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DEPRESSION IN THE EXCITABILITY OF RELAY CELLS OF LATERAL GENICULATE NUCLEUS FOLLOWING SACCADIC EYE MOVEMENTS IN THE CAT

By HIROHARU NODA

From the Department of Anatomy and Brain Research Institute, University of California, Los Angeles, California 90024, U.S.A.

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SUMMARY

1. The excitability of relay cells of the lateral geniculate nucleus during a saccadic eye movement was studied in alert cats. Excitability was assessed by the firing probability of the cells in response to electrical stimulation of the optic chiasm. Modifications in the excitability were evaluated during the period following eye movements, by triggering a stimulator from potential shifts in electro-oculogram and altering delays in the stimulus pulse.

2. The cells were classified into S and T cells, based on their response properties and the latencies to chiasmatic stimulation. With a saccade in a stationary patterned field, T cells showed a burst discharge, while the discharges of S cells were completely suppressed.

3. The excitability was depressed in both S and T cells for 150–200 msec after a saccade, when the eye movement occurred in light. However, the depression did not occur in complete darkness.

4. The depression occurred also in the absence of eye movement, when the patterned visual field was moved in a saccadic fashion.

5. The depression in S cells occurred during an inhibitory period. Since S cells do not receive signals on image movement directly from the retina, the depression was due to a recurrent inhibition by signals transferred through the T ganglion-relay cell channel.

6. The depression in T cells occurred concomitantly with the burst discharge. Since the recurrent inhibition was operating less effectively during the period, the depression may be due to a phasic occlusion of the test impulse by coincident high-rate firings in the same cell.

7. The impairment in transmission of visual information through the lateral geniculate nucleus during the period following eye movements has been discussed in connexion with a neurophysiological basis for saccadic suppression.

INTRODUCTION

The threshold for visual perception rises abruptly with eye movement. One does not notice, therefore, any blurring of images when rapidly shifting the eye from one point of fixation to another. The perceptual depression during a saccadic eye movement, called 'saccadic suppression' (Latour, 1962; Volkmann, 1962; Zuber & Stark, 1966; Beeler, 1967; Volkmann, Schick & Riggs, 1968), is accompanied by a decrease of visually evoked responses (Michael & Stark, 1967; Gross, Vaughan & Valenstein, 1967; Duffy & Lombroso, 1968; Chase & Kalil, 1972). The findings which may be interpreted as a neurophysiological basis for saccadic suppression have been reported also in experimental animals (Michael & Stark, 1966; Bizzi, 1966; Collewijn, 1969; Cohen, Feldman & Diamond, 1969; Ogawa, 1972; Adey & Noda, 1973; Noda & Adey, 1974*a*). A reduction in the amplitude of both cortical and subcortical potentials evoked by flash or optic tract stimulation has been demonstrated.

The present study of single units shows that the excitability of relay cells in the lateral geniculate nucleus, evaluated from the firing probability of the cells to stimulation of the optic tract, is profoundly depressed when the cat makes a saccadic eye movement. This confirmed the previous findings (Adey & Noda, 1973; Noda & Adey, 1974*a*) that the transmission of impulses, which transfer visual information to the visual cortex, is substantially impaired at the lateral geniculate nucleus during an eye movement.

On the other hand, recent acute experiments have shown that there are two classes of relay cells in the nucleus that have different response properties and may subserve different functional roles (Cleland, Dubin & Levick, 1971; Hoffmann, Stone & Sherman, 1972; Singer & Bedworth, 1973; Stone & Dreher, 1973; Dreher & Sanderson, 1973). The present study demonstrates that a similar depression in the excitability occurs in both classes of relay cells following saccadic image movements, either by eye or object movements. In one class of cells, however, a depression occurred during the period of inhibition of spontaneous activity, whereas in the other class of cells, it occurred concurrently with a transient excitation. It is suggested that neuronal mechanisms for the depression are different between cell classes.

METHODS

A total of 133 relay cells of the lateral geniculate nucleus was tested in the course of thirty-two experiments on nine chronic cats. The units were recorded mainly from the layers A and A_1, identified physiologically by testing the ocular dominance in individual units. Each unit was identified also by the latency of its evoked spike to optic chiasm stimulation.

Surgical preparation and experimental condition

The method of single unit recording from chronic cats through painless immobilization of the head has been described (Noda, Freeman, Gies & Greutzfeldt, 1971). In brief, about 1 week before the first experiment, a stimulating electrode was inserted in the optic tract, at the chiasm, under pentobarbitone anaesthesia, and a vertical cylinder was fixed in a trephined opening in the skull for later insertion of microelectrodes into the lateral geniculate nucleus. Two transverse tubes were then placed in the horizontal stereotaxic plane and embedded in acrylic cement on the skull. Later, during experiment, when the head was fixed in the stereotaxic frame, the system gave complete immobilization without painful pressure and permitted microelectrodes to be driven stereotaxically.

