1417
SCIENTIFIC AMERICAN OFFPRINTS

The Trajectories of Saccadic Eye Movements

by A. Terry Bahill and Lawrence Stark

SCIENTIFIC AMERICAN
JANUARY 1979
VOL. 240, NO. 1 PP. 108–117

PUBLISHED BY **W. H. FREEMAN AND COMPANY** 660 MARKET STREET, SAN FRANCISCO, CALIFORNIA 94104

The Trajectories of Saccadic Eye Movements

In which a close examination of the rapid, sharp rotations of the eyeball known as saccades provides new information concerning the way the human brain controls eye movement

by A. Terry Bahill and Lawrence Stark

As a person reads, looks at a picture, drives a car or even stares at a spot on the wall his eyes make a multitude of movements, the great majority of which are the staccato flicks of the eyeball known as saccades. Saccadic eye movements serve to place the small area at the center of the retina called the fovea on different parts of the visual field. The fovea is the part of the retina where visual receptor cells are most densely packed, and so it is the part most capable of mediating detailed vision. Information from the periphery of the retina is used to direct the various saccadic movements, most of which range in magnitude from four minutes of arc to 15 degrees. (Larger shifts of gaze are usually accomplished by a combination of head and eye movements.)

The trajectories of different saccadic eye movements are unusually closely related to the patterns of motoneural firing, or the control signals, that generate them. In the eye-movement system the load the eyeball presents to the muscles is small and constant, and the muscles are strong and able to contract rapidly. As a result the precise neurological control signals that drive the muscles are faithfully reflected in the movements of the eyeball. In other words, the shape of the signals can be deduced directly from the saccadic trajectories. Once the control signals have been identified it is possible to obtain a better understanding of the nature of saccades and of the mechanisms that generate them.

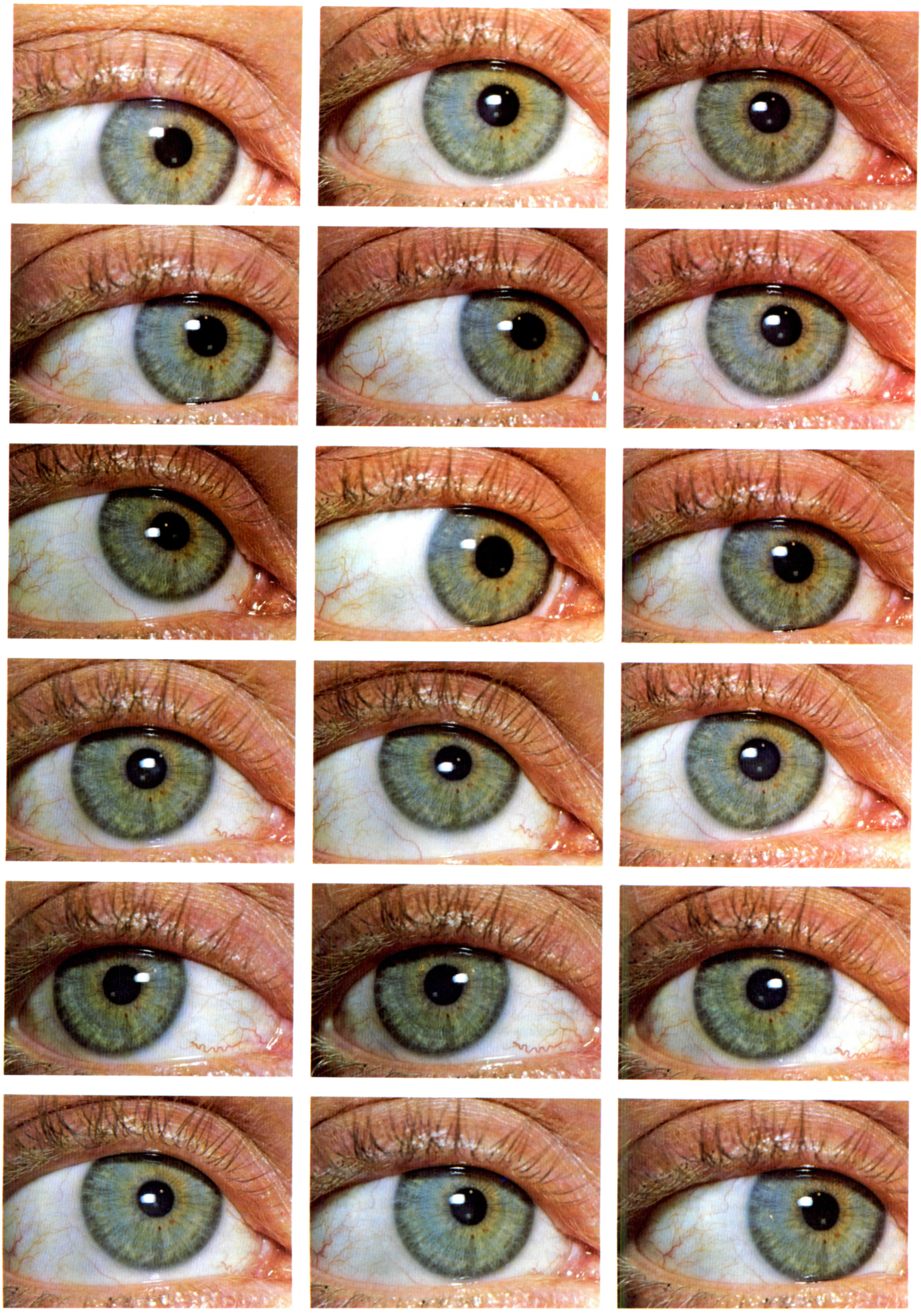
Here we shall describe the application of this method to several different kinds of saccadic eye movements: saccades that overshoot (or undershoot) their targets, double saccades and obliquely directed saccades. (Although much of the information that has been gathered on eye movements pertains to horizontal movements, most eye movements have a vertical component as well as a horizontal one.) First, however, we should describe our method of gathering eye-movement data and our tools for analyzing the data.

We used a photoelectric method to record the eye movements. In this method a pair of photodiodes mounted on spectacle frames are aimed at the border between the colored iris and the white sclera on each side of one of a subject's eyes. A small dot, the target, is made to move abruptly on a screen in front of the subject. When the eye turns, say toward the nose in response to a particular target jump, the photodiode nearer the nose is exposed to more of the dark iris and less of the light sclera, so that its photocurrent decreases; simultaneously the photodiode nearer the temple is exposed to more of the sclera and less of the iris, so that its photocurrent increases. The difference between the two currents provides an accurate measure of eye position. In our experimental system the photocurrents were converted into voltages, amplified and recorded on a computer disk-memory unit. The velocities of the recorded movements were computed by the computer itself. (Vertical movements were measured with a slightly modified apparatus.)

It is a common practice in recording eye movements to filter out high frequencies in order to eliminate instrumental noise and irregularities, but we maintained a large bandwidth: from zero to 500 hertz (cycles per second). This modification was important because high-frequency filtering would have removed the important fine details of the saccadic trajectories and also would have reduced the computed velocities. We were able to reduce instrumental noise to a very low level, so that it was much smaller than the biological signal we were measuring. Moreover, by continually calibrating and adjusting the instrumentation we were able to maintain a linear input-output relation for saccades of up to 20 degrees. Our experimental apparatus is quite inexpensive, and recently, in cooperation with William F. Hoyt of the University of California Medical School in San Francisco and B. Todd Troost of the University of Pittsburgh School of Medicine, we have employed it in gathering quantitative information to aid in the diagnosis of patients with various eye-movement disorders.

