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INFLUENCE OF EYE MOVEMENTS ON GENICULO-STRIATE EXCITABILITY IN THE CAT

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SUMMARY

1. The excitability of the geniculo-striate pathway during a saccadic eye movement was studied in alert cats with chronically implanted electrodes. Excitability was assessed by the amplitude of post-synaptic components of field responses in both lateral geniculate nucleus and visual cortex to electrical stimulation of the optic chiasm. Modifications in amplitude were evaluated during the period following eye movements, by triggering a stimulator from potential shifts in the electrooculograms and altering delays in the stimulus pulse.

2. The post-synaptic component of the geniculate response was markedly depressed for about 150 msec, reaching a trough at approximately 100 msec after the initiation of an eye movement. This effect was dependent on the visual environment and was not observed in complete darkness. A similar depression occurred when the visual field was abruptly moved by retinal impulses generated by a quick displacement of the image of the visual world associated with an eye movement. The depression reflected a reduction of cellular discharge to the orthodromic volley and hence a suppression of the transmission of visual information through the lateral geniculate nucleus. This may be a mechanism for saccadic suppression.

3. The post-synaptic components of the cortical response were enhanced for about 200 msec, reaching a peak at approximately 150 msec after the initiation of an eye movement. Although this facilitation occurred also in complete darkness, it did not occur when the visual field was abruptly shifted while the eyes were stationary. The fact that it occurred with eye movements and exclusively in the post-synaptic components suggests that it was caused by signals from a system closely related to eye movements. This may be a manifestation of the corollary mechanism.

INTRODUCTION

Visual perception of movement is generally regarded as of primary biological importance, yet surprisingly little is known about the neurophysiological mechanisms subserving it. There are many unsettled questions, in particular, the apparent stability of the visual world during voluntary eye movements.

A moving object in the visual world is perceived as moving by reason of a brain function that distinguishes image motion on the retina occurring with object movement from that associated with active eye movement. The most promising hypothesis for this mechanism is that of a 'corollary discharge' that somehow modifies the visual information in the central visual pathway during an eye movement (Sperry, 1950; von Holst & Mittelstaedt, 1950; Teuber, 1960). In this model, an efferent (oculomotor) discharge resulting in an eye movement would be accompanied by concurrent central discharge into the visual system, the effect of which would be to anticipate and counteract these changes in afferent (visual) input which result from the eye movement. Such a self-regulating compensatory mechanism would enable a constant visualized environment in normal conditions of active eye movements.

As a simple model of oculomotor-visual integration, concomitant modifications of visual information have been studied during saccadic eye movements. Psychophysical studies have shown a rise in the visual threshold (Ditchburn, 1955; Volkmann, 1962; Latour, 1962; Zuber & Stark, 1966; Beeler, 1967; Starr, Angel & Yeates, 1969; Volkmann, Schick & Riggs, 1968; Mitrani, Yakimoff & Mateef, 1970). This increment of the threshold is frequently called 'saccadic suppression' and seems to be accompanied by a diminution or a modification of visually evoked responses (Gross, Vaughan & Valenstein, 1967; Michael & Stark, 1967; Chase & Kalil, 1972). Similar changes in the evoked responses with eye movements have been reported also in experimental animals (Collewijn, 1969; Michael & Stark, 1966). Transmission of an orthodromic volley was suppressed at the lateral geniculate nucleus (Bizzi, 1966; Kawamura & Marchiafava, 1968; Malcolm, Bruce & Burke, 1970; Ogawa, 1972). However, Mackay (1970) has questioned whether eye movements are necessary at all to show a saccadic suppression, since he has demonstrated a threshold elevation for flashes that occurs whenever the retinal image of the visual world is abruptly displaced. He suggests that displacement of the visual world itself generates a transient 'neural disturbance' which may interfere with the subsequent detection of the test flash.

In studying these problems, we report here the following findings. (1) Retinal impulses from a quick displacement of the image induced by either

a saccadic eye movement or an object movement reduced transmission of visual information through the lateral geniculate nucleus. This may be a mechanism for saccadic suppression. As suggested by Mackay (1970), saccadic suppression would then be independent of the corollary mechanism. (2) Saccade-contingent extraretinal impulses, probably arising in the oculomotor system, enhanced the excitability of the visual cortex. This facilitation may be a manifestation of the corollary mechanism. Thus, the visual cortex could be one of the locations for oculomotor-visual integration.

