OCULOMOTOR EFFECTS ON VISION[1]

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FOVEATION

Over the past day and a half, we have heard many intriguing facts and speculations about the machinery used to rotate the eye and disorders that disrupt its normal function. My task is to describe how this machinery contributes to normal vision. This is not an easy task. It can be simplified by making two assumptions. The first is that much of this machinery compensates for movements of the body—not simply the large movements that we use to get about, but also small movements produced by such things as the heart beat and breathing. It also helps to assume that the stabilizing machinery works exceedingly well. It must in order to cope with a disease of the retina that I will call "hereditary localized hyperplasia".

The etiology of this disease is known. It derives from the widespread incidence of a fovea of which only a small portion has the neural substrate required for the kind of vision that has become important for modern human life. (Polyak called this region the foveal floor.) The foveal floor occupies only one one-hundredth of one percent of the area of the retina which forces high-quality human vision to rest on a speck of tissue floating in a sea of relatively crude light sensitive substance.

Many characteristics of human oculomotor performance make sense only as attempts to cope with this genetic defect. There would be no need to move the eyes if the retina were homogeneous and if the foveal floor covered its entire surface. If it did, machinery for head rotation and stabilization of the eye would be all we need to see everything very well. We would be even better off with lateral rather than frontal eyes. These would allow us to dispense with most head movements as well. Note, however, that I am not emphasizing the desirability of such arrangements in order to criticize Charles Darwin, or Jehovah, or whomever else you hold responsible for deficiencies in the way that we are constructed. Rather, I find it helpful to keep this affliction in mind while considering the effects of eye movements on vision. Many of our eye movements are made because of such shortcomings.[2]

Perhaps the simplest way to see how the oculomotor system copes with this disease is to look at the performance of the cat. This animal has a specialized central region but it is less hyperplastic than man's. Figure 1A shows one of three cats studied by Winterson in Professor Robinson's apparatus. The cat is alert and ready for two-dimensional recordings of the way in which she looks about in an ordinary visual scene. The scene the cat could see and the typical visual search pattern of the cat are also shown.
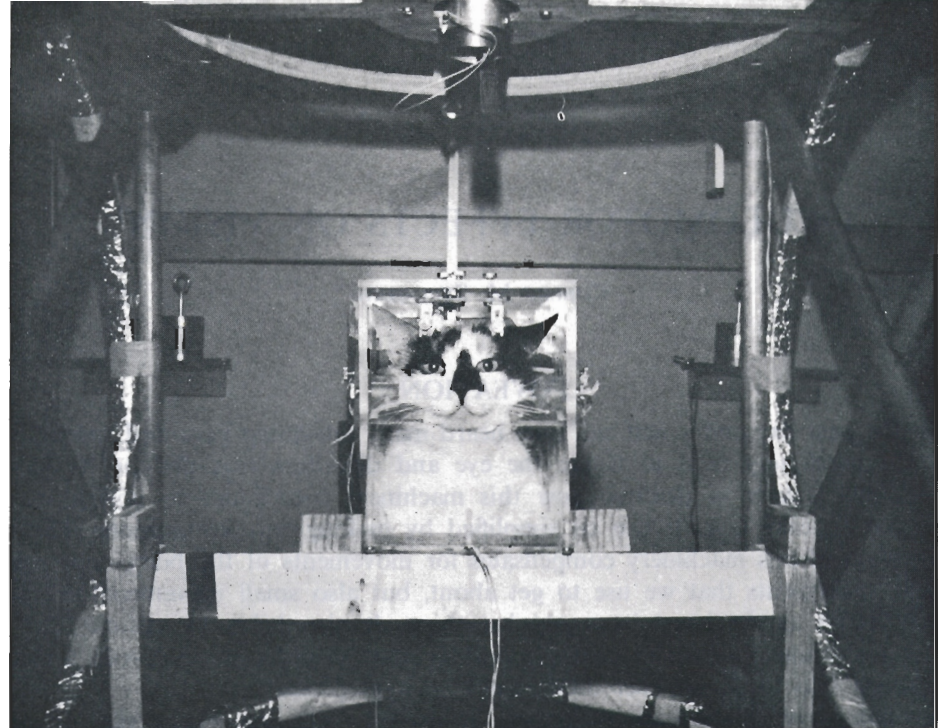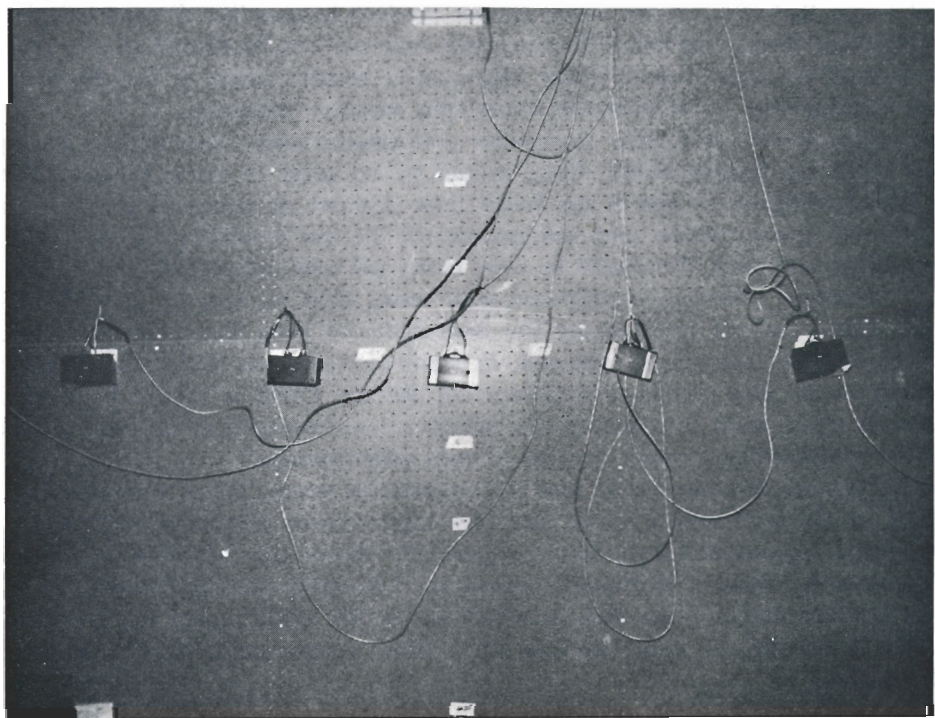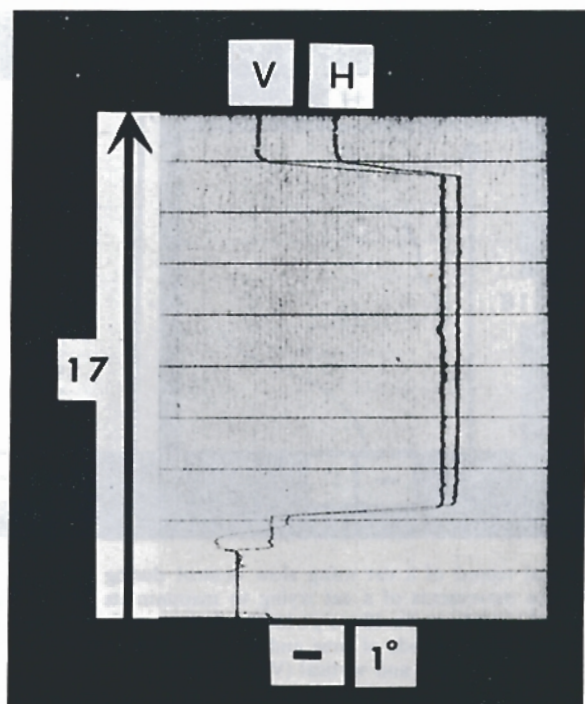
FIG. 1(A).

Fig. 1(C).

Fig. 1. (A) A cat positioned for recording eye movement by means of the magnetic field search coil technique. The search coil (not visible) is held to the sclera of the right eye and its leads are carried under the skin to a connector screwed to the skull just above the eye. The head is held in place with respect to the field coils surrounding the animal by means of a metal band screwed to the skull while the cat is supported and confined in a fitted plastic box. (B) Assorted objects located two meters in front of the cat's eye that could be inspected. (C) The typical two-dimensional search pattern of an alert cat. The record begins at the bottom. Repetitive horizontal lines show 1 sec periods of time and the bar shows 1° of arc on both the horizontal (H) and vertical (V) meridians.