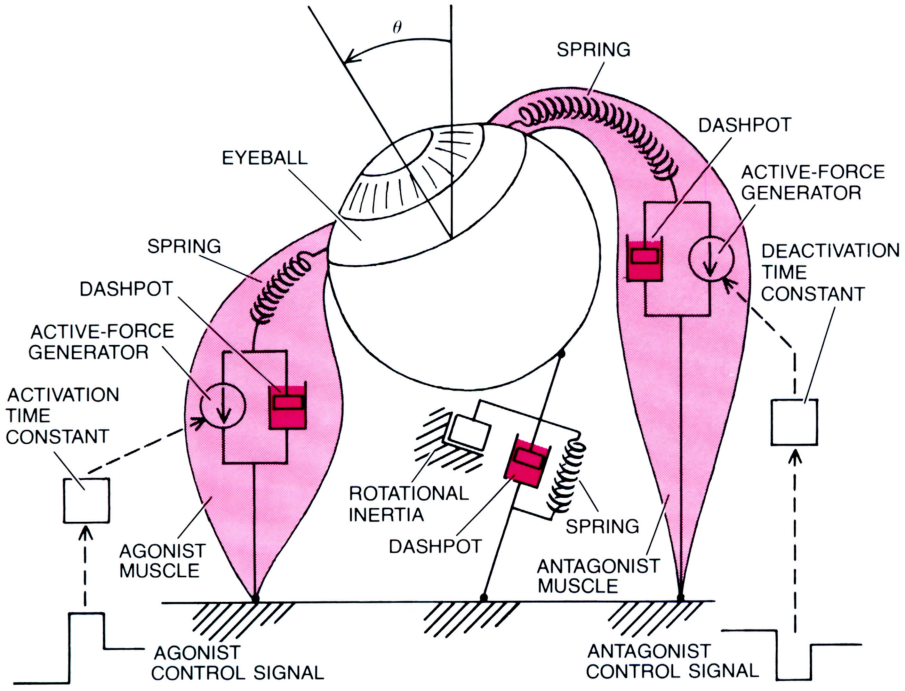
As our measuring instrumentation sampled the trajectory of the eye more finely, or frequently, we came closer to identifying the control signals driving each individual eye movement. At 10 samples per second (corresponding to a bandwidth of five hertz) we could study the fixation periods between saccades but not the rapid saccadic movements themselves. This rate is adequate for studying the eye movements in reading and the various paths the eye follows in scanning different scenes or objects. At 100 samples per second we could get information about the visual system's strategy for controlling the production of saccadic eye movements. For example, at that rate normal saccades can be

SACCADES SERVE TO MOVE the fovea of the eye, the small high-resolution area at the center of the retina, to different points in the visual field, as is depicted by the sequence of photographs shown here. The trajectories of these staccato rotations of the eyeball can be seen when a bright, undiffused light is shined on a subject's eye. As the subject looks at different points along a horizontal line (or even stares at a fixed point) the bright spot where the light reflects off the cornea provides a fixed reference point for viewing the saccadic eye movements that are generated. A person with normal vision typically executes about two saccades per second. Most of these eye movements range in magnitude from four minutes of arc to 15 degrees.



observed to occur at intervals as short as 200 milliseconds. In 1962 Laurence R. Young of the Massachusetts Institute of Technology and one of us (Stark) first suggested that the control system for the generation of saccades can be described as a sampled-data control system, that is, a control system that delivers output discontinuously. At 1,000 samples per second our instruments yielded a wealth of detailed quantitative information on the parameters of saccades, in particular peak velocity, duration and magnitude. And we could also see the minute variations in the shapes of the saccades, some of which are described below.

Two new conceptual tools were developed to help explain the variations in the saccadic trajectories. The first tool makes use of a standard engineering technique: when bioengineers want to analyze a very complicated system, they often resort to a model to describe the system in a less complicated way. Models are simpler and easier to deal with than real systems. For example, a street map is a model of the streets of a city, and it is easier to give directions by referring to the map than by referring to the real world. Many models have been developed to help study the neurological control of human movement. One of the earliest models of the oculomotor control system was developed by René Descartes, who first recognized that movements of the eyeball require the coordinated activity of at least two muscles: an agonist muscle that shortens and pulls against the eyeball to provide the turning torque for the eye movement, and an antagonist muscle that relaxes and lengthens. Hence normal eye movements can be made only through the reciprocal innervation of pairs of muscles. Descartes proposed this principle of reciprocal innervation in 1626, but it was not until 1963 that Gerald Cook of M.I.T. and one of us (Stark) incorporated it into a model of saccadic eye movement. Subsequently, in association with Michael R. Clark and Frederick K. Hsu of the University of California at Berkeley, we were able to develop a reciprocal-innervation model for all human eye movements.



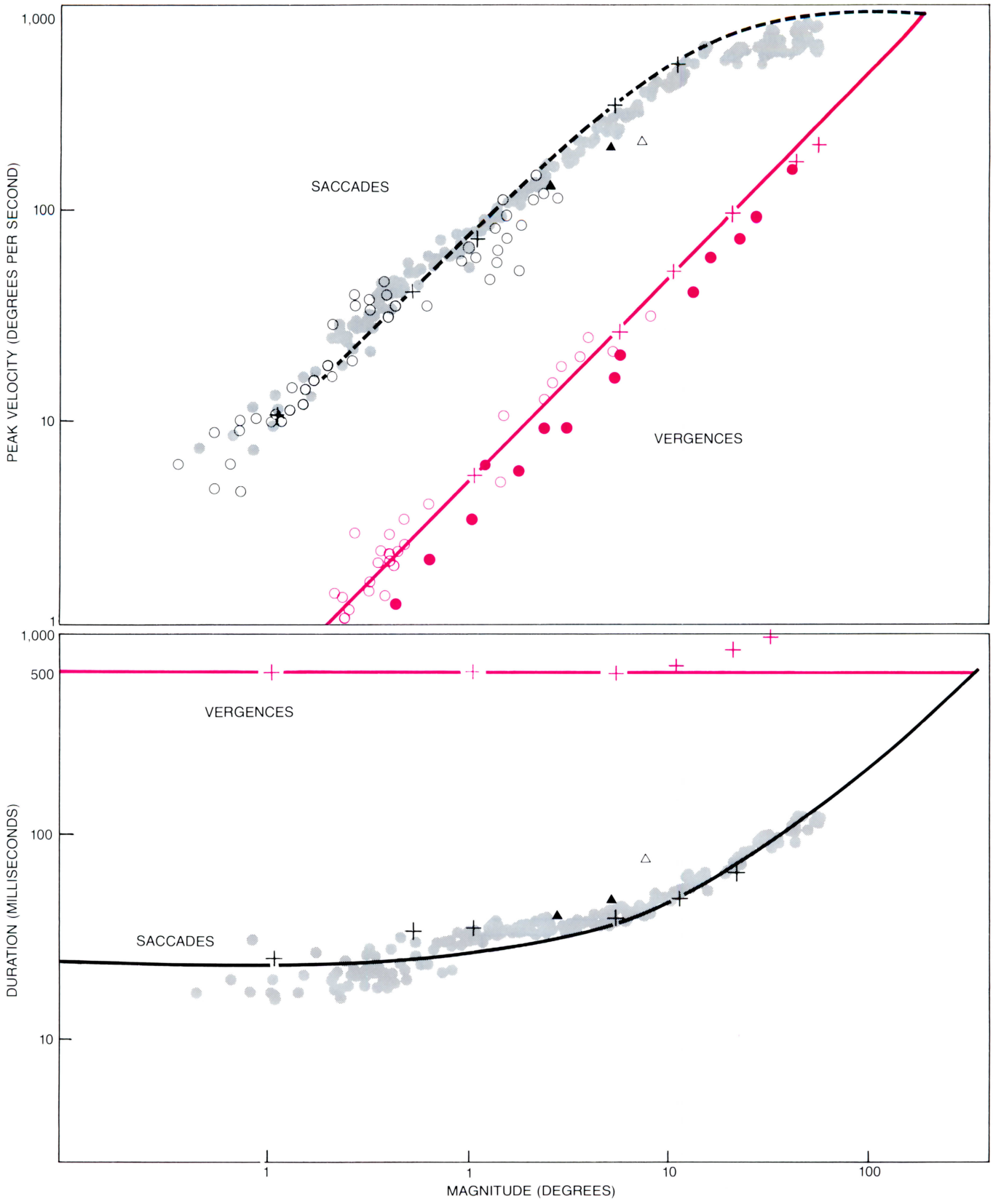
RECIPROCAL-INNERVATION MODEL the authors used to simulate various eye movements is based on the concept that eyeball rotation is effected through the innervation of at least two muscles: an agonist, which shortens and pulls against the eyeball, and an antagonist, which lengthens and relaxes. In the model neurological control signals, or patterns of motoneuron firing, such as the ones shown at the lower left and lower right, are given as input. Model elements known as activation and deactivation time constants reproduce the smoothing of the signals that occurs in the real eye system as the signals are delivered to and processed by the agonist and antagonist muscles. The inertia of the eyeball in rotation and the viscosity and elasticity of the eyeball-and-eye-muscle system are represented by the inertia of the rotating mass, the dashpot and the spring shown below the eyeball. (A dashpot, or shock absorber, is a viscous damping device.) The active-force generators in the model simulate the production of muscle force not as it is measured at the tendon of a real muscle but as it originates within the muscle. Each active-force generator is connected in series with a spring, which represents muscle elasticity. The output of the model is the eye position θ, the angle at which the eyeball is rotated away from the straight-ahead position. It is a special property of the real eye system that the trajectories of the eye are faithful reflections of the neurological control signals that generate eye movements. By experimenting with differently shaped control signals in the model, the authors were able to identify signals that generate particular variations in saccadic trajectories.