Eye movement recordings

Eye movements were recorded by electro-oculogram (e.o.g.) in both horizontal and vertical directions with silver–silver chloride electrodes (Schiller & Körner, 1971) implanted in the bone surrounding the orbit. The e.o.g. was amplified by d.c. differential amplifiers, one for horizontal and one for vertical eye movement. These amplifiers were specially designed to show no measurable drift with FET-input operational amplifiers (ZA 801M3, Zeltex). The method of e.o.g. calibration has been described (Noda, 1975*a*).

Experimental procedures

The firing probability of lateral geniculate nucleus cells to stimulation of the optic tract was evaluated during the period following saccades. The test shock was a rectangular 50 μsec pulse and its amplitude was selected for each unit by finding an intensity which gave a firing probability of 80–90% in the room light while the eyes were stationary. With these intensities, the behaviour of the cat was normal, although at higher intensities twitching of whiskers was observed with the electrical stimulation. However, the cat tended to fall asleep after a prolonged test, suggesting that the stimulus was not unpleasant. In some cases, amphetamine 0·3 mg/kg was administered to maintain alertness.

Electrical stimulation of the optic tract occurred with an eye movement, by triggering the stimulator from potential shifts in the horizontal e.o.g. For this purpose, the e.o.g. potential was led to a circuit which involved a rectifier and a comparator and generated a pulse when a potential shift reached the threshold. The threshold setting of the comparator determined the delay from the onset of eye movement to the moment when the circuit was activated (time 0). I usually selected a comparator threshold which gave a stimulus delay of approximately 30 msec. This value was large enough to neglect smaller eye movements and the stimulator was triggered only when the eye movement exceeded an angular distance of about 5°. A pulse from the circuit triggered another pulse generator (a modified Tektronix 161) which produced a gate of 2 sec. The gate was to ignore succeeding eye movements which might occur within 2 sec. This 2 sec pulse then triggered a Grass S4 stimulator. The delay of electrical shocks from the time 0 was varied by the Grass S4 stimulator. The output of this stimulator was led to a wave-form generator (Tektronix 162) and further to two independent pulse generators (Tektronix 161). One was used to trigger an oscilloscope externally a moment before the other pulse generator delivered an electrical shock to the optic tract via a stimulus isolation unit.

The modification in the excitability was tested in a stationary patterned field and in complete darkness. The stationary pattern was a vertically oriented grating

(examples are shown in Figs. 2 and 3) consisting of dark stripes 5° wide (5 cd/m²) separated by light stripes 5° wide (70 cd/m²). This pattern was projected on a tangent screen placed at 75 cm from the eyes of the cat. It subtended 60° of the visual field horizontally and 45° vertically.

In some experiments, the projected pattern was moved across the tangent screen by reflecting the image from a front surface mirror attached to the spindle of a galvanometer which was operated by the horizontal e.o.g. potentials recorded previously on magnetic tape. By this operation, the image motion which might occur during a saccadic eye movement could be simulated while the eyes were stationary.

In eleven experiments on four cats, an intravenous and a tracheal cannula were intubated under ether anaesthesia. The incision for the intravenous intubation was infiltrated thoroughly with 2% lidocaine hydrochloride (Xylocaine) and a small amount of adrenalin. The trachea was anaesthetized with lidocaine jelly. By maintaining the general anaesthesia with N_2O/O_2 (70%/30%), gallamine triethiodide (Flaxedil) was injected through the intravenous cannula to eliminate eye movements and the animal was respired artificially through the intubated tracheal cannula. The animal was unconscious and supported comfortably in the same way as alert cats by the method of painless immobilization of the head (Noda *et al.* 1971). The stimulus intensities were always within the range used for alert cats.

Several days later each cat was used again for chronic experiments. None of the four cats showed any signs of unwillingness to undergo the next experiment.