Cats make one or two saccades to shift their lines of sight to a region of their choice and then inspect that region for several seconds while using slow control to keep the eye in place. The average fixation pause of the three cats studied lasted 6 sec and very long pauses were occasionally observed. Figure 2A shows a cat using slow control during a 17-sec visual inspection of a single region in visual space and Fig. 2B shows that the cat's slow control is an active process. When visual stimulation is removed (the room lights are turned off), the eye rapidly drifts away from the preferred fixation direction. A saccade may be made to restore eye position when this happens but eye position cannot be maintained when there is no visible target to activate slow control. The fine grain nature of slow control is shown in Fig. 2C where drift direction can be seen to change 4 to 6 times each second. Such irregular drift oscillations keep the eye in place very well (standard deviations of the cat's line of sight do not exceed 2 to 4 min of arc during slow control).[3]

Now, let us consider how the naïve rhesus monkey looks at a visual target. The rhesus has a much higher degree of hyperplasia than the cat. It approaches that of man. Figure 3A shows a fixation record of one of the four monkeys studied in detail
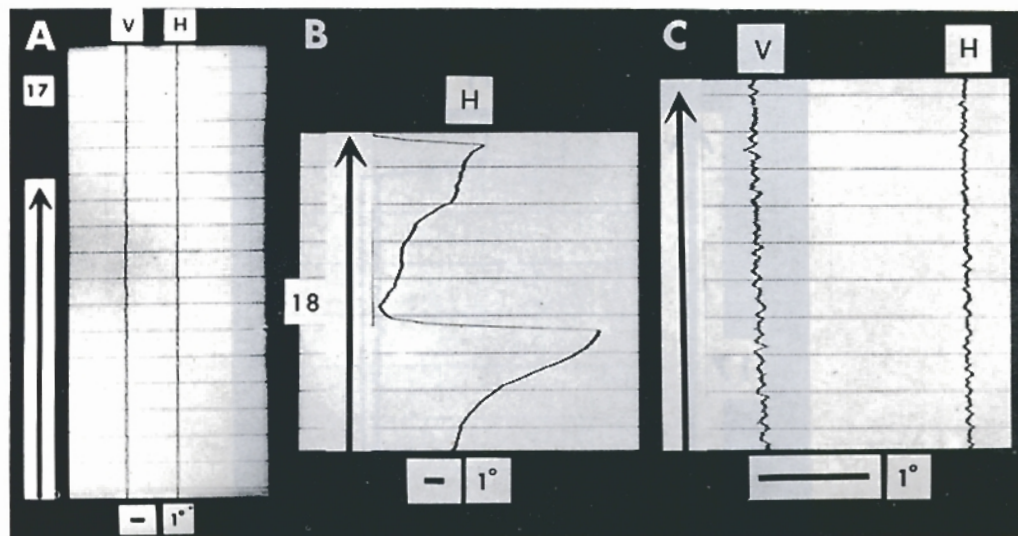
FIG. 2. (A) A two-dimensional record of a cat using slow control during a 17-sec inspection of a single object. (B) Horizontal (H) eye movements of a cat trying to maintain its line of sight in total darkness. (C) High-gain two-dimensional recording of a 10-sec period of slow control. All records begin at the bottom; repetitive horizontal lines show 1 sec periods of time and the black bars show 1° of arc on the horizontal (H) and vertical (V) meridians.

by Skavenski. Note that this monkey makes saccades frequently. Sometimes as often as three each second. Also, note that these saccades tend to be smaller than the cats'—about 2.5° on the average. Figure 4A shows the typical human fixation pattern recorded under much the same conditions. The subject's head was not bolted, but it was stabilized by a bite-board. Again we see fairly frequent saccades but they are one-thirtieth the size of those seen in the rhesus monkey—they average only 5 or 6 min of arc.

It is difficult to know what the drifts are doing in man and the rhesus because both jump around so much. Does a high degree of foveal specialization cause the slow control subsystem to be lost? This is not the case as can be seen in Fig. 4B where a human subject is shown using slow control in the record on the right. The difference is simply a matter of changing the instructions. Man has not lost the capacity (shown by the cat and the rabbit) to hold his eye in place with drifts. Skavenski has also shown this to be true of rhesus monkeys. First he spent several months training them to fixate like humans (make micro- rather than large saccades). This is shown in Fig. 3B. Skavenski then changed the discrimination problem, which encouraged the monkey to suppress saccades, and found that slow control kept the eye in place. A good example of this is shown in Fig. 3C.[4]

What is the significance of these comparative results? They suggest that retinal hyperplasia provokes a frenzy of saccadic activity. This is probably the most striking symptom of the disease. The best way to see these symptoms is to hold the head rigidly and require the patient to be very careful while he inspects a small portion of the visual array. The disease is most advanced in man where something more than genetic limitations are probably operating because there is no reason to believe that man requires smaller saccades than those seen in the fixation pattern of the untrained rhesus. This
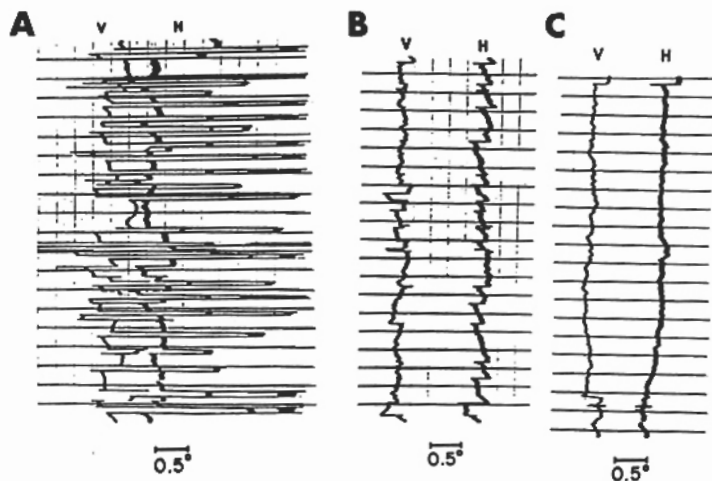
FIG. 3. (A) A representative two-dimensional record of rhesus fixation before special training. (B) A representative two-dimensional record of rhesus fixation after several months of special training. (C) A record of rhesus slow control. All records begin at the bottom; repetitive horizontal lines show 1 sec periods of time and the bars beneath the records show 0.5° of arc on both horizontal (H) and vertical (V) meridians.
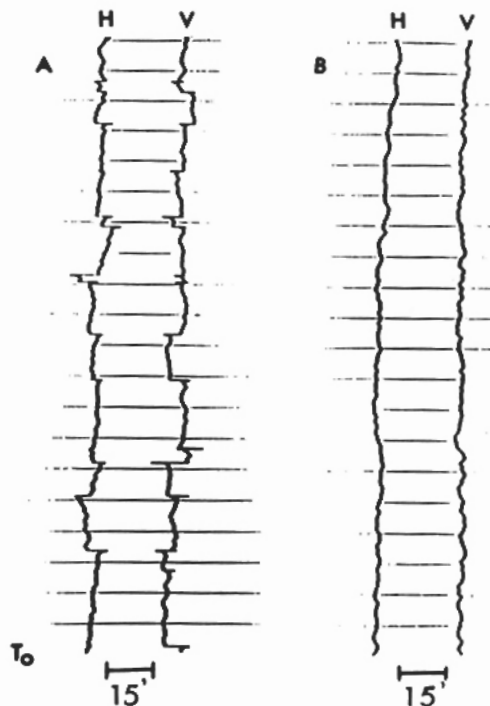