METHODS

Surgical preparation

Data were obtained from thirty-six experiments on seven chronically prepared cats. Surgery was performed under pentobarbitone anaesthesia approximately 1 week before the first experiment. Electrodes were implanted in the optic chiasm, lateral geniculate nucleus and visual cortex (Areas 17 and 18) by monitoring electrophysiological signs in the same way as described by Iwama, Sakakura & Kasamatsu (1965). First, a coaxial electrode was inserted in the optic chiasm, by finding a response (Fig. 1*A*) to a single flash of light. As this was for stimulation, the tips were placed deep in the chiasm so that both conductors recorded prominent monopolar responses. Next, by exploring the field potentials with another coaxial electrode, an orthodromic response (Fig. 1*C* upper trace) to chiasmatic stimulation was found in the lateral geniculate nucleus. Fig. 1*B* shows an antidromic response from the optic chiasm to geniculate stimulation, tested with the same electrodes by interchanging their roles. Finally, a typical response (Fig. 1*C*, lower trace) was found in the visual cortex to electrical stimulation of the optic chiasm.

The electrodes used for recording the electrooculogram (e.o.g.) were similar to the silver-silver chloride electrodes described by Schiller & Körner (1971). Four electrodes, two for horizontal and two for vertical eye movements, were implanted in the bones surrounding the orbit. The chiasmatic electrode was cemented to the skull with dentəl acrylic and connected to a 2-pin socket which was placed on the frontal bone. All other electrodes were connected to the terminals of a 9-pin socket which was secured on the centre of the skull with more acrylic. In minimizing the shock artifact, it was helpful to keep the leads of the stimulating electrode away from the other electrodes. Two transverse tubes were then embedded in the same acrylic mound on the skull.

Experimental conditions

During the experiment, the cat was comfortably supported by a hammock without restraint. The head was, however, held rigidly in the stereotaxic frame by inserting two pairs of ear bars into the transverse tubes mounted on the skull. The eye movement signals were amplified by d.c. differential amplifiers, one for horizontal and one for vertical eye movements. These amplifiers were specially designed to show no measurable drift. They were FET-input operational amplifiers (ZA801M3, Zeltex). Eye movements were monitored by combining the vector components of horizontal and vertical e.o.g.s and displaying them continuously on an oscilloscope screen as a moving spot. Experiments were performed in three visual environments; in patterned field, in diffuse illumination and in complete darkness. Two types of screen were used. One was a half-cylinder screen placed at 1 m from the head. This covered 180° of the visual field horizontally and 100° vertically. The other was a

tangent screen placed at 75 cm. It subtended 60° of the visual field horizontally and 45° vertically. The stationary pattern was a grating of vertically oriented 2° wide dark stripes (5 cd/m²) separated by 4° wide light stripes (70 cd/m²). Luminance over the diffusely lit screen was approximately uniform at 50 cd/m².

Experimental procedures

Orthodromic responses to electrical stimulation of the optic chiasm were recorded simultaneously from the lateral geniculate nucleus and visual cortex during a saccadic eye movement. The testing shock was a rectangular 50 μsec pulse of different intensity which was selected in each experiment after evaluating a strength–response curve for t_1 (Bishop & McLeod, 1954) and finding the intensity which produced t_1 of 50% maximum amplitude. With these intensities, the behaviour of the cat was normal, although at higher intensities we occasionally observed twitching of whiskers with the electric stimulation. However, the cat tended to fall asleep after a prolonged test, suggesting that the stimulus was not unpleasant. In such cases, amphetamine 0·3 mg/kg was sometimes administered to maintain alertness.

Electrical stimulation of the optic chiasm occurred with an eye movement, by triggering the stimulator from potential shifts in the horizontal e.o.g. For this purpose, the signal was led to a circuit which involved a rectifier and a comparator and generated a pulse when a potential shift reached the threshold. This pulse triggered another pulse generator which produced a pulse of 2 sec. This prevented triggering by succeeding eye movements which might occur within 2 sec. Finally, the 2 sec pulse triggered a Grass S4 stimulator which delivered a pulse to the optic chiasm, via a stimulus isolation unit. The threshold setting in the comparator determined the delay in the triggering pulse from the initiation of an eye movement. We usually selected a comparator threshold so that the final stimulator was triggered approximately 30 msec after the initiation of an eye movement. This value was large enough to ignore smaller eye movements and the stimulator was triggered only when the eye movement exceeded an angular distance of about 5°. The responses were displayed on an oscilloscope and photographed. Measurements were made on the image projected from an enlarger. Statistical calculations were performed by an IBM 360/91 computer.

In some experiments, the projected pattern was moved across the tangent screen by reflecting the image by a front surface mirror attached to the spindle of a galvanometer which was operated by a function generator. In other cases, the galvanometer was operated by e.o.g. potentials recorded previously on magnetic tapes. By this operation, the image motion which might occur during a saccadic eye movement could be simulated while the eyes were stationary. In five experiments on two cats, gallamine triethiodide (Flaxedil) was injected through a venous cannula to eliminate eye movements and the animal was respired artificially through an intubated tracheal cannula. The intubation of the venous and tracheal cannulae was performed under ether anaesthesia. Then general anaesthesia was continued with NO_2/O_2 (70%/30%). The incision for the venous cannula was infiltrated with 2% lidocaine hydrochloride (Xylocaine) and the trachea was anaesthetized with lidocaine jelly. Several days later both cats were used again for chronic experiments.