RESULTS

(1) *Properties of S and T cells*

In response to spontaneous eye movements in the stationary grating-field, relay cells of the lateral geniculate nucleus showed various firing patterns, which were characteristic of different cell classes. Based on the firing patterns, we have distinguished two classes of relay cells; S and T cells (Noda, 1975*a*). S cells changed the firing level tonically when the eyes were shifted from one point of fixation to another (e.g. Fig. 2*A*). This is due to the *sustained response* of the *S cells* to local differences in luminance. *T cells* showed *transient responses* to rapid shifts of retinal images during saccades (e.g. Fig. 3*A*) and did not respond to local differences in luminance (Noda & Adey, 1974*a*). Latencies of S cells to stimulation of the optic chiasm were longer (mean = 1·77 msec) than those of T cells (mean = 1·15 msec). When tested with diffuse light switched on and off over the tangent screen, *S cells* showed a *sustained response* either to the light or to darkness, whereas *T cells* responded *transiently* either to the onset or offset of the light, or to both. Therefore, S and T cells seem to possess properties of the previously identified X and Y cells, respectively, in acute experiments (Cleland *et al.* 1971; Hoffmann *et al.* 1972). In a total of 315 relay cells of the lateral geniculate nucleus, there were 114 S cells (36·2%) and 109 T cells (34·6%). The other ninety-two cells (29·2%) showed response properties in between. Since these cells showed *mixed responses*, i.e. transient responses to saccadic image shifts and sustained

responses to local differences in luminance, they had been called *M cells* (Noda & Adey, 1974*a*). In the present study, the excitability was tested in seventy-two S cells and sixty-one T cells. As behaviour of M cells during saccades was similar to that of T cells, data obtained from eleven M cells are not included. A detailed account for the 315 cells in response to stationary and moving visual stimulation is given in another report (Noda, 1975*b*).

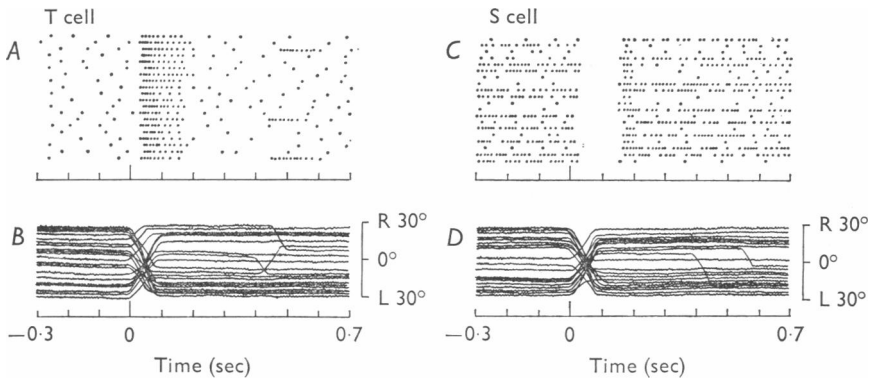

Fig. 1. Responses of a T cell (*A*) and an S cell (*C*) to saccadic eye movements in a visual field where the right hemifield was homogeneously illuminated. Discharges of the cells in response to saccades are shown for the period from − 300 to 700 msec in reference to the onset of saccades. The discharges are represented by horizontal rows of dots and the responses are aligned to the start of saccades (time 0). The time course of the corresponding saccades is shown below the responses by superimposed twenty consecutive sweeps of horizontal e.o.g. (*B* and *D*). In the e.o.g. calibrations, R 30° represents 30° to the right and L 30° means 30° to the left seen from the cat.

(2) *Discharges of S and T cells during saccades*

With eye movements in the light, T cells showed a burst firing, while discharges of S cells were completely inhibited. Fig. 1 shows this in an S and T cell. Spike trains are aligned to the onset of eye movements (time 0) and the spikes are represented by horizontal rows of dots (*A* and *C*). The time course of saccades is shown by superimposed twenty consecutive sweeps of horizontal e.o.g. (*B* and *D*).

The T cell discharged transiently in response to saccades, showing a burst for about 150 msec (Fig. 1*A*). The latency of the burst from the onset of saccades was approximately 30 msec. The burst appeared to every saccade, indicating that there was no preferred direction of eye movement in eliciting the burst responses.

On the other hand, the spontaneous firing of the S cell was completely suppressed by saccades (Fig. 1*C*). The time course of the suppression was

almost comparable to that of the burst in the T cell. The firing level was frequently different after a saccade. This is due to the sustained response of the S cell to local differences in luminance. However, these modifications in activity disappeared completely when eye movements occurred in total darkness.

(3) *Excitability changes in S cells following saccades*

The modification in the excitability of an S cell following a saccadic eye movement is shown in Fig. 2. It is tested in the stationary grating-field (Light) and in complete darkness (Dark). The excitability was evaluated by the firing probability to optic tract stimulation. When the horizontal e.o.g. was shifted by a saccade, the stimulus circuit was triggered and a test shock was delivered to the optic tract with various delays. The excitability was tested from moment to moment by changing the delay of the stimulus pulse at 10 msec intervals during the period following eye movements (0–300 msec).