FIG. 4. (A) A representative two-dimensional record of human fixation. (B) A representative two-dimensional record of human slow control. Both records begin at the bottom (T_0); repetitive horizontal lines show 1 sec periods of time and the bars show 15 min of arc on both horizontal (H) and vertical (V) meridians.
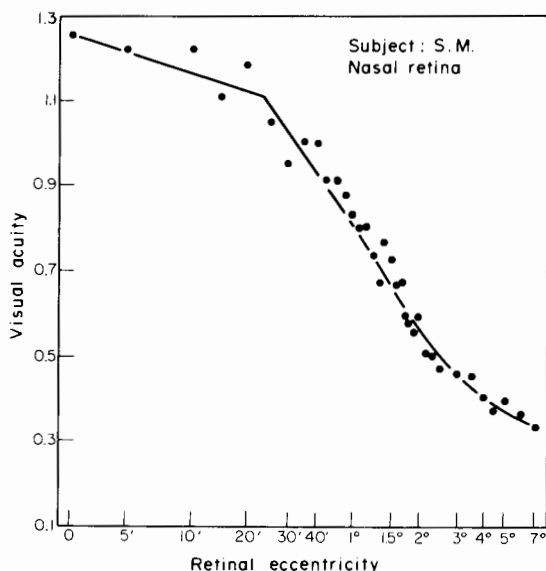
FIG. 5. Visual acuity as a function of retinal eccentricity. The abscissa is on a logarithmic scale. (The curve is drawn by inspection.) Taken from Millodot (1966).

is shown in Fig. 5 where one of Millodot's graphs of visual acuity as a function of retinal eccentricity is reproduced. Acuity dropped only about 25% as the test target moved from the preferred fixation position out to the edge of the foveal floor—about 40 min of arc away from the center. The 5 min of arc saccades made by human beings during maintained fixation seem much too small to be required for visual acuity. Particularly, since the fixation target is typically a point of light in darkness. Such targets appear much the same anywhere on the foveal floor.[5]

EYE MOVEMENTS USED TO ENHANCE OR TO MAINTAIN VISIBILITY

I will postpone speculation about this pathology and will now briefly review research that relates specific features of the human fixation pattern to visual processes. This research asks how miniature eye movements (micro-saccades, drifts, and physiological nystagmus) contribute to vision once a target is brought to the central fovea.

About 50 years ago Weymouth proposed that physiological nystagmus provided the basis of visual acuity. This proposal was developed quantitatively by Marshall and Talbot and Jones and Higgins in the 1940s. By the early 1950s Riggs and Ditchburn and their co-workers had shown that physiological nystagmus was too small to make a dynamic theory workable—tremor does not sweep the light distribution back and forth across any reasonable number of cones. Shortly after, we found out that small high-frequency displacements of the retinal image (by high frequency I mean frequencies above 8 Hz) actually have adverse affects on visual acuity. This was shown by moving targets that were stabilized on the retina by a variety of optical devices. Frequencies around 2 or 3 Hz prevent the disappearance of a stabilized image. This result suggests that drifts of the kind observed during slow control are used to maintain high-quality
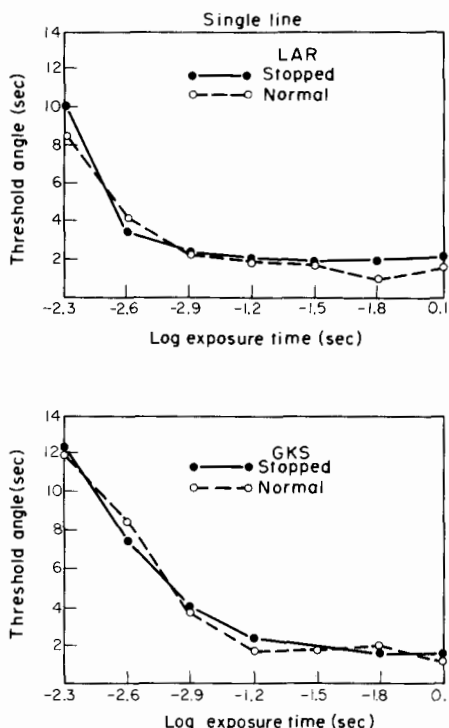
FIG. 6. Threshold curves for detection of single black lines as a function of exposure time under normal and stopped image conditions of viewing. Taken from Riggs (1965) who reproduced it from Keesey (1960).

vision. They are not, however, essential to vision as it is normally used. This can be seen in Fig. 6 where Keesey's data from a study on the effect of exposure duration on the minimum angle resolved is reproduced. Acuity was the same under both normal and stabilized viewing—even after exposure duration was long enough for acuity to be as good as it can be. Acuity is known to deteriorate when a stabilized image begins to fade but fading takes a second or longer—much longer than we normally use to process visual input. For example, we make three or four saccades each second when we read. Keesey's result suggests that we would see very well during reading pauses even if the eye did not move at all.[6]

EFFECTS OF MOVING THE EYE ON VISUAL THRESHOLDS

Not all eye movements maintain visibility. Drifts do. But saccades are different. Although required to place an attended target at the foveal center, they have adverse effects on visibility. These adverse effects, however, seem to be beneficial to the organism because they reduce blurring that would be caused by saccades when they jump the visual array across the retinal surface.

This observation was made long ago. The first report of what is currently called "saccadic suppression" was made by Erdman and Dodge in 1898 who noted that words that were recognized in the periphery during reading pauses could not be seen

during saccades that shifted the line of sight to another position in the text. Initially, Dodge thought that there was an anesthesia during saccades but by 1900 had rejected this notion because he noticed that with careful attention it is possible to see stimuli during a saccade. They are not invisible—only faint. Dodge suggested that the more intense stimulation seen following the saccade made the faint stimulation visible during the saccade barely perceptible. In this, Dodge seems to be anticipating a backward masking (or metacontrast) explanation—a phenomenon that would be reported 12 years later. Holt rejected this central explanation and proposed that impulses from the extraocular muscles blocked the visual afferent message during the saccade. Woodworth questioned both explanations because the visual world looks the same during a saccade as it does when the visual field is moved rapidly and the eye remains stationary.
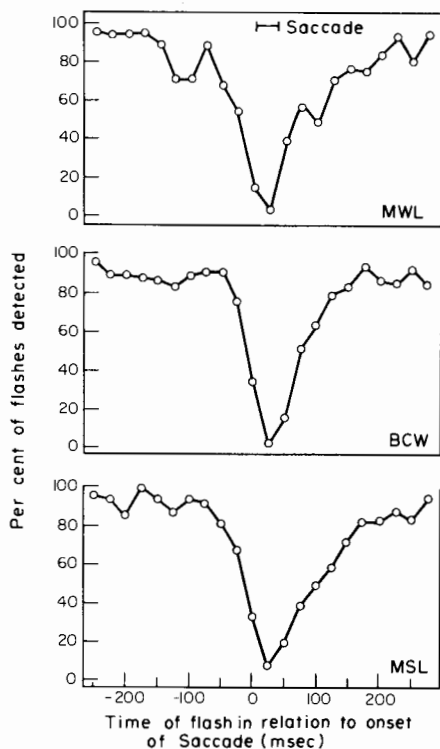

Fig. 7. Percentage of flashes detected by each of three subjects as a function of the temporal relation of the flash to the onset of a saccade. The average duration of the saccade is shown at the top of the figure. Taken from Volkmann et al. (1968).

Interest in this problem was rekindled by Ditchburn in the mid-1950s. Since then several mechanisms have been described and supported experimentally. The phenomenon is interesting because it cannot arise simply from the blurring of images during saccades—suppression can be shown with flashes too brief to allow blurring. It is also interesting because the threshold elevation precedes and follows the actual saccade. A typical result under photopic light conditions is shown in Fig. 7 where it can be seen that the hit-rate falls for a test flash delivered from 30 ms before to 50

ms after the saccade begins. Such results have been interpreted by Volkmann and others as evidence for central inhibition which begins when the saccade is programmed.

However, similar results are obtained when the eye remains stationary and the visual field is displaced rapidly. This result has led MacKay and a number of others to propose that backward masking is sufficient to explain the threshold elevation. Still other mechanisms have been proposed and supported. For example. Richards suggests that saccades set up shearing forces in the retina that introduce noise signals into the visual system. These signals are large relative to the ordinary visual afferent message and therefore travel rapidly toward the cortex arriving in time to reduce the detectibility of visual afferent signals sent before the saccade. Similarly, new shearing forces at the end of the saccade reduce detection during and sometimes after the eye movement is over.