RESULTS

(1) *Post-synaptic component of the lateral geniculate response*

The wave form of the geniculate response (upper trace of Fig. 1*C*) was similar to that seen in the anaesthetized cat (Bishop & O'Leary, 1940;

Bishop, 1953) and in the alert cat (Iwama *et al.* 1965; Eisman, Hansen & Burke, 1967). Following the terminology of Bishop & McLeod (1954), we call the positive-negative wave immediately following the shock artifact the t_1 response and regard it as the response of fast optic tract fibres. This is followed by a negative wave, designated r_1, which we regard as a post-synaptic response. The identification of these two components is based on the stability of t_1 to repetitive stimulation and on the respective latencies. With a high frequency stimulation, the r_1 falls off, because of a failure of synaptic transmission, and another stable negative wave t_2 becomes dominant (Fig. 1*E*).

The t_2 response arises in slow optic tract fibres. The r_1 deflexion is attributable to firing of geniculate cells. Fig. 1*D* illustrates a response of a geniculate cell to chiasmatic stimulation, recorded from a chronic cat in another experiment. The latency of t_1 in the field potential was comparable to that of t_1 in *C* and the cellular spikes appeared at the position corresponding to r_1 in *C*.

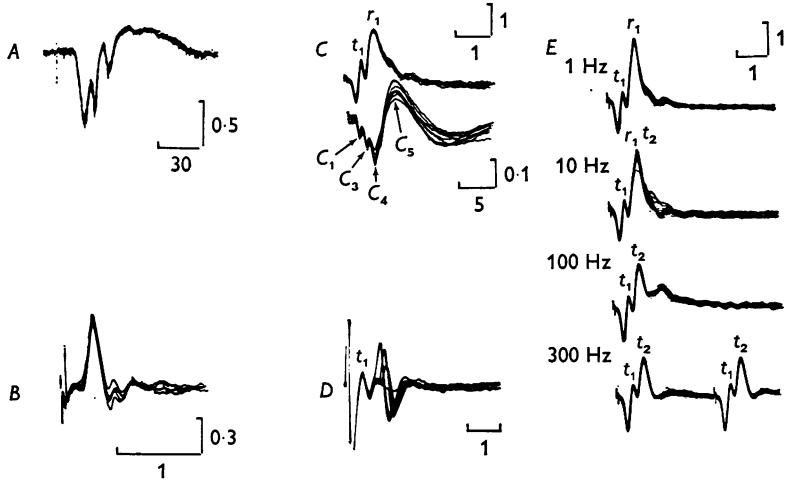

Fig. 1. *A*: evoked response of optic chiasm to a single flash of light. *B*: antidromic response of optic chiasm to electrical stimulation of lateral geniculate nucleus. *C*: simultaneous recording of lateral geniculate (upper trace) and visual cortical (lower trace) responses to electrical stimulation of optic chiasm. *D*: spike responses of a single geniculate cell, recorded with a stainless-steel micro-electrode in another experiment using identical preparation. Note that cellular spikes appeared on the position corresponding to the r_1 (post-synaptic geniculate response) in *C*. *E*: geniculate responses to repetitive stimulation of optic chiasm, at frequencies indicated. Note that r_1 fell off at 100 Hz and 300 Hz and the presynaptic t_2 representing slow optic tract fibres, was revealed. In each record 10–30 successive responses were superimposed. Horizontal bars represent time calibration and figures are in msec. Vertical bars represent amplitude calibration and figures are in mV.

(2) *Post-synaptic components of the cortical response*

The wave form of the cortical response (lower trace of Fig. 1*C*) was similar to that commonly seen in the primary cortical areas during thalamic stimulation. The response had, in addition to the slow positive and negative waves, a series of spike-like waves at its onset and continuing through part of the slow positive wave. Following the terminology of Malis & Kruger (1956), we used numbers to designate these components. It is known that C_1 and C_2 are responses of the presynaptic (geniculate) fibres and C_3 and C_4 are the post-synaptic responses which may arise from pyramidal and stellate cells by a series of synaptic relays (Bishop & Clare, 1953). The negative wave (C_5) reflects a depolarization of the upper apical