The reciprocal-innervation models are homeomorphic, in the sense of the word employed by the biomathematician Richard E. Bellman: there is a direct, detailed relation between the components of each of the models and the components of the real system. The advantage of working with a homeomorphic model of saccadic eye movements is that we were able not only to simulate the input of neurological signals and the output of eye movements, as might be done with a black-box model, but also to study how varying the individual parameters of the model affects output and how the physiological properties of the real muscles and tendons of the eye-movement system affect those parameters.

The parameters of our reciprocal-innervation model represent the inertia of the eyeball, its viscoelasticity on being rotated in the orbit of the eye, the elasticities of the ocular muscles and tendons and the apparent viscosity of the ocular muscles. (An inherent property of muscle, known as the Fenn-Hill-Katz force-velocity relation, makes the force-generating mechanism of muscle appear to be damped, as if by a shock absorber, or viscous damping element.) The model was implemented on a digital computer connected to a storage oscilloscope (an oscilloscope with a memory). Hence in our simulations of human eye movements we could vary the input of neurological control signals and immediately observe their effect on the output of saccadic trajectories. In particular we could experiment with differently shaped control signals until we found the one that generated the trajectory with parameters most like those of a particular type of saccade.

An important aspect of modeling is the validation of the model. We validated our model in several ways: qualitatively, by comparing the shapes of real saccades with those of model saccades; quantitatively, by comparing the parameters of the real saccades with those of model saccades; analytically, by performing a sensitivity analysis of the model (systematically varying individual parameters of the model in order to observe their influence on the total eye-movement system), and heuristically, by simulating eye movements the model was not designed to simulate.

The concept of our second tool comes from the Hertzsprung-Russell diagram, which astronomers use to classify and compare diverse types of stars. The diagram plots the luminosity of the stars

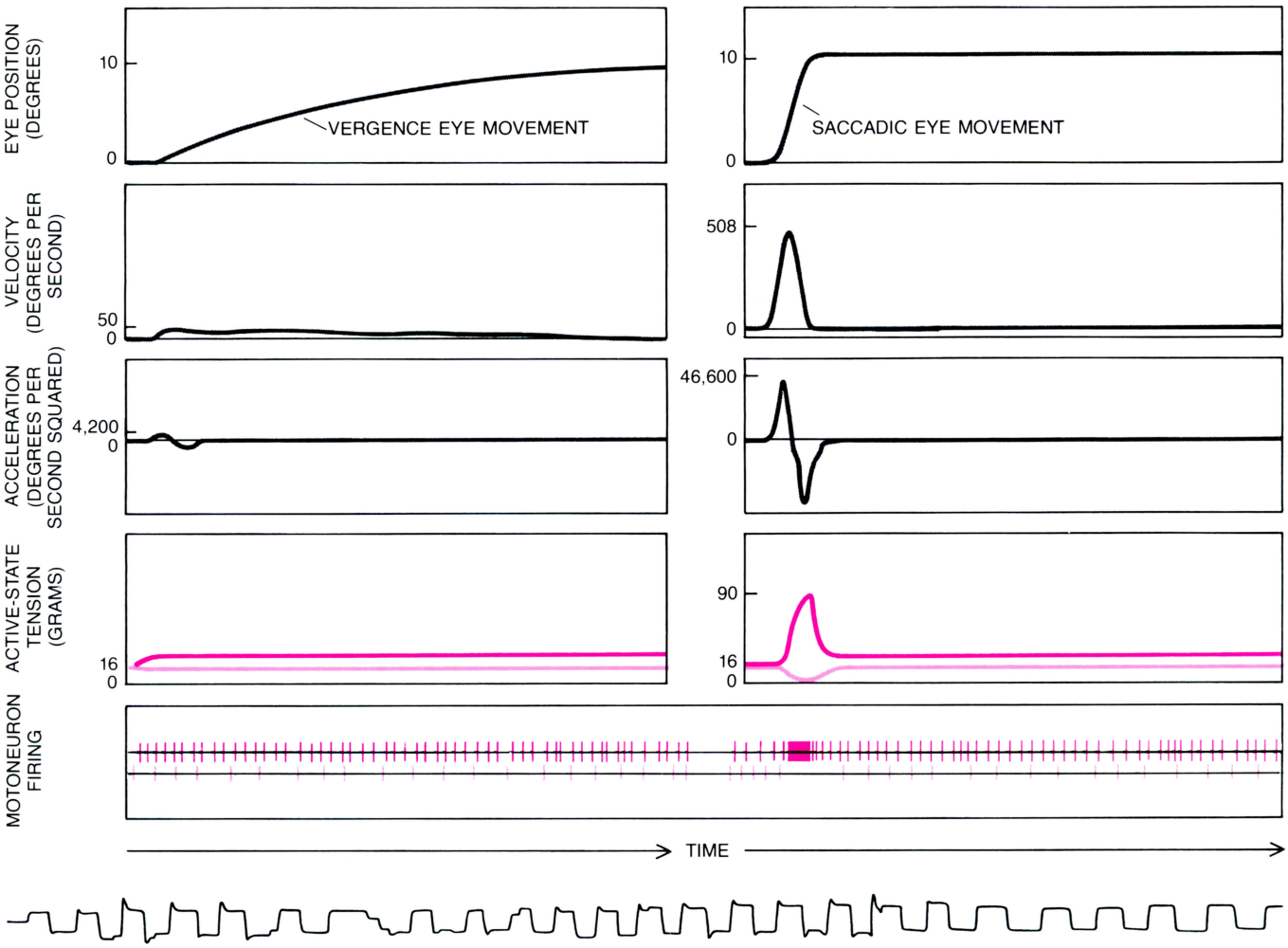


MAIN-SEQUENCE DIAGRAM for human eye movements plots the peak velocity (*top*) and the duration (*bottom*) of various eye movements as functions of their size. Experimental data on eye movements fall into two main groups on the diagram: the fast, staccato saccadic eye movements (*gray dots*) that make detailed vision possible and the slow, gliding "vergence" eye movements (*dots in color*) that track between far objects and near ones. There are variations in saccades; for example, some may overshoot their final position and return to it with a fast saccadic motion known as a dynamic overshoot (*black circles*). A slow, gliding movement known as a glissade (*circles in color*) may also return the eye to its final position. In some cases the eye may execute two small, closely spaced saccades instead of one large saccade to get from one position to another. As the diagram shows, it is not the overall movement (*open triangles*) that has the parameters of a normal saccade; rather it is the individual small movements (*filled triangles*) that are saccadic. The black crosses on the diagram represent saccades that were simulated on the reciprocal-innervation model (*see illustration on opposite page*); the crosses in color represent simulated vergences. The solid and broken lines shown in the illustration are analytical functions that fit the distribution of the experimental data.