We have then smear, shear, masking, and central inhibition as possible explanatory mechanisms. Most recently, Riggs and co-workers have shown that masking cannot be the whole story. They found that electrical phosphenes were suppressed by saccades made in a totally dark environment. There are at least two problems remaining. How many of these proposed mechanisms actually contribute to the threshold elevation and what is their relative contribution? Note, that although there is one report of a threshold elevation as large as 1.5 log units, it did not control for the retinal position of the test target. The usual result is only 0.3 to 0.4 of a log unit. At this point I see no basis for favoring one explanation over the others and propose as an impartial non-expert that, in the interest of fairness to a large number of distinguished investigators, we ascribe slightly less than 0.1 of a log unit to each mechanism.[7]

Later I will consider the importance of this phenomenon for position constancy but let me point out here that saccadic suppression, despite its small size, may be significant in considering the contribution of eye movements to vision. Note that it is a suppression and not an enhancement. We have seen that small slow drifts are required to maintain vision. Here we see that saccades have an opposite effect. They raise thresholds before, during, and after their occurrence. Occasionally, it is proposed that saccades produce significant visual information. Information that is used for seeing. The phenomenon I have just described suggests that quite the reverse may be true. This becomes important in looking for the significant visual message in the nervous system. It would seem safest to look only at neural events that precede and follow saccades by about 50 ms if you want to find the electrophysiological correlates of vision in a freely moving eye. Or, if working with an immobilized eye, the most natural input would be a small irregular drift-like movement of the stimulus rather than a sudden jump that simulates a saccade.[8]

EFFECTS OF EYE MOVEMENTS ON PERCEPTION

Next I will turn to how eye movements influence the way we perceive. We need to know a good deal more about the visual world than can be gleaned from a single foveal view. This view presents only a tiny fraction of the visual array with clarity. We must know the shapes of things, whether they are stationary or moving, and

where they are in respect to ourselves. Movements of the eye play an important role in piecing together this kind of information.

First, let us consider the role of eye movements in the perception of shape and size—an area in which eye movement theories have appeared on and off for more than 100 years. The most radical of these eye movement (or motor) theories hold that the eye-movement pattern is determined by the shape of the visual stimulus and that perception of shape is constructed from the sensations that accompany the eye movements used to inspect the stimulus. This kind of theory was proposed by Wundt (the man often considered to be the founder of modern scientific psychology).

Traditionally, such theories are supported by showing that eye movements are larger on the perceptually longer side of the Müller–Lyer illusion. Attacked by showing that the Müller–Lyer illusion is vivid when presented tachistoscopically, and revived by showing that the illusion becomes smaller when scanned repeatedly. This entire cycle had been completed by 1912 when interest in motor theories abated as Gestalt Psychology became ascendant in perception. Motor theories were revived by Hebb in 1949, who suggested that the perception of shape by adults was based, in part, on implicit as well as actual eye movements. This is a clever business. It eliminates the problem raised by the fact that an adult can see forms without actually making eye movements. Hebb also avoids the problem of awareness of the eye movement pattern by explaining perception on the basis of neurophysiological processes rather than sensations. He calls these processes "cell assemblies" and "phase sequences". Terms that refer to neural traces of corner analyzers and their connecting circuits. Both are built up slowly in childhood by using eye movements to look around. Such traces eventually allow the child to discriminate forms without actually moving his eye. By 1960 interest in this theory had waned because psychologists, speculating about neurological processes, turned to Hubel and Wiesel who had begun to describe the actual analyzers present in the visual cortex. Hebb's theory was weakened considerably by the fact that infant animals had the same hardware as their parents.

In the mid-1960s Festinger revived interest by proposing a motor theory that does not make the empiristic assumptions so prominent in Hebb's theory. Festinger's theory, like Hebb's, is not an overt motor theory. It links perception to programming rather than executing eye movements but it avoids Hebb's empiricism. It does not assume that these programs could not be hard-wired with their templates given by the genetic code. The theory seems timely because it borrows terminology from von Holst and talks of "efferent readiness" and "efferent copies" when it refers to physiological processes. So far support comes primarily from the study of visual illusions where some suggestive results have been obtained. The Müller–Lyer illusion is reproduced in Fig. 8. The reader can scan the illusion for several minutes and judge for himself how much the illusion is reduced by prolonged inspection.[9]
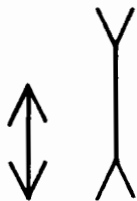

FIG. 8. The Müller–Lyer Illusion. The vertical line segments are the same length.

Next I would like to consider how we know where things are in space relative to ourselves. (By ourselves I mean the mid-saggital plane of the upper torso or the straight-ahead of the body image if you prefer psychological to anatomical terms.) To know where things are we must know the orientation of the head with respect to the body, the orientation of the eye with respect to the head, and where the object of interest falls on the retina. We have all this information but there is some question as to its source. The visual afferent message gives us the distance and direction of a visual object with respect to the center of the fovea. But how do we know the orientation of the fovea with respect to the head? (The third problem, how we know where the head is with respect to the body, I will leave to Professors Bizzi and Abrahams.)

Two schemes have been proposed. The most influential has been Helmholtz's who said that we know the direction the eye is pointing because we know the effort of the will we used to orient the eye. We not only know what we told the eye to do, we can remember what we told it to do for a considerable period of time. The alternative scheme, proposed by Sherrington, assumes that the large number of mechanoreceptors found in extraocular muscle tell the human how his eye is oriented in the orbit. This is sometimes called an "inflow" theory.

Helmholtz supported his view (sometimes called "outflow") by pointing out that passive displacements of the eye cause shifts of the positions of objects in space. He also pointed out that impeded attempts to move the eye lead to a loss of position constancy—objects appear to shift in the direction of the attempted but blocked eye movement. These elegant demonstrations have been interpreted as showing that the eye is devoid of a position sense—only knowing where it is because of the monitored and stored efferent command.

There have been developments in the past few years which show that the story is not this simple. Skavenski, who did this work, was probably the first to note the danger of making inferences about the oculomotor system from experiments that were concerned with the perception of direction rather than with the control of eye position itself.

He performed an experiment in which subjects used inflow to control the position of the eye in the absence of any other information. The way this was done and the results of the experiment are shown in Figs. 9 and 10. Note that both subjects began to correct eye position as the load was being applied. This means that very small proprioceptive signals can be used to maintain the position of the eye in the orbit.

Skavenski also used a forced-choice psychophysical procedure to show that the application and direction of the load could be sensed. This result conflicts with a report of Brindley and Merton who were not able to sense the position of their eyes. These authors probably had difficulty for two reasons. They used an insensitive psychophysical method and a distracting technique for displacing the eye. Grasping the eye with forceps could mask the relatively subtle but easily sensed feelings that are produced by passive displacements.

Next Skavenski and Haddad investigated the relative contribution of outflow and inflow to the perception of direction and quantified Helmholtz's observation that changing the efferent command to maintain eye position against a load causes shifts in the perceived location of the fixated target. They also put the outflow signal in conflict

FIG. 9. Subject RS in position in the apparatus used to record horizontal eye movements while loading his right eye. RS is biting on an acrylic-bite board which holds his head rigidly in place. Horizontal rotations of his right eye were recorded by means of an infrared light transmitting and a collecting fiber optic mounted on the microscope stage just to his right. Loads were applied by adding water to the plastic chambers shown below the pulleys on the left and right. Loads were transmitted to the eye by means of the dacron threads that pass over the pulleys and connect to the 3-cm stalk attached to the scleral contact-lens. The contact-lens was held firmly to the eye by suction applied through the thin polyethylene tubing that can be seen as it passes up over the bridge of the nose and, again, as it passes in front of the left eye patch on its way to the suction apparatus (not shown). The suction tubing was arranged so that it did not, at any time during these experiments, touch the nose or face at any place except where it was taped to the forehead near the hairline. This contact with the skin could not have provided eye position information because the lateral displacement of the end of tubing at this junction with the contact-lens was too small and the tubing too flexible to produce any tactile cues on the forehead. With this apparatus loads of various magnitudes could be applied to the right eye gradually and completely silently. Taken from Skavenski (1972).

with the inflow signal and found that a conflicting inflow signal did not influence the perceived direction of the fixated target. Taken together, these results show that some non-visual afferent can be used to control eye position. It can be sensed but does not change where things appear to be in space when its message conflicts with the efferent command.