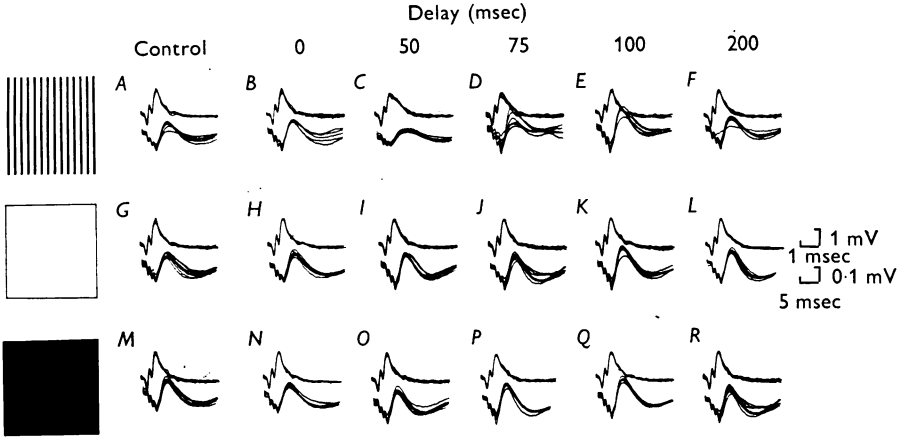

Fig. 2. Changes in lateral geniculate (upper trace) and visual cortical (lower trace) responses to chiasmatic stimulation in the period following saccadic eye movements. They were tested in three different visual environments, indicated on the left of each horizontal row, namely, in patterned environment (upper), in diffuse illumination (middle) and in complete darkness (lower). The chiasmatic stimulation was applied by triggering the stimulus circuits from potential shifts in the horizontal e.o.g. and altering delays in the stimulus pulse, indicated above each vertical row. Control responses were obtained by stimulating at 0·5 Hz while the eyes were steady. In each record, 5–10 successive responses were superimposed.

dendrites (Ochs, 1965). In the present study, as a post-synaptic response of the lateral geniculate nucleus, we measured the height of r_1, and as a post-synaptic response of the visual cortex, we measured the amplitude from C_4 to C_5. Unless otherwise specified, 'geniculate response' denotes the r_1 and 'cortical response' denotes the amplitude from C_4 to C_5.

(3) *Changes in geniculate and cortical responses following eye movements*

The modifications in excitability of geniculate and cortical domains following spontaneous rapid eye movements (saccades) were evaluated by testing the responses from moment to moment during the period following saccades (0–300 msec), by triggering the stimulus circuits from potential shifts in the horizontal e.o.g. and altering delays in the stimulus pulse. Fig. 2 illustrates the changes in the geniculate and cortical responses tested in three different visual environments, indicated on the left of each row. The control responses were obtained by stimulating at 0·5 Hz when the eyes were steady.

Four sets of recordings (*C*, *E*, *O*, *Q*) are particularly interesting. In *C*, tested in the patterned field at a delay of 50 msec, both geniculate and cortical responses were remarkably depressed. By contrast, the geniculate response in *E*, tested in the same condition at a delay of 100 msec, was smaller, but the cortical response was almost 1·5 times larger than the control (*A*). The transition can be seen in *D* (delay 75 msec) where the cortical response showed a considerable variation. However, when the cat was in complete darkness, the depression seen in *C* did not occur. In the recording *O*, tested in complete darkness at a delay of 50 msec, the geniculate response was comparable to the control (*M*) but the cortical response was slightly larger. In *Q*, tested in complete darkness at a delay of 100 msec, although the geniculate response was the same as the control, enhancement of the cortical response was still evident.

These changes were evaluated quantitatively and shown in Fig. 3. The results of the geniculate responses are plotted in the upper graph (*A*) and those of the cortical responses are shown in the lower graph (*B*). The data obtained in three conditions are expressed by different symbols; filled, open and half filled circles denoting complete darkness, diffuse illumination and the patterned visual field, respectively. Each circle represents an average amplitude of thirty consecutive responses and the vertical bar represents one standard deviation. They are expressed as percentages of the amplitude of the control response evaluated in complete darkness while the eyes were steady. It has been observed in anaesthetized cats that the synaptic responsiveness in the geniculo-striate system falls when tested in darkness (Chang, 1952). However, this effect was absent when the cat was alert, confirming the observation of Hansen, Bruce & Burke (1967).

The onset of eye movements is indicated by E.M. This occurred approximately 30 msec before the triggering moment (delay 0) and the time variation around the mean is shown by 1 S.D. (shaded area). The average duration of eye movements in this preparation that triggered the stimulus circuits was 65 msec in the light and 82 msec in complete darkness.