The background firing of the S cell during spontaneous eye movements in the stationary grating is shown in Fig. 2*A*. The firing was completely inhibited during the saccades. While the eyes were stationary, the cell showed different levels of activity that were related to the direction of gaze. As seen in the film records (Fig. 2*C*), the firing rate was higher in the light (*b*) than in complete darkness (*a*). This is due to the sustained response of the cell to the light. For the same reason, the cell showed a tonic high-rate discharge when the receptive field entered a light stripe of the grating (Fig. 2*A*).

Examples of film records tested at a stimulus delay of 30 msec in complete darkness (*a*) and in the stationary grating (*b*) are shown in Fig. 2*C*. The spike responses are recorded simultaneously on both faster (5 msec/sweep) and slower (0·5 sec/sweep) horizontal sweeps. The potential shifts in the e.o.g. (H) were displayed on the oscilloscope as a spot moving horizontally and recorded continuously on a running film. The firing probability at each stimulus delay was calculated from such film records by finding positive responses (marked by the stars) in twenty consecutive trials.

The graph of Fig. 2*B* compiles the data at different stimulus delays. They are expressed as percentages. The controls were those in the periods without eye movements and the firing probability was 80–90 % (see Methods). The horizontal bars at the left-upper of Fig. 2*B* show the start and finish of ten successive saccades, which are aligned to the time when the e.o.g. potential shift triggered the stimulator (time 0). The average latency from the onset of eye movement to the time 0 was approximately 30 msec (see Methods).

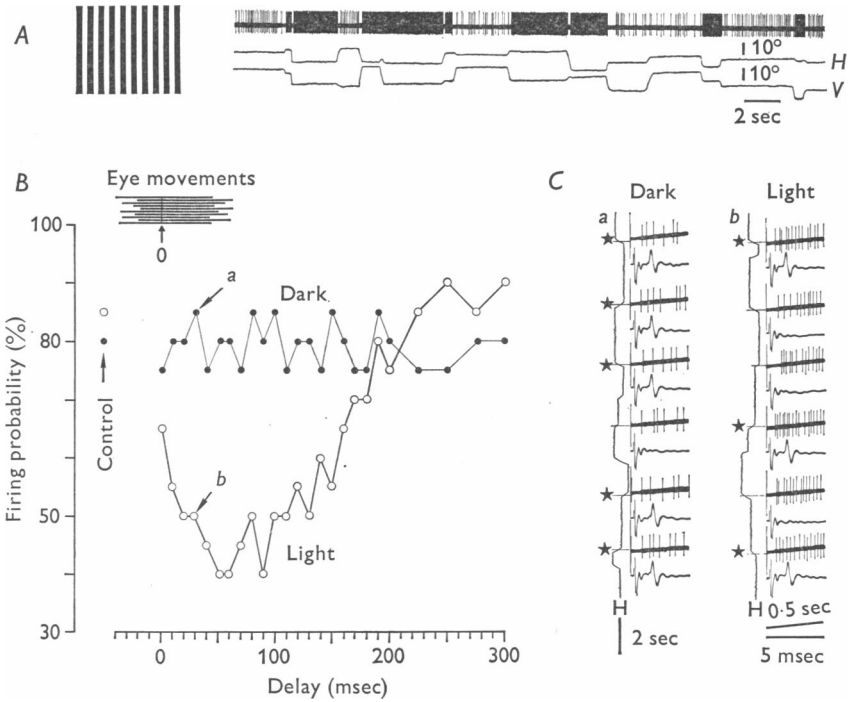

Fig. 2. Changes in firing probability of an S cell of the lateral geniculate nucleus upon stimulation of the optic chiasm during the period following saccadic eye movements. *A*, discharges of the cell during spontaneous eye movements scanning the stationary patterned field (shown at left). The higher activity level represents the *sustained response* of the cell to the light stripe of the grating. *H*: horizontal e.o.g. *V*: vertical e.o.g. Note the suppression of the discharges during the saccades. *B*, firing probability was tested by changing the delay of the stimulus pulse at 10 msec intervals (the testing order was shuffled randomly) in the patterned field (light, open circles) and in complete darkness (dark, black circles). Firing probability was calculated from twenty consecutive responses. Horizontal bars at the left-upper show the start and end of ten successive saccades, they are aligned to the moment when the potential shift triggered the stimulator (time 0). *C*, original film records tested at a stimulus delay of 30 msec in complete darkness (*a*, dark) and in the patterned field (*b*, light). The spike responses are shown by vertical deflexions in both faster (5 msec/sweep) and slower (0·5 sec/sweep) horizontal sweeps, and the potentials shifts in the horizontal e.o.g. (*H*) are displayed horizontally as a moving spot and recorded continuously on a running film. To avoid triggering of the stimulator by subsequent eye movements occurring within 2 sec of the first movement, a gating circuit inhibited activation of the stimulator until an eye movement occurred more than 2 sec after the preceding pulse (see methods). Positive responses are marked by stars on the left. Latency of the spike was 1·66 msec.