Recently there has also been work by the Matins and co-workers who measured what happens to position constancy during saccadic eye movements. They found that the map of retinal position with respect to perceived direction shifts in rather complicated ways. The shift begins about 130 ms before the saccade and continues after the eye movement is completed. During this period subjects have difficulty reporting the spatial location of a test target. Eventually objects appear to be appropriately
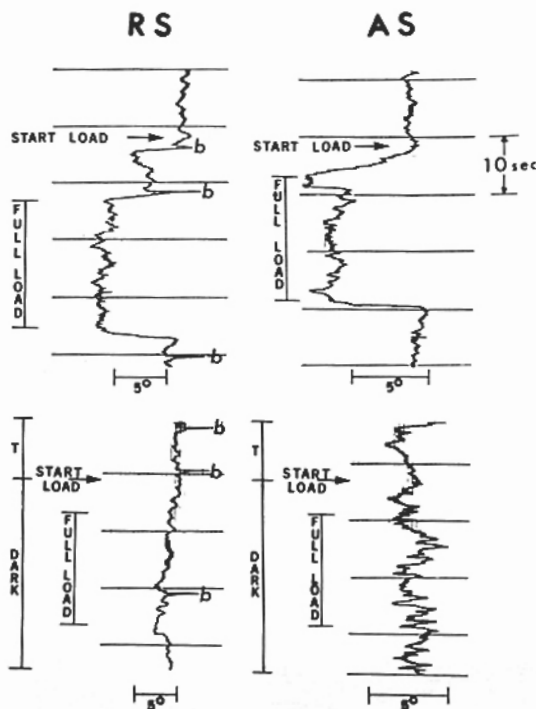
FIG. 10. Representative records of RS's and AS's horizontal eye-position control when loads were applied to their right eyes. In the upper records Ss fixated the visible target with their left eyes. Right eyes were occluded. Application of the load began at the time indicated by the arrow head and the verical bar on the left indicates the period of time the *full load* was applied. The final seconds of each record show eye position returning to baseline as the load was removed. The two lower records show RS's and AS's ability to correct eye position when loads, equal to those applied in the upper records, were applied during the dark period. For these records S's right eyes were uncovered and their left eyes were closed and covered. Records begin with 10 sec of fixation of the visible target (T). The target was switched-off at the time indicated by the arrow and the remainder of the record shows eye position control in the dark (*DARK*). Application of the load began just after the time indicated by the arrow and the vertical bar shows the period the *full load* was applied. The load was removed during the final seconds of each record. Pulse-like changes in the trace, labeled *b*, are blinks. Horizontal bars beneath each record show 5° arc rotations on the horizontal meridian. Taken from Skavenski (1972).

placed but this takes about 400 ms. Note, however, that saccadic suppression occurs during the same period. This has lead Ethel Matin to suggest that saccadic suppression, despite its modest size, has functional significance. It attenuates perception of the poorly localized visual world while a saccade is programmed and made. But this disturbance of visual localization only applies to the perception of direction, not to the control of eye position. Hallett and Lightstone have shown recently that the oculomotor system is not confused during saccades. It processes information about a change in the position of a fixation target that is displaced during a saccade with a high degree of accuracy. Here, once again, we see that our oculomotor system can use information that differs from what we perceive. There seems to be no simple correspondence between what the little engineer in the brainstem sees and the perceptions of the little man in the cortex who tells us what is really out there in the visual world.[10]

WHAT *IS* THE ROLE OF EYE MOVEMENTS IN VISION?

This completes my review of highlights from the laboratory where the effect of eye movements on visual and perceptual processes has been studied extensively during the past 25 years. The impression one gets from most of the visual research is that eye movements are fundamental. They establish and maintain visibility. Their role in perception is less clear but interest continues and it seems only a matter of time before we know how we perceive as well as how we see.

There is, however, something disturbing about how we interpret much of this painstaking research. We tend to talk as though we believe that the eye moves because image motion is required to drive many of the visual neurons. Neurons that respond to changing rather than constant stimulation. This seems to suggest that eye movements evolved to drive these neural elements. I think that quite the reverse is true. It seems more likely that the oculomotor system evolved to stabilize retinal images because the retinal image is displaced by everything we do. Our ordinary activities place an enormous burden on the oculomotor system. Just how large this burden is had not been studied which encouraged Winterson, Robinson, Skavenski, and I to begin to look at what the eye actually does in natural circumstances—circumstances in which the head is not held rigidly in place. How this was done is shown in Fig. 11 which

FIG. 11. Skavenski in position for simultaneous two-dimensional recording of rotations of his head and right eye by means of the magnetic field search coil technique. The eye search coil is embedded in a tight-fitting scleral contact lens held to the eye by suction. The head search coil is mounted to a dental bite plate. The field coils can be seen surrounding the subject who is looking at a target at optical infinity.

shows Skavenski rigged-up to use Robinson's search coil technique to make simultaneous recordings of the rotations of his head and eye while he attempts to sit as still as possible and maintain his line of sight on a fixation target. Figure 12 shows a record of both eye and head rotations. There was a great deal of head rotation and the eye was very busy trying to compensate for these rotations.
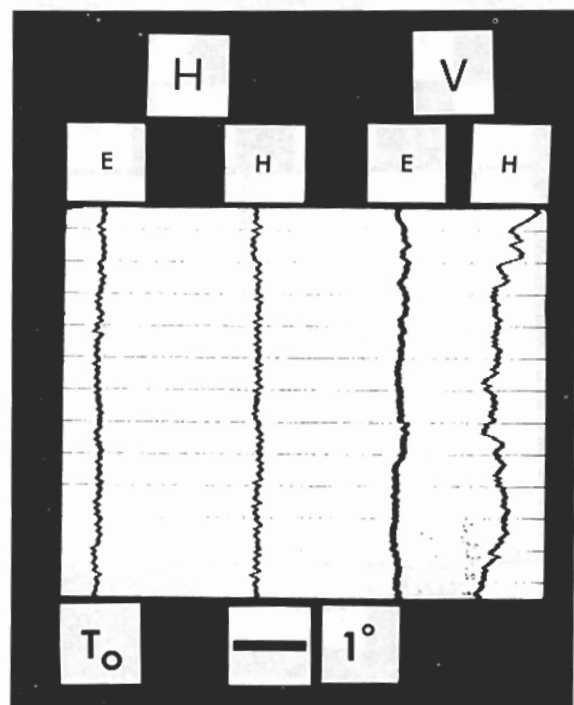

FIG. 12. A representative simultaneous eye (E) and head (H) recording of rotations on the horizontal (H) and vertical (V) meridians made while the subject sat as still as possible. The record begins at the bottom (T_0); the repetitive horizontal lines show 1 sec periods and the bar shows 1° of arc for both eye and head traces.

We have not analysed these records and are not yet prepared to say whether all eye movements other than voluntary saccades are made in response to rotations and translations of the head signalled either by its orientation, acceleration, or by displacements of the retinal image. But, what with rotations caused by breathing (which have a peak to peak amplitude of about half of a degree), the pulse (which requires about 10 min of arc compensation every second or so), and oscillations in the servo working to hold the head steady (which have a frequency of about 3 to 5 Hz and an amplitude of about 10 min of arc), the eye is kept quite busy trying to maintain the target image in the center of the fovea.

Can a subject do better than this under the most ideal conditions—when the chin is supported by the hands and the breath is held? Both of these acts are in the normal behavioral repertoire. They might be used to steady the head while trying to pay close attention to some detail in the visual world. Figure 13 shows eye and head traces when Skavenski supported his head, held his breath, and tried to keep

his eye on a visual target. There was still a great deal of activity. Such activity is not confined to Skavenski's head. His rotations are not early symptoms of Parkinsonism. We all have this problem. The head rotations of five other subjects have been recorded. They all moved as much no matter how hard they tried to keep still.[11]
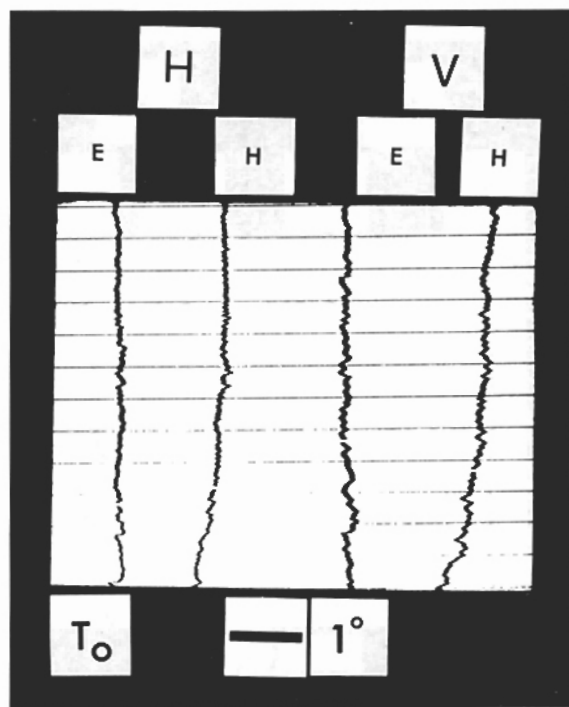

FIG. 13. A representative simultaneous eye (E) and head (H) recording of rotations on the horizontal (H) and vertical (V) meridians made while the subject sat as still as possible while supporting his head on his hands and held his breath. The record begins at the bottom (T_0); the repetitive horizontal lines show 1 sec periods of time and the bar shows 1° of arc for both eye and head traces.