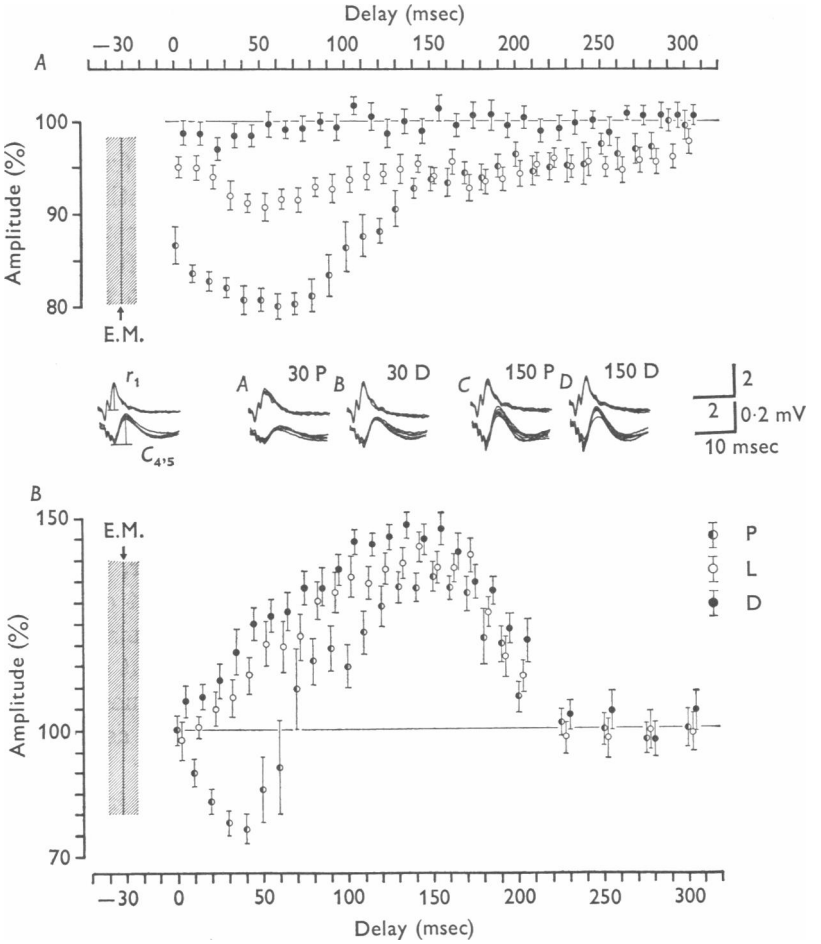

Fig. 3. Changes in amplitude of r_1 (post-synaptic response of lateral geniculate) (*A*) and C_4–C_5 (post-synaptic response of visual cortex) (*B*) to electrical stimulation of optic chiasm, during the period following saccadic eye movements. A stimulator was triggered by potential shifts in the horizontal e.o.g. and a single shock was given to the optic chiasm with variable delays indicated by the abscissae. Each circle represents an average amplitude of 30 responses and the vertical bar represents one standard deviation. E.M.: an average and 1 S.D. (shaded area) of the initiation of saccadic eye movements before the moment when the stimulator was triggered (delay 0). P: in patterned environment. L: in diffuse illumination. D: in complete darkness.

(4) *Quantitative evaluation of the geniculate depression*

When an eye movement occurred in the patterned visual field, the geniculate response was depressed. The amplitude fell to approximately 80 % of the control for the period from 40 to 70 msec. This represents a considerable depression, since in this preparation maximum reduction of the r_1 deflexion by repetitive stimulation at 300 Hz showed a value of 65 % of the control. This 65 % was attributed to the t_2 response of the presynaptic fibres. However, the depression did not occur when eye movements occurred in complete darkness. The slightly smaller values in the range from 0 to 40 msec were not significant. When eye movements occurred in a homogeneous field, a weaker but significant depression occurred. This abortive depression was due to an incomplete *Ganzfeld*. It was found later that the luminance difference at the edges of a 180° half-cylinder screen caused the depression. When a dark stripe 1° wide was shown at the edges, the depression became stronger than with diffuse field illumination. When the grating pattern was shown in the peripheral visual field 60° from the edges while at the same time the central 60°, where the eyes were normally directed, was left homogeneous, a strong depression occurred, although the degree varied in different conditions. This suggests that the depression was caused by signals arising in the retina where the ganglion cells over a wide area were excited as the image of the grating pattern was shifted by eye movements.

(5) *Quantitative evaluation of the cortical facilitation*

The behaviour of cortical responses differed markedly from that of geniculate responses. When tested in the patterned visual field, cortical responses were depressed in the period from 10 to 60 msec, then enhanced for about 150 msec. At the transition (60–70 msec), their amplitude was highly variable, as seen by the large standard deviation. Facilitation also occurred in complete darkness and diffusely lit environments. It occurred with a longer delay than for geniculate depression. Facilitation started at approximately zero delay, where the geniculate depression was already prominent, and reached a peak at approximately 150 msec. The early depression seen in the patterned environment was due to reduced afferent volleys. Since the discharges of geniculate cells were strongly suppressed, as seen in the reduction of both geniculate response and the presynaptic C_1 component of cortical responses, we conclude that the volume of impulses reaching the cortex would be markedly reduced.