against their surface temperature, or color. The majority of normal stars lie on a "main sequence": the swath of stars that runs diagonally across the diagram [see "The New, or Modified, Form of the Theory of Stellar Evolution," by Henry Norris Russell; SCIENTIFIC AMERICAN, October, 1925]. We have developed a similar diagram that enables us to classify and compare diverse saccadic phenomena. In our main-sequence diagram the peak angular velocity and the duration of eye movements are plotted as functions of the magnitude of the eye movements [*see illustration on preceding page*]. Our eye-movement data fall on two separate branches in the main-sequence diagram: one branch is made up of the rapid saccadic eye movements and the other is made up of the slow "vergence" eye movements that are utilized in tracking between distant objects and close ones. (The two other major types of eye movements—the vestibulo-ocular movements, which maintain visual stability during head movements, and the smooth-pursuit eye movements, which follow a moving object such as a flying bird—do not appear on the main-sequence diagram, since neither of these types of movement is elicited by target jumps.)

The main-sequence diagram demonstrates that there is little variation in the parameters of saccadic eye movements. For example, for a normal unfatigued subject the range in the duration of 10-degree saccades is from 38 to 45 milliseconds and the range in peak velocity is from 420 to 520 degrees per second. Similarly, a saccade covering 10 minutes of arc would last between 15 and 22 milliseconds and would attain a peak velocity of between 11 and 15 degrees per second. These ranges are small compared with those of other biological systems. We maintained small ranges of variation for the main-sequence data by (1) rejecting data from fatigued subjects, (2) plotting the parameters as functions not of the size of the target movement but of the size of the actual eye movement and (3) defining the size of the saccadic eye movement as being the size of the initial saccade and ignoring drift and noise at the end of movements. As a result, although the saccades of different human subjects have slightly different parameters, saccades whose parameters fall outside the main-sequence ranges are usually indicative of some pathological condition.

In 1965 observations of similar main-sequence data led Cook, Bert L. Zuber and one of us (Stark) to postulate that there is no basic difference between saccades. In other words, the parameters of all saccades fit on the main-sequence diagram, no matter how large or small the saccades are, what their purpose is or what kind of stimulus elicits them. Sub-



ABRUPT TARGET JUMPS generate two types of eye movements: slow, smooth vergence eye movements (*left*) and sharp, rapid saccadic eye movements (*right*). A vergence is generated by a step signal to the eye muscles: a small, sustained increase in motoneuron firing activity for the agonist muscle (*dark color*) and a similar decrease in firing activity for the antagonist muscle (*light color*). In this type of movement the difference between the actual eye position and the eye position coded by the new steady-state firing level provides the driving force that moves the eye slowly into its new position. A saccade is generated by a pulse-step signal to the eye muscles: a high-frequency burst of motoneuron firing receding to a new increased step level of firing for the agonist muscle (*dark color*), and a sharp drop in firing followed by a rise to a decreased step level of firing for the antagonist (*light color*). In this case it is the pulse that provides the driving force, moving the eye quickly to its new position; the step signal holds the eye in place. Irregularities in the pulse-step signals create differently shaped saccadic eye movements, as in successive movements between two targets 10 degrees apart shown at the bottom.

sequent experimentation has supported this theory of the unified nature of saccades.

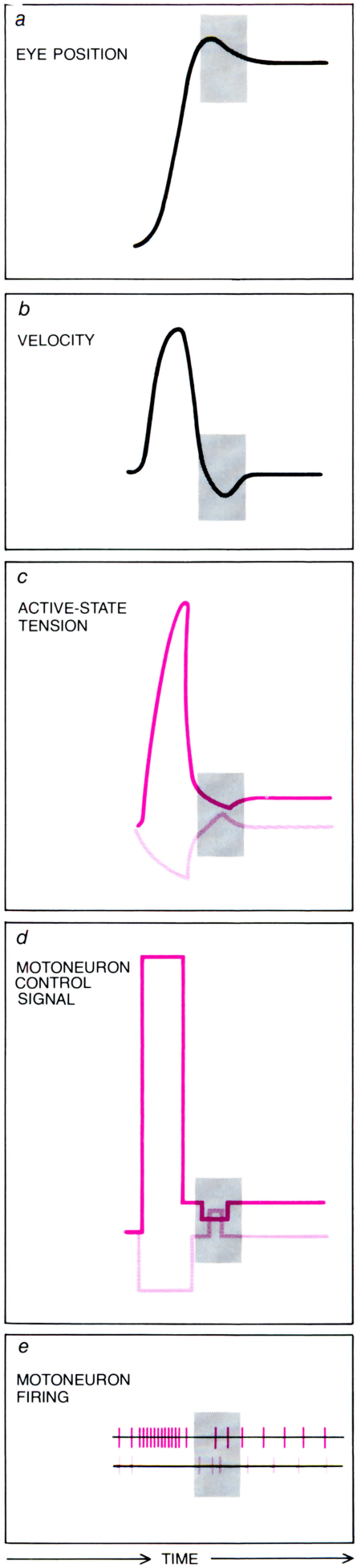
Experimental data also reveal, however, that although the main-sequence parameters and the general shape of saccades vary little, the structure of saccades varies greatly, that is, there are many small deviations from the normal saccadic trajectory. For example, some saccades drift past their target, others do not complete their movements with a single standard saccade and so on. These deviations appear quite irregularly, changing from day to day or from eye movement to eye movement, and they are often monocular, or limited to only one eye. (Indeed, the variations in the saccadic trajectories of a subject's two eyes are often quite different.) The explanation of this seemingly paradoxical phenomenon—the presence of trajectory variations in eye movements with stereotyped main-sequence parameters—is found by taking a closer look at the saccadic eye movements.

So far we have considered saccades as units, but in actuality each saccade is made up of several dynamic components, or smaller eye movements. It is the variation in the number and arrangement of these smaller components that gives rise to the variation in saccadic eye movements. We have been able to determine that all the variations in saccadic trajectories—all the changes in the configurations of the dynamic components of saccades—are due to variations in the neurological control signals of saccades. (In people with weakened eye muscles, of course, that is not the case.)

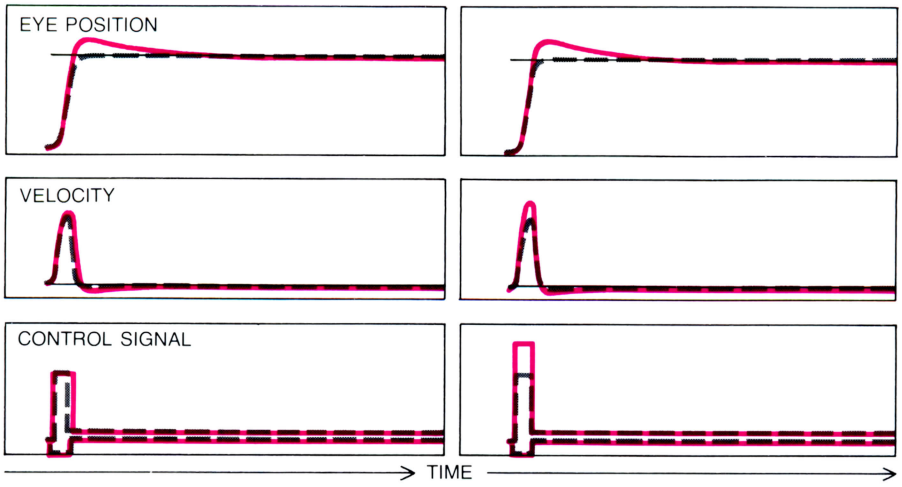
The reasons for the irregularities in normal control signals are not well understood; they do not depend on the amplitude of the saccades, the nature or visibility of the targets, the general lighting conditions or the purpose of the saccades. We do know that fatigue is responsible for some deviations, and it appears that psychological stress can also play a role. In any case the control signals do vary, and they give rise to the variations found in saccadic trajectories. As we have explained, the trajectories reflect the signals so faithfully that it is possible to see exactly how the changes in the signals affect the shape of the saccades.

