: 810–819.

37. TAKAHASHI, Y., M. J. RABINS, and D. M. AUSLANDER. 1970. "Control and Dynamic Systems," Addison-Wesley, Reading, Mass.

38. THOMAS, E. L. 1968. Movements of the eye. *Sci. Amer.* **219**: 88–95.

39. THOMAS, J. G. 1967. The torque-angle transfer function of the human eye. *Kybernetik* **3**: 254–263.

40. VOSSIUS, G. 1960. System der augenbewegung. *Z. Biol.* **112**: 27–57.

41. WEBER, R. B., and R. B. DAROFF. 1971. The metrics of horizontal saccadic eye movements in normal humans. *Vision Res.* **11**: 921–928.

42. WESTHEIMER, G. 1954. Mechanism of saccadic eye movements. *AMA Arch. Ophthalmol.* **52**: 710–724.

43. ZUBER, B. L., J. L. SEMMLOW, and L. STARK. 1968. Frequency characteristics of the saccadic eye movement. *Biophys. J.* **8**: 1288–1298.

44. ZUBER, B. L., and L. STARK. 1968. Dynamic characteristics of the fusional vergence eye-movement system. *IEEE Trans. Sys. Man. Cybernetics* **SSC-4**: 72–79.

45. ZUBER, B. L., L. STARK, and G. COOK. 1965. Microsaccades and the velocity amplitude relationship for saccadic eye movements. *Science* **150**: 1459–1460.