Facilitation, however, appeared to arise in another mechanism, Although the size of the afferent volley reaching the cortex was constant during the stimulus cycle in complete darkness, as seen in the constant size of genicu-

late responses and the presynaptic C_1 component, the post-synaptic C_3 and C_4 components became larger and led to a strong depolarization in the cortical cells, as indicated by a larger C_5. The fact that it occurred also in complete darkness and the modification occurred exclusively in the post-synaptic components suggests that the signals were of extraretinal origin, and may have arisen in the oculomotor system. They converge on cortical cells and increase their responsiveness to orthodromic impulses. By testing cortical responses to chiasmatic stimulation, Kiyono & Iwama (1965)

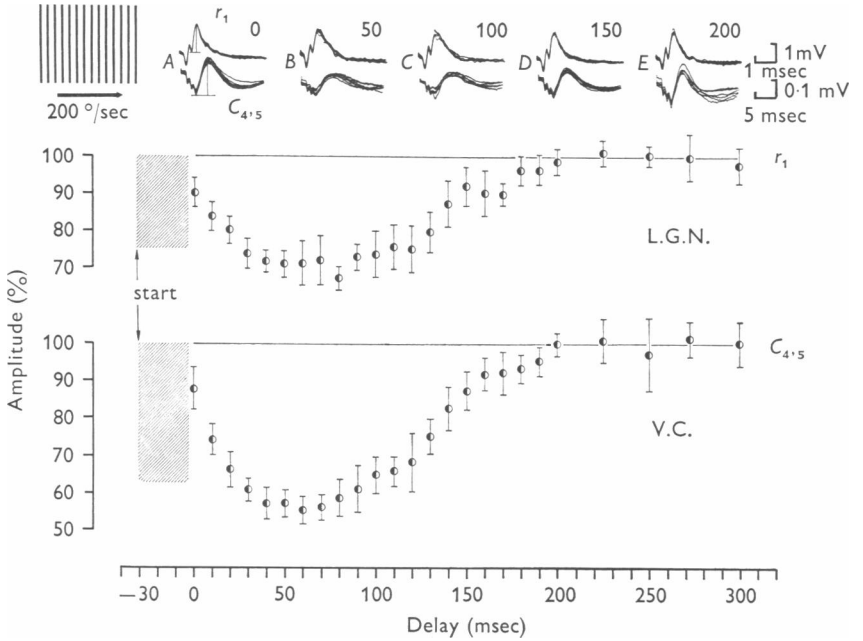

Fig. 4. Changes in amplitude of r_1 (post-synaptic response of lateral geniculate) (upper) and C_4–C_5 (post-synaptic response of visual cortex) (lower) to electrical stimulation of the optic chiasm, following a quick displacement of grating at an angular velocity of 200°/sec. Shaded areas represent the duration of movement of the grating. Control was obtained when the grating was stationary and changes in amplitude are expressed as percentages. Each circle represents an average amplitude of 30 consecutive responses and the vertical bar represents 1 S.D.

observed that the effect of electrical stimulation of the pontine reticular formation was biphasic in dim environments. Initial inhibition lasted about 50 msec and was replaced by facilitation which peaked at 150 msec. The whole effect lasted about 400 msec. Our results in the patterned field are almost identical, except that decay of facilitation was faster in our data. Our findings would thus support a role for the pontine reticular formation as a source of the oculomotor signals.

(6) *Changes in geniculate and cortical responses following target motion*

The preceding data showed that depression of geniculate responses was attributable to signals arising in the retina and that facilitation of cortical responses was due to saccade-contingent extraretinal signals. This conclusion was further supported by the results shown in Fig. 4, where the grating pattern was moved across the tangent screen at approximately 200°/sec, while the eyes were steady. This speed is comparable to the average angular velocity of saccadic eye movements of the cat (Stryker & Blakemore, 1972).

Geniculate and cortical responses were tested, starting 30 msec after the onset of the target motion in the same way as during the course of eye movements. A prominent depression occurred in both geniculate and cortical responses, peaking at approximately 70 msec after onset of target motion. Facilitation in the cortical responses seen during eye movements did not occur. The finding that depression in both geniculate and cortical responses was comparable would support the interpretation that depression of the cortical response seen here and during the early period following an eye movement (Fig. 3*B*) reflected the reduced afferent volleys reaching the cortical domain. Similar results have been obtained in other experiments with immobilized cats, in which the grating pattern was moved by driving the mirror attached to the galvanometer with potentials of e.o.g.s previously recorded on magnetic tapes. This simulated the image motion which might occur on the retina during a saccadic eye movement.