First, consider the control signals that generate normally shaped eye movements. Saccadic eye movements and vergence eye movements, which form the two branches of the main-sequence diagram, are generated by two different types of neurological control signals. A slow, smooth vergence eye movement (in particular a divergence in which one eyeball rotates away from the other) is the result of a step change in the overall firing rate of the motoneurons that innervate the eye muscles: a sustained, low-frequency increase in the motoneuron activity for the agonist muscle and a similar decrease for the antagonist muscle produce a new steady-state firing level. The driving force for the eye movement is the difference between the actual eye position and the eye position coded by this new level of firing. A saccadic eye movement is generated by a two-part signal known as a pulse-step control signal. In this case the driving force for the eye movement is the pulse: a short burst of high-frequency motoneuron firing for the agonist and a corresponding pause in motoneuron activity for the antagonist. The pulse serves to move the eye rapidly from one point to another, and the step signal that follows serves to hold the eye in its new position. A pulse signal moves the eye much faster than a simple step signal; for example, a 10-degree vergence eye movement lasts for about 500 milliseconds, or about 10 times longer than a saccade of the same magnitude.

To understand how irregularities in pulse-step control signals create the minute variations in saccadic trajectories consider an example: the variation called saccadic overshoot, in which the eye travels beyond its ultimate fixation, or final position, and then returns, coming to rest on the fixation. Most saccades of the human eye display some type of overshoot. There are three distinct types, distinguished according to the way the eye moves back to its final position. In dynamic overshoot there is a fast return phase; for example, in a saccade with one degree of dynamic overshoot the return usually takes about 20 milliseconds, with the eyeball attaining a peak velocity of about 60 degrees per second. In glissadic overshoot there is a slow, gliding return, lasting for more than 200 milliseconds and for one degree of overshoot achieving a peak velocity of about five degrees per second. In static overshoot the eye remains fixed in the off-target position for between 150 and 200 milliseconds, until finally visual feedback elicits a corrective sac-



MODEL SIMULATION of a 10-degree saccade displays the variation in trajectory called dynamic overshoot: a normal saccade is followed by a small movement in the opposite direction that carries the eye back to a position it overshot (*a*). As the time-velocity relation (*b*) suggests, the return phase of a saccade with dynamic overshoot is itself a saccade. The driving force for the second small saccade is a momentary role reversal for the agonist muscle (*dark color*) and the antagonist muscle (*light color*) near the end of the saccadic eye movement (*c*). This change in the active-state tensions of the two muscles is in turn generated by a reverse pulse in the middle of the pulse-step signal generating the saccade (*d*), that is, there is a temporary reversal in the motoneuron activity for the saccadic movement. The firing patterns of a typical agonist motoneuron and a typical antagonist motoneuron are displayed at bottom (*e*).



SLOW, GLIDING GLISSADES at the end of a saccade can result from an oversize pulse component (*color*) in an otherwise normal pulse-step control signal (*broken black line*). When the pulse signal is too high (*right*), either because too many motoneurons were recruited for firing or because their firing rate was too high, the difference between the eye position at the end of the pulse and the eye position coded by the step signal generates a slow, smooth movement, following the primary saccade, that brings the eye back to the position it overshot. The same type of movement is generated when motoneuron firing goes on for too long, so that pulse is too wide (*left*). Thin black lines mark final eye position and velocity. Quantitative analysis of model simulations suggests that in subjects with normal vision pulse-width errors are commoner.

cade. Static overshoot is the only type that can be perceived with the unaided eye, and so it is the one most frequently observed by clinicians. Excessive static overshoot is often symptomatic of a disorder of the cerebellum.

Most saccades show dynamic overshoot. This irregularity seems to appear quite capriciously, so that a subject may exhibit dynamic overshoot in most of his saccades one day and in only a few the next day, and different subjects exhibit very different patterns of the variation. Dynamic overshoot seems to be independent of the size of the saccade; we have recorded saccades of all sizes (from a few minutes of arc up to 50 degrees) with and without dynamic overshoot. Moreover, dynamic overshoot does not seem to be limited to any initial eye conditions or direction of travel; it has been detected in saccades that begin with the eye looking straight ahead and in others that begin with the eye rotated as much as 35 degrees away from that position, and it has been detected in saccades directed toward the nose and in others directed away from it. The size of the overshoot increases with the size of the saccade, but in a normal saccade of, say, 10 degrees with dynamic overshoot the magnitude of the return is typically .25 degree. A comparison of the return phases of saccades having dynamic overshoot with the main-sequence data reveals that these return phases have the same main-sequence parameters as small saccades.

The eye system—the eye muscles, the eyeball and the surrounding tissue—is overdamped, as though there were large shock absorbers throughout the system to slow movement and eliminate oscillations of the eyeball. Therefore the fast return phase of a saccade with dynamic overshoot, which looks like an oscillation, cannot originate in the internal mechanics of the system; it must be of neural origin. Since the return phases are shaped like saccades, it might be expected they would be generated by saccadic motoneuron signals. To investigate this possibility we simulated saccades with dynamic overshoot on the reciprocal-innervation model.

The simulations show that a saccade with dynamic overshoot is generated when there is a reversal of motoneuron activity—an extra, reverse pulse—in the course of a standard saccadic pulse-step control signal. For the agonist muscle, then, the control signal consists of a sharp rise in motoneuron firing, followed by a sharp drop and then a rise to the new steady-state level determined by the step signal. Conversely, the signal to the antagonist muscle consists of a sharp drop in firing (the original pulse for the relaxing muscle) followed by a sharp rise and then a drop to the new steady-state level. As a consequence of these firing patterns the direction of the force generated by the active-state tensions of the two muscles is reversed in the middle of the saccadic movement, pulling the eyeball back toward a position it overshot and finally holding it there. (The active-state tension of a muscle is the force developed by the muscle not as it is measured at the tendon of the muscle but as it originates in protein interactions within the muscle.)

What is special about the control signal with the reversal of motoneuron control activity that creates saccades with dynamic overshoot? Clark and one of us (Stark) have shown that, to use the terminology of engineering optimality control, this saccadic pulse-step control signal is time-optimal, that is, there is no shape for a control signal that would move the eye faster between two points in the visual field than the one used by the brain for saccades. Recent electromyographic data obtained from human subjects by Alan Scott, Robert Kenyon and one of us (Stark) have verified that the control signal reversals predicted by the reciprocal-innervation model are indeed found in human beings. The decision to generate a saccade with dynamic overshoot would have to be made relatively early, before the signal is actually constructed. We found, however, that not all variations originate that early; for example, glissadic overshoot is generated when there is a mistake in the actual construction of the pulse-step control signal.

Remember that a saccadic eye movement with glissadic overshoot is composed of a normal saccade that overshoots its final position followed by a slow, gliding movement back to that position. The slow return movement was named a glissade by Robert Daroff and his co-workers at the University of Miami. Glissadic overshoot appears much less often than dynamic overshoot in the saccades of normal, unfatigued subjects. Its frequency is greatly increased, however, by fatigue and certain pathological eye conditions. When fatigue can be ruled out as a controlling factor, analysis of glissades can aid in the diagnosis of internuclear ophthalmoplegia, a syndrome often observed in multiple sclerosis.