These data suggest that there may have been too much emphasis on the importance of eye movements for guaranteeing retinal image motion in recent years. The retinal image is in no danger of standing still if the head is not attached to a rigid metal plate. It seems likely to me that the oculomotor system evolved to compensate for such disturbances in orientation of the eye—disturbances that are inescapable outside of the laboratory. The problem for man and other foveate animals is colossal. We must not only compensate for bodily movements we must also adjust the orientation of the fovea so that we can see attended objects. We also must be able to track smoothly so that attended moving objects can be kept relatively stationary within this tiny specialized region. A great deal of machinery must develop to allow these tricks. We also need more than the ability to stabilize and swing the eyeball. We need considerable cognitive capacity to construct a perceptually meaningful world from the sequence of small snapshots that we typically acquire in a haphazard order. We also need selective attention to acquire what we need to know when we need to know it. We also need short- and long-term memory to put this material together

FIG. 14. A chameleon.

and to hold on to it long enough to know where we are relative to where we have been. In short we need a human brain.

It is tempting to propose that man's high degree of perceptual and cognitive development arose in his evolutionary effort to compensate for problems inherent in foveal specialization—or "hereditary localized hyperplasia" as I called this burden earlier. I find this an attractive way to think about the development and role of the oculomotor system in visual processing. It makes this system seem terribly important. I suspect, however, that not everyone will share my enthusiasm for this approach particularly since it is probably not without shortcomings. This can be seen in Fig. 14 which shows a chameleon—an animal with a high degree of foveal specialization. Its pit is deeper and more densely packed than man's. I know almost nothing about reptilian brains and even less about reptilian behavior. Is this lizard really as wise as she looks?[12]

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NOTES

1. This article is an abridged version (fewer figures) of the paper read in Stockholm. The assigned topic was very broad. It covered several areas of visual and perceptual research. Any one of these areas could easily fill a chapter in this volume. Each has been reviewed in recent years and is discussed in current textbooks on visual and perceptual processes. For this reason I decided to publish my lecture as it was given and add notes to provide the unspecialized reader with additional information and some of the sources that influenced my thinking. See Ditchburn (1973) and Festinger (1971) for alternatives to my approach to this topic. See Walls (1962) for a similar approach to mine written before slow control had been demonstrated in man.

2. The primate retina (and man's in particular) shows a great deal of foveal specialization. The classical source for its structure is Polyak (1941). I chose this characteristic to organize the lecture—inventing an imaginary disease to make the presentation consistent with the title of this symposium. This specialization is not only anatomical. A good deal of differential function has also been demonstrated psychophysically—a fact that makes this "disease" plausible. My emphasis on specialization is old-fashioned, however, because it has become popular recently to use transfer functions to describe the visual system—a methodology that is easier to use if the functional properties of the human retina are treated as though they were relatively uniform across large regions. (See Cornsweet, 1970, for an introduction to the use of the transfer function to study the spatial resolving power of the retina.) I have chosen to be old-fashioned because the human scan pattern is easier to understand when the retina is viewed as exceedingly heterogeneous. I am not at all convinced, however, that normal humans actually have the kind of tunnel vision my

"disease" implies. Phenomenologically the visual world seems to have excellent form and color vision throughout. The extent to which this compelling subjective impression is constructed by memory through serial processing of foveal snapshots is by no means worked out. See Monty and Senders (1974) for a recent discussion of this problem at a specialists' meeting on the role of eye movements on psychological processes. It is my impression that at this time it is as reasonable to believe that eye movements are used to confirm already organized percepts as to believe that eye movements are used to piece together the visual world from a series of foveal snapshots.

3. Winterson (1974) described the normal visual exploration of the cat in greater detail recently and a complete report of this material is in press (Winterson and Robinson). Some of her results do not agree with the other published studies of the cat's fine-grain oculomotor performance (Hebbard and Marg, 1960; Pritchard and Heron, 1960). Winterson found that the cat, unlike ourselves but like untrained monkeys, never makes microsaccades (flicks smaller than 10 min of arc). Occasional microsaccades had been reported by Hebbard and Marg (1960). Winterson never observed any in several thousand seconds of recording and concluded that the prior report of microsaccades was a misinterpretation of the cat's eye movement pattern. Hebbard and Marg only recorded on the horizontal meridian. Winterson's two-dimensional recordings showed that "microsaccades", observed occasionally on one meridian, were actually components of rotation associated with large saccades on the other meridian. Pritchard and Heron (1960) reported that their cats' intersaccadic drifts were rapid and uncompensated. It is not clear why these authors failed to notice slow control—the most striking feature of feline oculomotor performance. It might have been because their cats were drowsy following the administration of anesthesia. A drowsy cat, like a drowsy man, drifts all over.

The fine-grain characteristics of slow control in the cat are very much like those of the rabbit who has been extensively studied by Collewijn and co-workers (1969, 1970, 1972). The rabbit has no fovea, does not show fixation saccades, and uses slow control exclusively to maintain eye position. Slow control in the rabbit, like the cat and man (Skavenski and Steinman, 1970), is lost when visual stimulation is not available.

4. Recently, Skavenski (1974b) reported these characteristics and described the training procedures for establishing them. A description of earlier phases of his work can be found in Steinman, et al. (1973). This article also contains description of the discovery of slow control in the human and a summary of several lines of evidence supporting the notion that the miniature saccades, typically seen in the fixation pattern of adult humans, represent an overlearned motor skill unique to man and probably without any useful visual function.

5. I chose to use Millodot's (1966) data for the VA/eccentricity function in the fovea because these measurements were made very carefully under conditions where the exact placement of the test target could be known. Millodot (1972) finds that, in general, the isoacuity area is relatively large in the central fovea (ranging from 24 to 50 min arc depending on the type of target). Other authors consider this function to be steeper. For example, LeGrand (1967, p. 136) draws his curve summarizing data gathered from a number of experimenters such that acuity seems to fall off very rapidly as tests are made small distances from the fixation point. Many of the data points, however, seem consistent with Millodot's measurements. I would prefer to believe that acuity is relatively uniform on the foveal floor because it makes it easier to understand the fixation stability/eccentricity function. See Timberlake et al. (1972) for a discussion and data that bears on this problem.

6. There is a very large literature on the role of eye movements in visual processes studied by means of the stabilized image technique. See Alpern (1972) for a recent discussion of the current status of such research and Weymouth et al. (1923) for descriptions of the "dynamic" theory of visual acuity that inspired much of the subsequent stabilized image research. Also, see Ditchburn (1973), Riggs (1965), and Yarbus (1967) for summaries of research done in the laboratories most active in studying this problem during the 1950s and 1960s (much of this material was reviewed by Heckenmueller, 1965). See Gerrits and Vendrik (1970, 1972, 1974) for current work by very ingenious investigators and Arend (1973) for a theoretical paper that imputes a very large role to monitored eye movements in the processing of contours, brightness, and color.

The stabilized image technique has also been used to study visual perception. Here I am referring to the work of Pritchard et al. (1960) and Pritchard (1961) who reported meaningful perceptual fragmentation of stabilized complex stimuli. These results received widespread attention probably because they bridged the gap between vision and visual perception with material that could be readily described in introductory psychology texts. Many of us suspected at the time of the initial reports that these perceptual fragments might not represent visual organizing processes revealed by stabilization because direct phenomenal report was used in these studies. Such reports are easily biased. The frequent reappearances of the stimuli also suggested that the stabilization was poor as well. Over the ensuing years a number of authors have expressed such concerns (e.g. Cornsweet, 1970, p. 408), and recently Shuck and Leahy (1966) and Shuck (1973) have shown that similar phenomenal reports are obtained without stabilized images when subjects describe ambiguous figures verbally. As matters now stand it seems parsimonious to assume that constraints of language

determine the way ambiguous figures will be described and that most, if not all, of the perceptual effects reported by Pritchard are based on cognitive rather than visual processes.