DISCUSSION

This study shows that excitability of cells at lateral geniculate and cortical levels of the visual pathway is differently affected in the course of a saccadic eye movement. The geniculate response to orthodromic volleys was depressed, whereas the cortical response was enhanced. The difference occurred not only in the nature of the altered response but also in the behaviour of these responses to changes in the visual environment. This suggests that the modification was induced by two independent mechanisms which involve signals arising in different sources.

(1) *The mechanism for depression of geniculate responses*

The excitability of geniculate cells was depressed for about 150 msec, reaching a trough of approximately 100 msec after the initiation of an eye movement. The effect was predominantly post-synaptic, as indicated by the unchanged amplitude of the presynaptic component t_1.

Single unit recording from identical preparations showed that the firing probability of geniculate cells to a chiasmatic stimulation was markedly

reduced during the course of eye movements (Noda & Adey, 1973). These effects were dependent on the visual environment in which the eye movements occurred. On the other hand, a prominent depression occurred when the grating was abruptly moved, simulating the image motion which might occur on the retina during a saccadic eye movement. These results would indicate that the depression in geniculate responses was caused by signals arising in the retina where the ganglion cells would be excited as the image of the grating was moved by an eye movement.

By recording single unit activity in the optic tract of alert cats, we have found in other studies that one class of ganglion cells discharged in bursts with eye movements, and another showed sustained discharges in certain directions of gaze, reflecting local luminance (H. Noda & W. R. Adey, unpublished). In 93·9 % of the units of the first class, which we designated T (transient) units, the response properties were identical with those of Y-cells of Enroth-Cugell & Robson (1966) and they had a specific sensitivity to a quick target motion. From the fact that they have receptive fields in relatively peripheral portion of the retina and their excitability is affected by an object movement shown at 20–50° from the receptive field centre, known as the 'periphery effect' (McIlwain, 1964; Cleland, Dubin & Levick, 1971; Ikeda & Wright, 1972), the T units would be ideal candidates for transfer of signals on image motion. They showed transient responses to image motion caused by eye movement and also responded with an identical burst to a quick shift of grating pattern (H. Noda & W. R. Adey, unpublished).

(2) *The mechanism for facilitation of cortical responses*

Excitability of visual cortex to orthodromic volleys was enhanced for about 200 msec after onset of eye movement, reaching a peak at approximately 150 msec, and also occurring in total darkness. During eye movements in total darkness, the volume of impulses from the lateral geniculate nucleus remained the same, as measured by presynaptic response components, yet the post-synaptic C_3 and C_4 components increased, suggesting that the facilitation was post-synaptic and caused by impulses impinging on the cells through a route independent of the geniculo-striate pathway. Simulated image motion without eye movements did not produce the effect. The fact that it occurs only in association with eye movements suggests that the facilitation was caused by signals from a system closely related to eye movements and not located in the retina. If this were the case, afferent impulses would reach the cortex and impinge on the visual system simultaneously with the efferent discharge which gives rise to an eye movement. The existence of such 'corollary' discharges has been postulated to account for the perceptual constancy of environment during

active eye movements (Sperry, 1950; von Holst & Mittelstaedt, 1950; Teuber, 1960). In this scheme, the visual cortex would represent the first level in the geniculo-striate system subserving mechanisms of oculomotor-visual integration. The existence of such an integration in the striate cortex has also been suggested by modified cell activity during eye movements. Some single units in striate cortex of the alert cat were facilitated for about 200 msec following saccadic eye movements in complete darkness (Noda, Freeman & Creutzfeldt, 1972).

(3) *A critique of contributing physiological mechanisms*

The suggestion has been made that the oculomotor-visual integration occurs in the lateral geniculate nucleus. Bizzi (1966*a*) showed that the majority of geniculate cells discharged in bursts in complete darkness 15–20 msec after initiation of rapid eye movements during REM sleep and suggested that the bursts were due to corollary discharges. He also demonstrated depolarization of optic tract terminals 10–15 msec after eye movements (Bizzi, 1966*b*), suggesting a presynaptic inhibition associated with eye movements. Similarly, presynaptic inhibitions was observed in lateral geniculate nucleus during 'tracking eye movements' of midpontine pretrigeminal cats (Kawamura & Marchiafava, 1968). Burst activity of geniculate cells with rapid eye movements of sleep was observed by Sakakura (1968). During vestibular nystagmus in the encéphale isolé cat, Jeannerod & Putkonen (1971) found that activity of 66% of geniculate cells was modified. By recording multiple unit activity from the optic radiation of encéphale isolé cats, Corazza & Lombroso (1970) also found a transient increase in spike discharges associated with rapid eye movements.