We began our study of glissadic eye movements by considering how the glissades fit into the main-sequence diagram. We observed that the velocity and duration of glissades do depend on their magnitude and that those parameters fall on the vergence branch of the diagram. It seemed likely, then, that like vergence eye movements they would be driven by the force generated by a difference between an actual eye position and an eye position coded by a step change in motoneuron firing. To test that possibility we simulated saccades with glissadic overshoot on the reciprocal-innervation model.

The simulations suggested that a glissade results in instances where the pulse and step components of a saccadic control signal are mismatched. For example, glissadic overshoot is caused when the step component of a control signal is correct with respect to a particular target distance but the pulse component is too high (either because too many motoneurons have been recruited for firing or because their firing frequencies are too

high) or too wide (because the duration of the pulse is too long). The pulse then propels the eye into a new position beyond the target position, but since the step signal is coded for the earlier position, the eye slowly drifts back until an equilibrium is reached. Similarly, if the pulse of a signal is too small, the eye stops short of its target position and is pulled slowly forward by the step signal. This phenomenon is called glissadic undershoot.

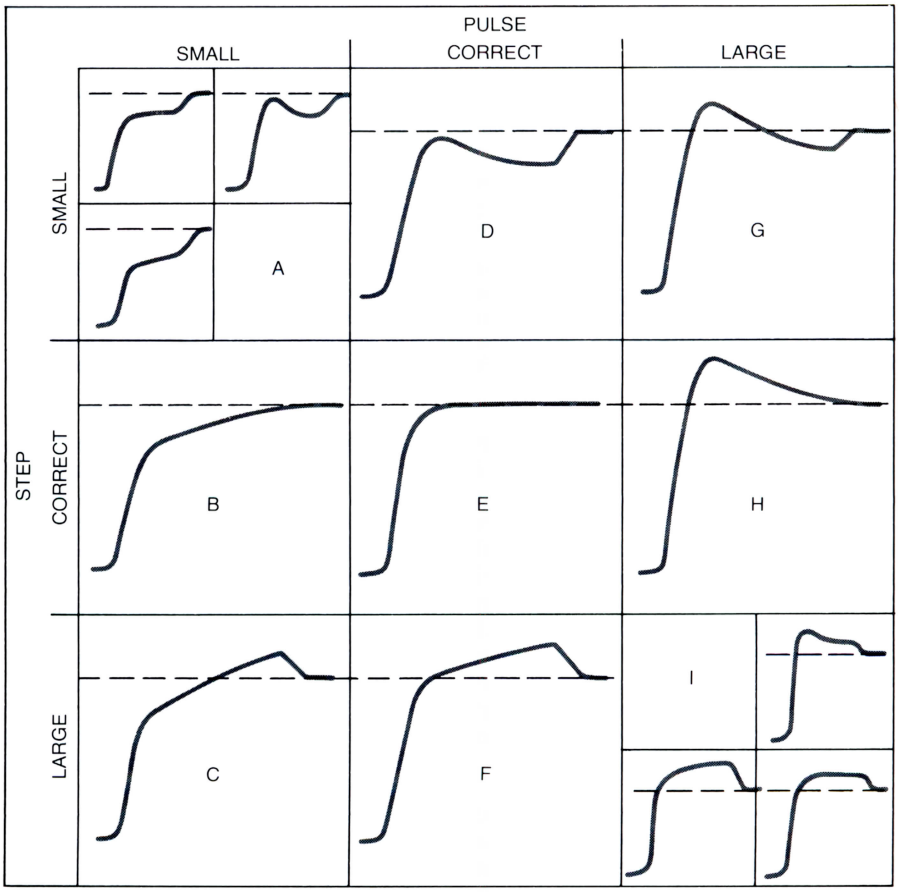
It is important to note that glissadic eye movements are not vergence eye movements. In spite of their similarities, the two types of movement differ in several ways. For example, vergences are a binocular phenomenon and glissades are usually monocular. Furthermore, there is a pause between a target jump and the resulting vergence eye movement, but there is no such pause between the saccadic error and the glissade that corrects it.

There are many different ways in which the pulse and step components of a control signal can be mismatched. In any particular saccade either one or both of the components can be undersized, oversized or correct. When it is the step component that is incorrect, being either too large or too small for a particular target position, a steady-state position error is established; the eye remains off-target for hundreds of milliseconds, until visual feedback elicits a corrective saccade. When both the pulse and step components are either too large or too small, the resulting saccadic eye movement will have a glissade to either the left or the right or will have no glissade at all, depending on which component has the larger error.

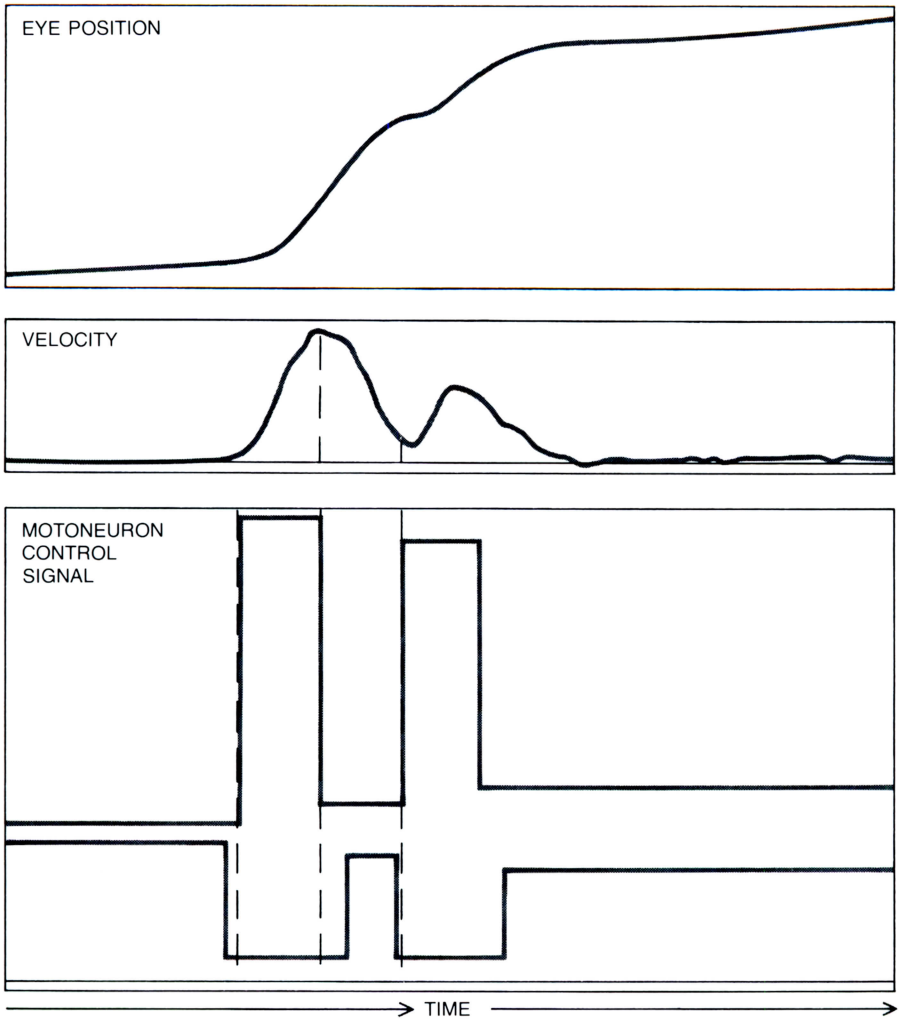
Glissades are only one of the many types of saccadic variation whose frequency is increased by fatigue. Fatigue creates irregularities in the saccadic control signals, so that saccades are slowed down and even broken up into multiple movements. After a subject has executed, let us say, 500 10-degree saccades, his fixations become more inaccurate, requiring more corrective saccades. Aberrant saccades, whose parameters do not fit on the main-sequence diagram, begin to appear. In some cases the eye changes its fixation not with one large saccade but with two smaller saccades, called double saccades. At that point if the subject is asked to make "more accurate" saccades, he can do so. After about 1,200 10-degree saccades the subject will still be able to make some normally shaped saccades, but their maximum velocities will be some 10 percent lower than those of the subject's first saccades of the day. It is at this point that overlapping saccades begin to appear: double saccades so close together that their velocity profiles overlap. All saccades the subject produces after this time will exhibit signs of fatigue.