7. I am greatly indebted to Ethel Matin (1974b) for allowing me to read a preprint of her review on saccadic suppression. This excellent article analyzes methodological problems and evaluates theoretical issues in this area of research. It also provides a very complete bibliography of the large number of publications this problem has generated over the past 75 years.

8. See Fischer and Krüger (this volume) for an example of an electrophysiological study where I believe this caution is warranted. The electrophysiological responses produced by saccade-like movements of a stimulus outside of the receptive field do not seem likely to provide significant visual information to the cat. The barrage of activity associated with these step displacements might, however, be the neurological basis of or produce the need for a saccadic suppression mechanism.

9. See Murphy *et al.* (1974) for a brief discussion of motor theories of form processing. These authors found that simple forms, confined to the foveal floor, do not constrain the oculomotor pattern. The problem is still open for extra-foveal forms whose shape can not be seen without using saccades to scan the display. I believe that the oculomotor system provided with photopically effective stimulation is largely free from stimulus constraints. See Steinman (1974) for a summary of evidence supporting this view. Most of the reported effects of eye movements on form perception or visual illusions are very small and subject to alternative interpretations. See Bolles (1969), Boyce and West (1967, 1968), Delabarre (1897), Festinger *et al.* (1968), Festinger (1971), Hebb (1949), Judd (1905), McLaughlin *et al.* (1969), Lewis (1908), Lötze (1852), Mollon (1968), and Wundt (1910) for some representative material on the role of eye movements in the perception of form and illusions of visual extent.

Some comment about the relationship of Hubel and Wiesel's (1962) observations and Hebb's (1949) motor theory of form perception is necessary. For Hebb all percepts more complicated than "primitive unity" (a segregation of figure from ground that has no shape) are built-up empirically by the infant as it scans the visual field. The first constructions are line, slope, and corner detectors. These are subsequently connected by the eye-scan pattern. The infant's eye tends to look from corner to corner. These repeated visual stimulations establish "phase sequences" the physiological correlate of the perceived form. Hubel and Wiesel's (1962) demonstration of orientation specific cortical units is, as they point out, suggestive of the elements that might, working together, form corner analyzers a notion similar to the kind of cortical organization Hebb suggested. However, these units, according to Hebb, should not be present without prior coordinated visual and oculomotor experience. This seems not to be the case according to Hubel and Wiesel (1963). The most distinctive feature of their results with newly sighted kittens is the demonstration of pre-wired functional arrangements similar to their parents'. The most distinctive feature of Hebb's theory, on the other hand, is its emphasis on empiricism. Eye movements are used to construct percepts which do not exist without prior "learning". I find it hard to reconcile Hebb's theory with Hubel and Wiesel's conclusion. The problem for the newly sighted kitten seems to be visuomotor (they do not track targets or avoid obstacles) rather than visual if the Hubel and Wiesel cells are responsible for the perception of form an assumption that is open to question (Blum, in preparation). Even the Hubel and Wiesel conclusion with the kitten is open to question (Blakemore and Cooper, 1970; Spinelli *et al.*, 1972) and it may be some time before we know enough about how form is abstracted in the visual system to be able to find out how or if the oculomotor system contributes (see Blum, 1973, for a recent discussion of alternative models for form perception).

10. See Skavenski and Steinman (1970), Skavenski (1971, 1972, 1974a), and Skavenski *et al.* (1972) for recent work on oculomotor inflow and references to prior work. See Matin (1972) for a description of the work of his group on eye movements and perceived direction and Ethel Matin (1974a) for a discussion of the importance of saccadic suppression for the maintenance of position constancy during saccades. See Hallett and Lightstone (in preparation) and Jurgens and Becker (this Symposium) for evidence that the saccadic system processes information during the period that perception of direction is disturbed. Also, see MacKay (1970, 1973) for evidence that shifts in apparent spatial location can be induced by passive as well as active displacements of the retinal image and recent discussion of the role of the eye movements in the perception of spatial location.

11. See Steinman (1974) for a quantitative treatment of the rotational components of the head movements of five subjects. See Troelestra (1972) for measurements of the effects of normal physiological activities on motions of the eyeball.

12. A number of problems that relate eye movements to visual or perceptual processes were not included because of time (space) limitations. See Royce *et al.* (1966) and Levy (1972) for an evaluation of the methodology and bibliography on autokinesis an illusion that has been studied for more than a century and Crone and Verduyn Lunel (1969) and Matin and McKinnon (1964) for particularly interesting modern experimental papers that relate this phenomenon to the eye movement pattern. I also omitted consideration of the role of eye movements in the perception of motion (see, for example, Gibson, 1968; Kinchla and Allan, 1969; Orban *et al.* 1973 for representative papers) and also their role in determining the perceived orientation of the visual field (see, for example, Howard and Templeton, 1964; Petrov and Zenkin, 1973).

REFERENCES

ALPERN, M. (1972) Eye movements. In: *Handbook of Sensory Physiology*, vol. VII/4, JAMESON, D. and HURVICH, L. (Eds.). Springer-Verlag, New York.

AREND, L. E. (1973) Spatial differential and integral operations in human vision: implications of stabilized retinal image fading. *Psychol. Rev.* **80**, 374.

BLAKEMORE, C. and COOPER, G. F. (1970) Development of the brain depends on the visual environment. *Nature*, **228**, 477.

BLUM, H. (1973) Biological shape and visual science (Part 1). *J. Theor. Biol.* **38**, 205.

BLUM, H. (1975) Biological shape and visual science (Part 2). *J. Theor. Biol.* (in preparation).

BOLLES, R. (1969) The role of eye movements in the Müller-Lyer illusion. *Percept. & Psychophys.* **6**, 175.

BOYCE, P. R. and WEST, D. C. (1967) A perceptual effect on the control of fixation. *Optica Acta*, **14**, 119.

BOYCE, P. R. and WEST, D. C. (1968) Reply to "Fixation and Perception". *Optica Acta*, **15**, 299.

COLLEWIJN, H. (1969) Optokinetic eye movements in the rabbit: input-output relations. *Vision Res.* **9**, 117.

COLLEWIJN, H. (1970) The normal range of horizontal eye movements in the rabbit. *Exptl Neurol.* **28**, 132.

COLLEWIJN, H. and VANDERMARK, F. (1972) Ocular stability in variable feedback conditions in the rabbit. *Brain Res.* **36**, 47.

CORNSWEET, T. N. (1970) *Visual Perception*. Academic Press, New York.

CRONE, R. A. and VERDUYN LUNEL, H. F. E. (1969) Autokinesis and the perception of movement: the physiology of eccentric fixation. *Vision Res.* **9**, 89.

DELABARRE, E. B. (1897) A method of recording eye movements. *Am. J. Psychol.* **9**, 572.

DITCHBURN, R. W. (1973) *Eye Movements and Visual Perception*. Oxford Univ. Press, New York.

FESTINGER, L. (1971) in *The Control of Eye Movements*, BACH-Y-RITA, P. and COLLINS, C. C. (Eds.). Academic Press, London.

FESTINGER, L., WHITE, C. W. and ALLYN, M. R. (1968) Eye movements and decrement in the Müller-Lyer illusion. *Percept. Psychophys.* **3**, 376.

GERRITS, H. J. M. and VENDRIK, A. J. H. (1970) Artificial movements of a stabilized image. *Vision Res.* **10**, 1443.

GERRITS, H. J. M. and VENDRIK, A. J. H. (1972) Eye movements necessary for continuous perception during stabilization of retinal images. *Bibl. Ophthal.* **82**, 339.

GERRITS, H. J. M. and VENDRIK, A. J. H. (1974) The influence of stimulus movements on perception in parafoveal stabilized version. *Vision Res.* **14**, 175.

GIBSON, J. G. (1968) What gives rise to the perception of motion? *Psychol. Rev.* **75**, 335.

HALLET, P. E. and LIGHTSTONE, A. D. (1975) Saccadic eye movements due to stimuli triggered during prior saccades. (in prep.)