In spite of abundant observations suggesting existence of oculomotor-visual integration in the lateral geniculate nucleus, we failed to detect any signs of a corollary discharge during spontaneous eye movements. Although depression of the geniculate responses occurred with eye movements, it was due to signals arising in the retina. During these periods of depression, the amplitude of presynaptic components showed no changes and the effect was exclusively on post-synaptic elements. It is possible that subtle modifications occur in the functional connexions between the lateral geniculate nucleus and the oculomotor centre, depending on states of alertness. Such a possibility has been suggested by Bizzi & Brooks (1963) from their observations during REM sleep. Potentials in the lateral geniculate nucleus associated with eye movements have been commonly observed during REM sleep of the cat (Jouvet & Michel, 1959; Bizzi, & Brooks 1963; Mouret, Jeannerod & Jouvet, 1963; Munson & Schwartz, 1972). However, from locations in the lateral geniculate nucleus showing typical orthodromic and antidromic responses, we could not record such potentials

associated with eye movements in our alert cats, confirming Ogawa's observation (1972) in the squirrel monkey. During REM sleep the majority of geniculate cells discharged in burst with rapid eye movements (Bizzi, 1966). However, after testing approximately 450 geniculate cells in alert cats, we did not find any cells which changed activity with eye movements in complete darkness, although all cells showed either transient or sustained changes or both in firing when the eyes moved in the light (H. Noda & W. R. Adey, unpublished). This confirms the recent studies in the rhesus monkey (Büttner & Fuchs, 1973) in which most geniculate cells failed to fire with eye movements.

Another possible basis for the disagreement might relate to observations during different types of eye movement. When eye movements were elicited by direct or indirect stimulation of the brain stem oculomotor centres, it appears likely that such stimulation itself would cause not only changes in levels of alertness, but also aberrant volleys which might impinge upon the geniculate cells. In testing orthodromic responses of the lateral geniculate nucleus in monkeys, Doty, Wilson, Barlett & Pecci-Saavedra (1973) observed that the mesencephalic reticular stimulation caused a biphasic effect on the responses, with an initial inhibition followed by a facilitation. This facilitation was not found in the present study during spontaneous eye movements. A similar explanation may be given for the observation of Jeannerod & Putkonen (1971) during vestibular nystagmus in the encéphale isolé cat. Although they found that about one third of geniculate cells changed activity with nystagmic eye movements in complete darkness, we did not find any responsive cells during spontaneous eye movements (H. Noda & W. R. Adey, unpublished). During eye movements induced by vestibular stimulation, nystagmic impulses might be transferred to the lateral geniculate nucleus. However, it is not clear that such impulses are comparable to the corollary discharges subserving normal visual perception. During the period of such involuntary eye movements, we are aware that the perceived field is never stabilized, suggesting that the corollary mechanism is no longer operating effectively. In *summary*, it would appear that in normal conditions of active eye movements, the lateral geniculate nucleus does not participate in oculomotor-visual integration.

(4) *Functional significance*

From this study we conclude that the visual cortex is probably a site for oculomotor-visual integration. There is also disagreement in the literature regarding this point. The apparent divergence may be partially explained as an inappropriate interpretation of the corollary mechanism. Saccadic suppression has often been regarded as a manifestation of this

mechanism. Researchers were puzzled by the finding that neurophysiological changes in the visual system occur only after eye movements, as the saccadic suppression was observed before and during eye movements. In fact, except in a thin cell layer located deep in the superior colliculus (Wurtz & Goldberg, 1971; Schiller & Körner, 1971), changes in neuronal activity preceding saccades have not been observed in the visual system.

However, as suggested by Mackay (1970) from psychophysical observations and by neurophysiological data presented here, saccadic suppression might not be due to a corollary discharge. The present study shows that a considerable suppression of visual information in the lateral geniculate nucleus during saccades is due to retinal influences. If this were the case, there remains no ground for the assumption that changes in neuronal activity should precede eye movements. The corollary mechanism would play an important role predominantly during the perceptual stage of visual processing. In conformity with this hypothesis, a peak around 150 msec in the enhancement of the cortical responses does not appear unduly long.

The pathway for the corollary discharge as well as the neurophysiological basis for the long latencies are not yet known. The geniculo-striate pathway has been ruled out by the present study. It may well be transferred through other thalamic nuclei as suggested by Büttner & Fuchs (1973) in rhesus monkeys. They found that although most lateral geniculate neurones did not change activity, neurones in the pregeniculate nucleus showed burst activity 80–90 msec after the onset of an eye movement. Neuronal activity reached its peak 100–200 msec after the onset. This nucleus in the monkey corresponds to the ventral lateral geniculate nucleus of the cat. However, we have not found such neurones in this nucleus. This pathway therefore remains to be determined.

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