It should be noted that larger saccades are more fatiguing than smaller ones. After making only 30 50-degree saccades or 80 30-degree saccades a typical subject cannot make normally shaped saccades. Moreover, a subject who becomes fatigued on small target jumps still shows signs of fatigue if he switches to larger target jumps, whereas a subject who becomes fatigued on large target jumps is sometimes able to execute smaller normal saccades before the effects of fatigue set in again.

We are using the word fatigue in a very general sense, because in our experiments we have no way of differentiating among muscle fatigue, sensory adaptation to stimuli and central-nervous-system habituation to impulses. In any case fatigue does bring about deviations in the neurological control signals and so creates variations in saccadic trajectories. Overlapping saccades may be the most irregular of these saccadic variations. Simulations on the reciprocal-innervation model suggest that overlapping saccades are two saccades so close together that the eyeball does not have time to decelerate to zero velocity before the second saccade begins. This does not mean the pulse portions of the two control signals overlap, however; the pulse of a normal saccade is usually completed about halfway through the movement, so that there is a brief drop in the high-frequency motoneuron activity between the two saccades in an overlapping pair. The main-sequence parameters of these pairs of saccades indicate that their generation depends on more than just a breakdown in the motoneuron signal for one saccade that would have covered the same distance as the two small saccades. If the saccades were simply a temporal decomposition of the larger saccade, then the sum of their parameters, but not the individual parameters themselves, would fit into the main-sequence data. Such is not the case. Rather, it is the individual parameters that fit into the main sequence, implying it is the magnitude and not the duration of the larger saccade that has



PULSE AND STEP COMPONENTS of a saccadic control signal can be mismatched in a variety of ways, resulting in the 13 trajectories shown here. For example, when the step is correct but the pulse is too large (*H*), a glissadic overshoot is generated (*see illustration on opposite page*). When the pulse is too small (*B*), the resulting saccade stops short of its final position and a glissade finishes the movement. When the step component is incorrect (either too strong or too weak), the saccade moves the eye to an off-target position, where it remains until visual feedback instigates a corrective saccade, either forward to the target (*D*) or backward to it (*F*). When both the pulse and the step are too large (*I*) or too small (*A*), the primary saccade may be followed by a rightward glissade, a leftward glissade or no glissade at all, depending on the relative sizes of components. Broken line through each trajectory marks final, or target, eye position.



OVERLAPPING SACCADES (*top*) are two normal saccades spaced so close together that their velocity profiles overlap (*middle*). Model simulations indicate there is no such overlap, however, in the motoneuron signals that generate the saccades (*bottom*). In most saccades pulse ends about halfway through eye movement, so that in overlapping saccades there is a drop in motoneuron activity between two signals. Fatigued subjects often show overlapping saccades.

been divided into two parts. Therefore each saccade in an overlapping pair is generated by a normal pulse-step signal for a saccade of its magnitude. Since the overlapping saccades are not the result simply of a pause in motoneuron activity, the motor control center that constructs the neurological control signals must make an early decision to abandon the single-saccade, time-optimal control strategy and construct a pair of overlapping saccades, each of which is time-optimal for its magnitude.

Let us now take up the oblique saccades, which have both horizontal and vertical components of movement. Main-sequence analysis of these components reveals that the movement in both directions is basically saccadic. Vertical saccades are somewhat slower than horizontal ones, so that they are shifted slightly from the horizontal-saccade data on the main-sequence diagram, but otherwise vertical and horizontal saccades are similar in their overall dynamical structure. The same types of irregularities appear in horizontal and vertical saccades with about the same frequency. Since there seems to be no intrinsic difference between the trajectories of the two components, there is no need to discuss the neurological origins of the vertical-saccade variations: dynamic overshoots, overlapping saccades and so on. There is, however, a most interesting question still to be answered: What is the relation between the trajectories of the two components of oblique saccadic eye movements?

The horizontal and vertical components of oblique saccades are physiologically independent, that is, they are implemented by separate neural channels with different motor control centers, different motoneurons and different eye muscles. We believe that in addition the components are temporally and dynamically independent, with unrelated saccadic trajectories generated by distinct patterns of neurological control signals. This thesis is supported by two main observations: there seems to be no relation between the timings of the two components, and the particular variations in the trajectory of one component seem to be unrelated to the particular variations in the trajectory of the other.

The temporal independence of the components is demonstrated by the fact that oblique saccades are infrequently straight. A saccade can be straight, of course, only if its horizontal and vertical components begin and end simultaneously. Main-sequence analysis of the components of typical oblique saccades shows, however, that in most cases the components of a saccade have different durations. For example, a typical five-degree saccade inclined at a 53-degree angle from the horizontal has a three-degree, 30-millisecond horizontal component and a four-degree, 40-millisecond vertical component, so that it is clearly impossible for the two components to begin and end simultaneously. The shorter component of an oblique saccade may start at any time in the course of the longer component, and on occasion the end of one component may occur before the beginning of the other, giving rise to an *L*-shaped trajectory.

The dynamic independence of the horizontal and vertical channels is demonstrated by the curvedness of oblique trajectories. In other words, the irregularities in the shape of an oblique saccade result from the differences in the dynamic structures of the two components. For example, there seems to be no connection between the appearance of, say, dynamic overshoot in the trajectory of one component and its appearance in the trajectory of the other.

As we have shown, the variations in saccadic trajectories are generated by diverse types of irregularities in the pulse-step control signals: motoneuron-firing reversals, pulse-step mismatches and so on. Hence the dynamic independence of the components' trajectories further confirms our belief that they are generated by control signals that are in turn generated by independent motor control centers in separate parts of the brain. We have observed that the trajectories of oblique saccades are much more varied than those of horizontal saccades, which might be expected in movements where irregularities can be introduced through deviations in two different sets of neurological signals.