With eye movements in the light, the excitability of the S cell decreased markedly for about 160 msec following the saccades (open circles). The depression continued considerably longer than saccades. On the other hand, it did not occur when tested with saccades in complete darkness (filled circles). Firing probability of the cell then remained within the range of chance levels ($80 \pm 10\%$) throughout the period.

(4) *Excitably changes in T cells following saccades*

A similar depression in the firing probability occurred also in T cells. An example is shown in Fig. 3. It is important to find that the depression occurred concurrently with an increase in the discharge of the same cell. As seen in Fig. 3*A* and also in another example of Fig. 1*A*, T cells generally show a burst discharge in response to a quick shift of retinal images. A similar burst response to saccade has been found also in T ganglion cells of the retina (Noda & Adey, 1974*b*), which are connected directly to T relay cells of the lateral geniculate nucleus (Cleland *et al.* 1971). Therefore, the burst discharge represents increased excitatory volleys.

With eye movements in the light, the firing probability of the T cell in response to optic tract stimulation decreased for about 150 msec (open circles), while this was not seen in complete darkness (filled circles). Since the T cell was strongly driven by the burst discharge, the decreased excitability may be due to an increased chance for the test stimulus to fall in an unresponsive period of the cell (read Discussion).

The depression in the excitability of the T cell is not attributable to the prolonged inhibition, which may be the case with S cell. There was evidence to infer that the inhibitory mechanism was operating less effectively during the same period. Generally, it has been accepted that relay cells of the lateral geniculate nucleus respond to optic tract stimulation with a single spike (or an e.p.s.p.) which is immediately followed by a long silent period (Bishop & Davis, 1960; Bishop, Burke & Davis, 1962; Fukuda & Iwama, 1972). This period is ascribed to a recurrent inhibition (or i.p.s.p.) (Burke & Seften, 1966; McIlwain & Greutzfeldt, 1967; Singer & Creutzfeldt, 1970; Kato, Yamamoto & Nakahama, 1971). Concurrently with the burst period of T cells, the duration of recurrent inhibition, duration (X), became considerably shorter (Fig. 3*B*, lower graph). This is due to an overwhelming excitatory drive from T ganglion cells. The duration (X) at each point is expressed as a percentage of the control (mean duration in complete darkness). With eye movements in the stationary grating-field (light, open circles), the duration became less than 20% for the period 50–100 msec. It was larger than the average level in darkness (dark, filled circles), when tested at a delay longer than 200 msec, as seen in the overshoot beyond 220 msec (Fig. 3*B*, lower graph). This was commonly observed. As the