HEBB, D. O. (1949) *The Organization of Behavior*. John Wiley & Sons, Inc., New York.

HEBBARD, F. W. and MARG, E. (1960) Physiological nystagmus in the cat. *J. Opt. Soc. Am.* **50**, 151.

HECKENMUELLER, E. G. (1965) Stabilization of the retinal image: A review of method, effects, and theory. *Psychol. Bull.* **63**, 157.

HOWARD, I. P. and TEMPLETON, W. B. (1964) Visually induced eye torsion and tilt adaptation. *Vision Res.* **4**, 433.

HUBEL, D. H. and WIESEL, T. N. (1962) Receptive fields, binocular interaction and functional architecture in the cat's visual cortex. *J. Physiol.* **160**, 106.

HUBEL, D. H. and WIESEL, T. N. (1963) Receptive fields of cells in striate cortex of very young, visually inexperienced kittens. *J. Neurophysiol.* **26**, 994.

JONES, L. and HIGGINS, G. (1948) Photographic granularity and graininess. IV. Visual acuity thresholds; dynamic versus static assumptions *J. Opt. Soc. Am.* **38**, 398.

JUDD, C. H. (1905) Movement and consciousness. *Psychol. Monogr.* no. 34.

KEESEY, U. T. (1960) Effects of involuntary eye movements on visual acuity. *J. Opt. Soc. Am.* **50**, 769.

KINCHLA, R. A. and ALLAN, L. G. (1969) A theory of visual movement perception. *Psychol. Rev.* **76**, 537.

LEGRAND, Y. (1967) *Form and Space Vision*. Bloomington, Indiana Univ. Press.

LEVY, J. (1972) Autokinetic illusion: a systematic review of theories, measures, and independent variables. *Psychol. Bull.* **78**, 457.

LEWIS, D. O. (1908) The effect of practice on the perception of the Müller-Lyer illusion. *Brit. J. Psychol.* **2**, 294.

LÖTZE, R. H. (1852) *Medicinische Psychologie*. Reprinted by E. J. Bonct, Amsterdam, 1966.

MACKAY, D. M. (1970) Mislocation of test flashes during saccadic image displacement. *Nature*, **227**, 731.

MACKAY, D. M. (1973) in *Handbook of Sensory Physiology* JUNG, R. (Ed.), vol. **VII/3A**. Springer, Heidelberg and New York.

MCLAUGHLIN, S. C., DESISTO, M. J. and KELLY, M. J., JR. (1969) Comment on "Eye Movements and decrement in the Müller-Lyer illusion". *Percept. Psychophys.* **5**, 288.

MARSHALL, W. and TALBOT, S. (1942) Recent evidence for neural mechanisms in vision leading to a general theory of sensory acuity. *Biol. Symp.* **7**, 117.

MATIN, E. (1974a) Role of eye movements in maintaining a phenomenally clear and stable world. Presented at the specialists' meeting on eye movements and psychological processes. Princeton, New Jersey (to be published by NRC).

MATIN, E. (1974b) Saccadic suppression: a review and an analysis. *Psychol. Bull.* **81**, 899.

MATIN, L. (1972) in *Handbook of Sensory Physiology*, vol. VII/4, JAMESON, D. and HURVICH, L. (Eds.) Springer-Verlag, Berlin.

MATIN, L. and MACKINNON, G. E. (1964) Autokinetic movement: selective manipulation of directional components by image stabilization. *Science*, **143**, 147.

MILLODOT, M. (1966) Foveal and extra-foveal acuity with and without stabilized retinal images. *Brit. J. Physiol. Optics.* **23**, 75.

MILLODOT, M. (1972) Variation of visual acuity in the central region of the retina. *Brit. J. Physiol. Optics.* **27**, 24.

MOLLON, J. D. (1968) Fixation and perception. *Optica Acta*, **15**, 295.

MONTY, R. A. and SENDERS, J. W. (1974) Organizers of the specialists' meeting on eye movements and psychological processes. Princeton, New Jersey (to be published by NRC).

MURPHY, B. J., HADDAD, G. M. and STEINMAN, R. M. (1974) Simple forms and fluctuations of the line of sight: Implications for motor theories of form processing. *Percept. Psychophys.* **16**, 557.

ORBAN, G., DUYSENS, J. and CALLENS, M. (1973) Movement perception during voluntary saccadic eye movements. *Vision Res.* **13**, 1343.

PETROV, A. P. and ZENKIN, G. H. (1973) Torsional eye movements and constancy of the visual field. *Vision Res.* **12**, 2015.

POLYAK, S. L. (1941) *The Retina*. University of Chicago Press, Chicago.

PRITCHARD, R. M. (1961) Stabilized images on the retina. *Sci. Amer.* **204**, 72.

PRITCHARD, R. M. and HERON, W. (1960) Small eye movements of the cat. *Can. J. Psychol.* **14**, 131.

PRITCHARD, R. M., HERON, W. and HEBB, D. O. (1960) Visual perception approached by the method of stabilized images. *Can. J. Psychol.* **14**, 67.

RIGGS, L. (1965) Visual acuity. In: *Vision and Visual Perception*, GRAHAM, C. (Ed.). John Wiley & Sons, Inc., New York.

ROYCE, J. R., CARRAN, A. B., AFTANAS, M., LEHMAN, R. S. and BLUMENTHAL, A. (1966) The autokinetic phenomenon: a critical review. *Psychol. Bull.* **65**, 243.

SCHUCK, J. R. (1973) Factors affecting reports of fragmenting visual images. *Percept. Psychophys.* **13**, 382.

SCHUCK, J. R. and LEAHY, W. R. (1966) A comparison of verbal and nonverbal reports of fragmenting visual images. *Percept. Psychophys.* **1**, 191.

SKAVENSKI, A. A. (1971) Extraretinal correction and memory for target position. *Vision Res.* **11**, 743.

SKAVENSKI, A. A. (1972) Inflow as a source of extraretinal eye position. *Vision Res.* **12**, 221.

SKAVENSKI, A. A. (1974a). The nature and role of extraretinal eye position information. Presented at the specialists' meeting on eye movements and psychological processes. Princeton, N.J. (to be published by NRC).

SKAVENSKI, A. A. (1974b) Fixation eye movement in rhesus monkey. Paper read at ARVO, May 1974.

SKAVENSKI, A. A., HADDAD, G. M. and STEINMAN, R. M. (1972) The extraretinal signal for the visual perception of direction. *Percept. Psychophys.* **11**, 287.

SKAVENSKI, A. A., and STEINMAN, R. M. (1970) Control of eye position in the dark. *Vision Res.* **10**, 193.

SPINELLI, D. N., HIRSCH, H. V. B., PHELPS, R. W. and METZLER, J. (1972) Visual experience as a determinant of the response characteristics of cortical receptive fields in cats. *Exptl Brain Res.* **15**, 289.

STEINMAN, R. M. (1974) Role of eye movements in maintaining a phenomenally clear and stable world. Presented at the specialists' meeting on eye movements and psychological processes. Princeton, New Jersey (to be published by NRC).

STEINMAN, R. M., HADDAD, G. M., SKAVENSKI, A. A. and WYMAN, D. (1973) Miniature eye movement. *Science*, **181**, 810.

TIMBERLAKE, G. T., WYMAN, D., SKAVENSKI, A. A. and STEINMAN, R. M. (1972) The oculomotor error signal in the fovea. *Vision Res.* **12**, 1059.

TROELSTRA, A. (1972) Intraocular noise: origin and characteristics. *Vision Res.* **12**, 1313.

VOLKMANN, F. C., SCHICK, A. M. L. and RIGGS, L. A. (1968) Time course of visual inhibition during voluntary saccades. *J. Opt. Soc. Am.* **58**, 562.

WALLS, G. L. (1962) The evolutionary history of eye movements. *Vision Res.* **2**, 69.

WEYMOUTH, F., ANDERSEN, E. and AVERILL, H. (1923) Retinal mean local sign: a new view of the relation of the retinal mosaic to visual perception. *Am. J. Physiol.* **63**, 410.

WINTERSON, B. The way cats look. Paper read at ARVO, May 1974.

WUNDT, W. (1910) *Grundzüge der Physiologischen Psychologische*, 5th ed., vol. 2. W. Engelmann, Leipzig.

YARBUS, A. L. (1967) *Eye Movements and Vision*. Plenum Press, New York.