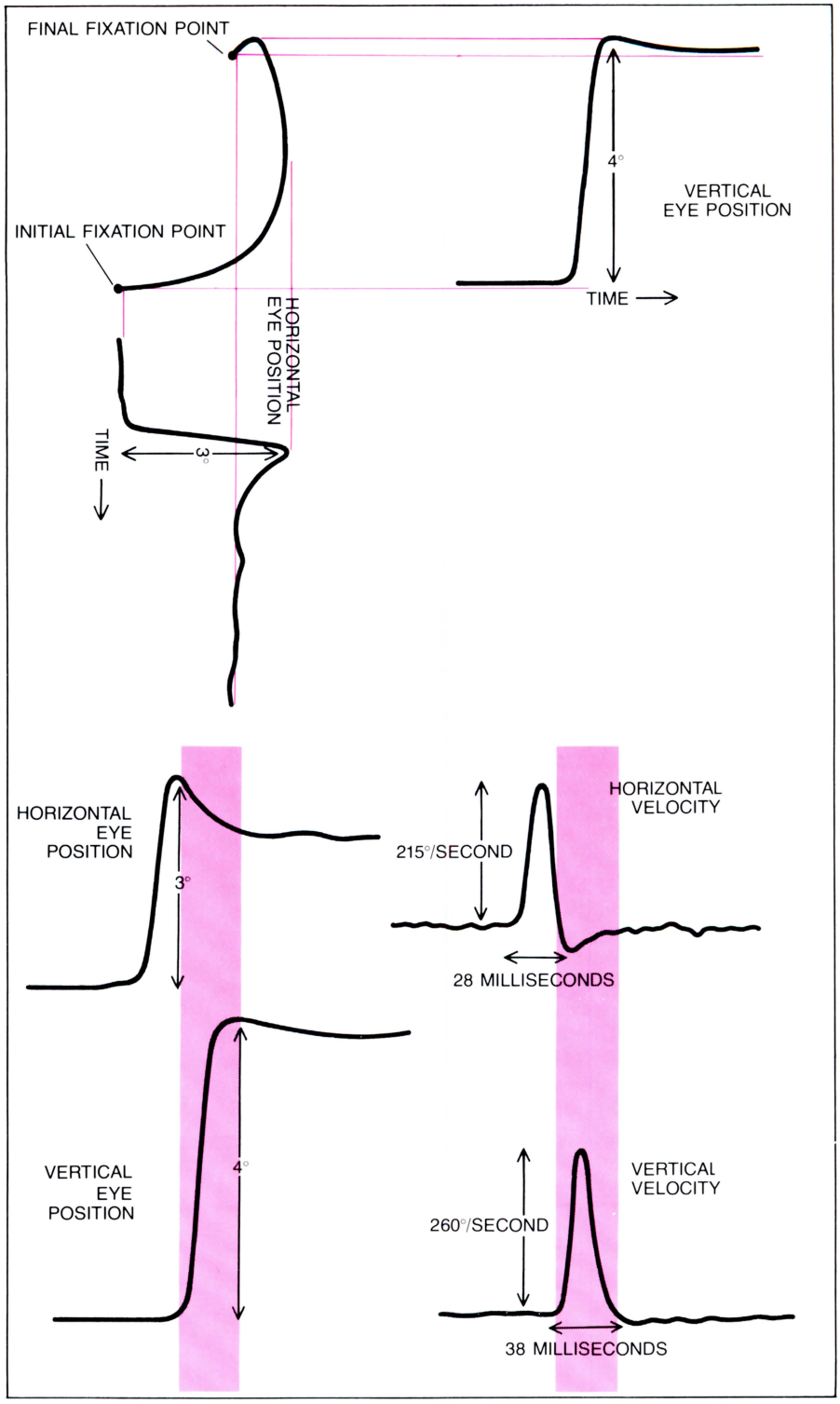
It should be noted that in fact some information—perhaps only noise—is exchanged between the neural channels that implement the horizontal and vertical saccadic eye movements. For example, purely horizontal target jumps rarely elicit totally linear saccades. In most cases the horizontal saccade elicited by such a target jump is accompanied by a transient vertical movement, as was not-

ed by Raymond Dodge of Wesleyan University some 80 years ago. It appears that noise from the horizontal-movement channel affects the vertical-movement channel as well, provoking the extraneous vertical component.

Hence when the vertical components of eye movements as well as the horizontal ones are considered, a greater variability in the trajectories of saccades is observed. Successive saccadic eye movements between two fixed points usually have different trajectories, and often they have dramatically different trajectories. Even when succeeding oblique saccades have identical horizontal and vertical components, the dynamic and temporal relations of the components in each movement (and by inference the relations of the neurological control signals behind each pair of components) are almost always different. If the eye-movement system is further enlarged to include both eyes, an even greater variability is found. In particular, dynamic overshoot, glissades, double saccades and overlapping saccades are monocular phenomenona.

Surprisingly, however, the variability in saccadic trajectories does not seem to be a matter of importance to the brain. Vision is not disturbed by it, perhaps because vision is suppressed before and during a saccade. (This suppression can be demonstrated by switching one's gaze back and forth between two objects in a room. Although the image on the retina clearly moves, the room is not seen in swirling motion.) Furthermore, before each saccade the brain computes the expected change in the eye's frame of reference, so that the viewer is not subsequently aware of the shift in his retinal image. When the movement has been completed, a comparison is made between the expected image and the actual image. It seems likely that if the trajectories of saccadic eye movements became too extreme, the brain would interfere and modulate them, interposing some form of control or recalibration, possibly accomplished in the cerebellum, to make the trajectories less bizarre.

In our investigation of the trajectories of saccadic eye movements we have made extensive use of the techniques of bioengineering, as is demonstrated by our experimental apparatus, which was explicitly designed to match the high velocity of the eye; our utilization of a digital computer for data collection, data analysis and simulation; our application of the main-sequence concept to encompass the diverse data on eye movements, and our development of the reciprocal-innervation model as a means of conceptualizing the control of eye movements. With these tools we have gained a much deeper understanding of the nature and control of various eye movements. Indeed, it has been possible to identify the level of neurological control activity at which many specific types of variation in eye movement originate. As a result we have in many instances been able to show precisely how fatigue, stress and disease affect the neurological control signals for eye movement and therefore the normal functioning of the eye. Clinical applications of these advances are now being pursued, and we believe they will lead to better diagnosis and treatment of patients with a variety of physical disorders.



OBLIQUE SACCADIC EYE MOVEMENT (*top*) has a vertical component of movement as well as a horizontal component, each of which is itself a saccade. As the position and velocity plots of the two components (*bottom*) indicate, the components of oblique saccades seem to be dynamically independent, with different durations and desynchronized trajectories. In this case the horizontal saccade has dynamic overshoot but the vertical saccade does not; moreover, the horizontal saccade is nearly finished by the time the vertical saccade (*band in color*) begins.

The Authors

A. TERRY BAHILL and LAWRENCE STARK are bioengineers interested in the control of movement. Bahill is assistant professor of electrical engineering and bioengineering at Carnegie-Mellon University and assistant professor of neurology at the University of Pittsburgh School of Medicine. He was educated at the University of Arizona, San Jose State University and the University of California at Berkeley, where he obtained his Ph.D. in 1975. As director of the Neurological Control Systems Laboratory at Carnegie-Mellon, Bahill studies the control of eye movements and locomotion in man. He is also investigating the development of walking in infants, with his one-year-old son Alexander serving in the pilot study. Stark is professor of physiological optics and engineering science at the University of California at Berkeley and professor of neurology at the University of California at San Francisco Medical Center. He did his undergraduate work at Columbia University and received his M.D. from the Albany Medical College in 1948. After his internship and a research fellowship in England he was on the faculties of the New York Medical College, Yale University, the Massachusetts Institute of Technology and the University of Illinois. He moved to Berkeley in 1968. Stark's research involves computer analysis of the control of movement of the human eye and hand. He writes: "My hobby, bird-watching, brings together problems in the neurological control of flying and the cognitive pattern-recognition of bird species."

Bibliography

THE ANGLE VELOCITY OF EYE MOVEMENTS. Raymond Dodge and Thomas Cline in *Psychological Review,* Vol. 8, pages 145–157; 1901.

DERIVATION OF A MODEL FOR THE HUMAN EYE-POSITIONING MECHANISM. Gerald Cook and Lawrence Stark in *Bulletin of Mathematical Biophysics,* Vol. 29, pages 153–174; 1967.

NEUROLOGICAL CONTROL SYSTEMS: STUDIES IN BIOENGINEERING. Lawrence Stark. Plenum Press, 1968.

DYNAMIC OVERSHOOT IN SACCADIC EYE MOVEMENTS IS CAUSED BY NEUROLOGICAL CONTROL SIGNAL REVERSALS. A. Terry Bahill, Michael R. Clark and Lawrence Stark in *Experimental Neurology,* Vol. 48, pages 107–122; 1975.

TIME OPTIMAL BEHAVIOR OF HUMAN SACCADIC EYE MOVEMENT. Michael Clark and Lawrence Stark in *IEEE Transactions on Automatic Control,* AC-20, pages 345–348; 1975.

GLISSADIC OVERSHOOTS ARE DUE TO PULSE WIDTH ERRORS. A. Terry Bahill, Frederick K. Hsu and Lawrence Stark in *Archives of Neurology,* Vol. 35, pages 138–142; March, 1978.