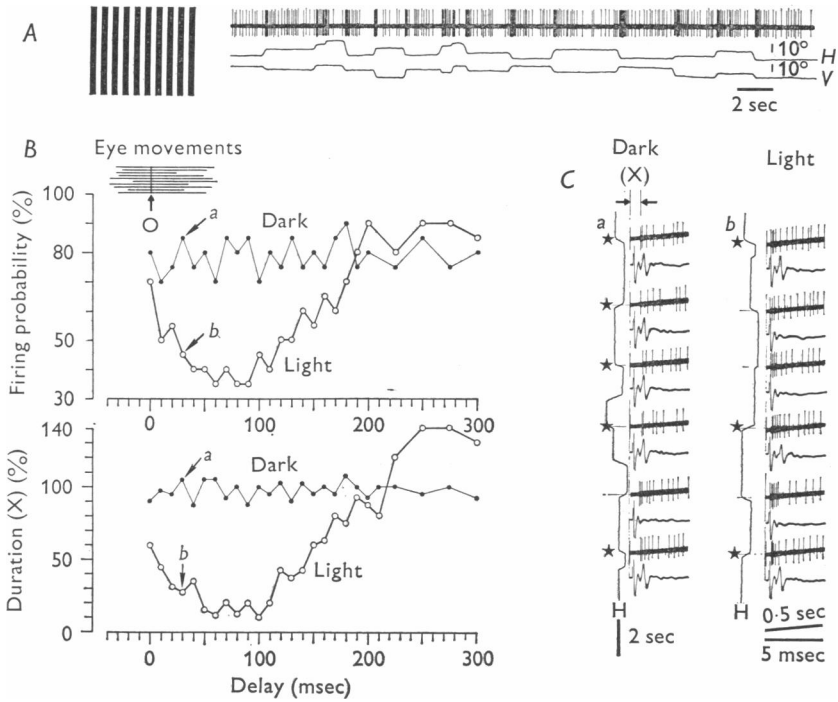

Fig. 3. Changes in firing probability of a T relay cell of the lateral geniculate nucleus upon stimulation of the optic chiasm during the period following saccadic eye movements. *A*, transient responses of the cell to spontaneous eye movements. *H*: horizontal e.o.g. *V*: vertical e.o.g. Note that the firing level did not alter while the eyes were stationary, indicating that the cell did not respond to local differences in luminance. *B*, upper graph: firing probability of the cell in the patterned field (light, open circles) and in complete darkness (dark, filled circles), tested during the period following saccades. Firing probability was calculated from twenty consecutive spike responses. Horizontal bars at the left-upper show the start and end of ten consecutive saccades, aligned to the time 0. Lower graph: changes in the duration (*X*), measured from the evoked spike to the first spike after the silent period in the slower sweeps in the film records. The duration (*X*) is attributed to the recurrent inhibition and each value is expressed as a percentage of the control, which is the mean duration in complete darkness. *C*, original film records tested at a stimulus delay of 30 msec. Note the shorter duration (*X*) in the patterned field (*b*, light) that was followed by a burst discharge. Positive responses are marked by stars on the left. Latency of the spike was 1·1 msec.

duration of burst impulses lasted only about 150–200 msec (an example of bursts is seen in Fig. 1*A*), the overshoot may represent a relief from the excitatory drive, indicating also that an inhibitory effect remains for a longer period. These two seemingly conflicting phenomena occurred

simultaneously in T cells; i.e. depression of the excitability (upper graph) and reduction of the inhibitory period (lower graph) occurred in the light. However, both effects did not occur in complete darkness, as shown by the curves with black circles.

(5) *Effect of image movements*

There were data indicating that the movement of retinal images during a saccade is important to the depression; (i) the amount of depression was dependent upon the visual environment, (ii) the depression did not occur with eye movements in complete darkness. A visual field with a grating of alternating dark and light stripes of 5° wide was very effective. A less prominent depression could also be seen when only one stripe was shown at the periphery of an otherwise homogeneously illuminated half-cylinder screen. Even a vertical stripe placed at peripheral 60° was also effective. Therefore, the depression was due to retinal processes initiated by image movements.

This notion is supported further by the fact that a similar depression occurred in the absence of eye movement, when the retina was stimulated with moving images. In some experiments, the patterned field was moved in a saccadic fashion before the *stationary* eyes (see Methods). For this purpose, the grating image was reflected from a front surface mirror attached to the spindle of a galvanometer, which was actuated by potentials of horizontal e.o.g. previously recorded on magnetic tape. The vertically oriented grating was then moved horizontally at a speed and amplitude comparable to the movement of retinal images during an actual eye movement. The changes in excitability were studied in the same way as for eye movements. This time, however, the stimulator was triggered by the potential shifts in the recorded e.o.g. The firing probability of relay cells to optic tract stimulation was similarly reduced by the movement of retinal images. The amount and time of the depression was almost comparable to those of actual eye movements. It was thus concluded that the movement of retinal images during a saccade produced the depression in excitability of relay cells and that eye movement *per se* was not essential for the depression.

DISCUSSION

(1) *Significance of the depression in S cells*

An important finding in the present study is that the depression in the excitability could be demonstrated in S cells. These cells receive direct excitatory drive from the slow conducting axons of ganglion cells (Cleland *et al.* 1971), which have response properties similar to those of S cells, hence called S ganglion cells (Noda & Adey, 1974*b*). The fact that the depression occurs in S cells may be convenient in interpreting the neuronal

events during eye movements in relation to saccadic suppression. The functional significance of S cells in vision has been recently postulated in both the retina (Cleland *et al.* 1971; Fukada & Saito, 1971; Ikeda & Wright, 1972; Cleland *et al.* 1973) and the lateral geniculate nucleus (Cleland *et al.* 1971; Hoffmann *et al.* 1972; Singer & Bedworth, 1973; Stone & Dreher, 1973; Dreher & Sanderson, 1973). Having a small and sharply definable receptive field in the central retina, the function of S cells would be primarily concerned with the analysis of spatial characteristics of visual stimuli and subserve the mechanism of form recognition. The S cells may thus play an important role in the central (foveal) vision.

A reduction in the excitability of T cells had been predicted by previous studies of evoked potentials (Bizzi, 1966; Ogawa, 1972; Adey & Noda, 1973). It was shown that the r_1 component of the orthodromic response of the lateral geniculate nucleus became smaller following eye movements. Since the r_1 is attributable to firing of the fast lateral geniculate nucleus cells (Bishop & McLeod, 1954), the smaller r_1 suggested a depression in the excitability of T cells, which are connected to the fast conducting axons of T ganglion cells.

In the evoked potential studies, however, the behaviour of the r_2 component (attributable to S cells) has not been investigated reliably for the following two reasons: (i) because the r_2 had a higher threshold, a stimulus intensity which was optimal to observe the depression in r_1 was frequently not strong enough to evoke the r_2, even when a stronger stimulus was applied to the optic tract to elicit the r_2; (ii) because the r_2 had a longer latency and hence it appeared superimposed on the field potential of the r_1, it was difficult to evaluate a genuine change in the r_2 amplitude that was solely ascribable to the behaviour of S cells. The present single unit experiment enabled me to study the behaviour of individual cells by finding the optimal stimulus intensity for the depression in each cell.

(2) *T ganglion cells transfer impulses of saccadic image movements*

The depression in the excitability of relay cells was caused by the movement of retinal images during a saccade; because (i) the amount of depression was dependent upon the visual environment, (ii) it did not occur with eye movement in complete darkness, but (iii) occurred in the absence of eye movement, during retinal stimulation with moving images. Therefore, the depression was a consequence of retinal processes initiated by image movements and independent of the eye movement *per se*. A saccadic displacement of retinal image may cause a transient excitation in a group of ganglion cells, which in turn may transmit a burst of impulses to relay cells of the lateral geniculate nucleus and cause a phasic depression in their excitation.

In a previous study, we found that there were S and T types also in retinal ganglion cells whose response properties were almost identical with S and T geniculate cells, respectively (Noda & Adey, 1974*b*). The T ganglion cells showed a burst discharge to rapid image motion of a saccade. The burst discharge was so explosive that the firing rate became frequently more than 10 times greater. It started 20–30 msec after the initiation of saccades and lasted for about 100–200 msec (Noda & Adey, 1974*a*). The time course of the burst, therefore, corresponded fairly well to the period of the depression.

A striking feature of the relay in the lateral genicular nucleus is that there is little convergence of S and T afferents in their direct excitatory actions on relay cells (Cleland *et al.* 1971; Hoffmann *et al.* 1972). The S and T relay cells receive excitatory impulses from S and T ganglion cells, respectively, and the response properties of the S and T channels are well preserved (Cleland *et al.* 1971). Since S ganglion cells are unresponsive to saccadic image movement (Noda & Adey, 1974*b*), the impulses for the depression of S and T relay cells must depend on the burst discharge of T ganglion cells. The burst discharges of T relay cells that appeared after saccadic eye movements (Figs. 1*A* and 3*A*) are signs of the impulses transmitted via the axons of T ganglion cells.

(3) *Neuronal mechanisms for the depression*

The mechanism of the depression in S cells may be different from that in T cells. Although S cells did not receive burst impulses directly from the retina, their spontaneous activity was completely suppressed concurrently with the burst discharge of T cells (Fig. 1). It is likely, therefore, that the inhibition was brought about by the burst of T cells. There is evidence suggesting that inhibitory effect introduced in the lateral geniculate nucleus can modify the activity of both S and T cells simultaneously and the effect is no longer specific to either cell classes (Hoffmann *et al.* 1972; Singer & Bedworth, 1973). On the other hand, when a single shock is applied to the optic tract, the initial excitatory effect in a relay cell is commonly followed by a prolonged inhibition, as seen in slower records of Figs. 2*C* and 3*C*. This is attributed to a recurrent inhibition. In both the rat (Burke & Sefton, 1966) and the cat (Kato *et al.* 1971) the inhibitory mechanism involves interneurones, which are activated by the recurrent collaterals of relay cell axons and inhibit both S and T cells in the same population. This inhibition can occur in an S cell at a stimulus intensity far below the threshold for eliciting a spike in the same S cell. As the threshold for T cells is typically lower than that for S cells, as observed also by other authors (Hoffmann *et al.* 1972; Singer & Bedworth, 1973), even such a low intensity stimulation can evoke a spike in T cells, which in

turn might activate inhibitory interneurones through the recurrent collaterals. It appears likely that the depression in S cells is a result of recurrent inhibition, caused indirectly by the burst discharge of T cells.

On the other hand, the depression in T cells may not be attributable to the prolonged inhibitory effect. Two seemingly conflicting phenomena, i.e. a transient excitation and a reduction in the responsiveness to orthodromic volleys, occurred simultaneously in T cells. The transient excitation is due to the burst of excitatory impulses transferred through the axons of T ganglion cells which are excited transiently by quick shifts of retinal image during saccades. A suppression of discharges associated with an increase in background activity is known in some neuronal events. For example, a cessation of firing during excessive depolarization of membrane is known as 'inactivation processes' in hippocampal cells (Kandel & Spencer, 1961). However, this appears not to be the case with the cells in the lateral geniculate nucleus, because the self-control mechanism involving recurrent inhibition seems to be well developed in this nucleus (Burke & Sefton, 1966).

Since depression in T cells occurred concomitantly with an increase in firing rate, it may be ascribable to a phasic occlusion of the test impulse by coincident high frequency discharge of the cell. Recovery of responsiveness following a spike discharge seems to require a considerable long period in relay cells of the lateral geniculate nucleus. By applying a pair of shocks at different intervals to the optic tract of the rat, Burke & Sefton (1966) have found that the recovery of responsiveness in some P cells (relay cells) to the second optic tract shock takes as long as 40–80 msec. Therefore, the reduction in the firing probability in T cells may be due to an increased chance for the test stimulus to fall in the unresponsive period which follows each discharge during the high frequency firing of the transient response. An increased activity in the lateral geniculate nucleus cells does not necessarily mean that information transmission through the nucleus is facilitated. On the contrary, transmission of impulses of visual information may be inhibited by the burst discharge of T cells. The depression in excitability of S cells, which are believed to play an important role in the foveal vision, would indicate that the transmission of visual information through the lateral geniculate nucleus is impaired during the period following saccadic eye movements.

(4) *Functional implications*

Previous psychophysical studies have shown that the threshold for perception of a test flash is raised during a saccade (Latour, 1962; Volkmann, 1962; Zuber & Stark, 1966). Probably because of this 'blanking out' effect, vision seems to be continuous even though eye movements occupy

a portion of time spent in reading or inspecting objects in the visual field. This type of perceptual depression has been found as much as 40 msec before the onset of eye movements and up to 80 msec after its completion (Latour, 1962; Volkmann *et al.* 1968). Because the greatest elevation of threshold occurs prior to the onset of eye movements, most investigators, until recently, had attributed the suppression effect to central influences, which are coordinated with the efferent motor impulses for eye movement (Zuber & Stark, 1966; Volkmann *et al.* 1968).

Recent findings have shown, however, that the movement of retinal images during a saccade is of considerable importance to the perceptual depression. Such depression can occur in the absence of eye movement, during retinal stimulation with moving images (MacKay, 1970) or by tapping the stationary eye (Richards, 1968). Moreover, if the visual field is displaced in a saccadic fashion before a stationary eye, perceptual reduction prior (-40 msec) to the onset of background movement has been observed (MacKay, 1970). Since there was no possibility of 'warning signals' from the oculomotor system in these cases, MacKay (1970) has inferred that the displacement of the visual world itself generates a transient 'neural disturbance' which may interfere with the subsequent detection of the test flash.

Although a direct extrapolation of the data from the cat to interpret these psychophysical observations may be somewhat imprudent, the present findings have furnished a good neurophysiological basis for MacKay's statement. It was shown that the transmission of visual information was impaired by the burst discharge of T ganglion-relay cell channel as a consequence of retinal processes initiated by image movements.

The neuronal mechanism proposed in the present study, however, does not explain why the perceptual depression occurs before the onset of image movement. Neurophysiological signs which precede the onset of image motion have not yet been discovered in the primary visual system of experimental animals (Michael & Stark, 1966; Bizzi, 1966; Collewijn, 1969; Cohen *et al.* 1969; Ogawa, 1972; Adey & Noda, 1973). The present study also agrees with this statement. However, it is important to recall that neurophysiological findings have been limited to sensory processes, while data from psychophysical experiments have dealt mainly with the products of perception. It is possible that the transient impulses generated by saccadic image movement may arrive at the visual centre in time to upset the processing of information about a target (or a flash) which was briefly presented before the movement began (Ditchburn, 1973). The neuronal mechanism underlying this event, however, may not be so simple as could be explained by differences in the conduction time of informations interacting with each other at the higher visual centre.

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