CORTICOFUGAL INFLUENCE IN THE MACAQUE LATERAL GENICULATE NUCLEUS

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CENTRIFUGAL influence has been demonstrated in all sensory systems except olfaction, and has replaced the old conception of sensory systems as simple relays of information to the cortex. Corticogeniculate fibers have been demonstrated anatomically (MONAKOW, 1895; RAYMOND Y CAJAL, 1903; TELLO, 1904; MINKOWSKI, 1914; BIEMOND, 1930; NAUTA and BUCHER, 1954; SHOLL, 1955; BERESFORD, 1961; SZENTÁGOTHAI, 1961, 1963, 1964, et al., 1966, GUILLERY, 1967) and stimulation of the cat's visual cortex has been shown to produce both inhibition and facilitation of afferent impulses (WIDÉN and AJMONE-MARSAN, 1960; AJMONE-MARSAN and MORILLO, 1961; IWAMA et al., 1965; KWAK, 1965; SUZUKI and KATO, 1965; VASTOLA, 1967). Previous studies of corticofugal influence in the visual system have been limited to cats, have employed only restricted electrical stimulation or polarization of cortex, and have not been concerned with color vision. The present research was done in an attempt to discover whether or not centrifugal influence could be demonstrated in the monkey's lateral geniculate nucleus (LGN), if so, what its major effect on afferent information is, and whether it may be implicated in some aspect of the monkey's excellent color vision.

METHODS

The animals used in these experiments were 14 Macaca irus monkeys, aged 2-5 yr. In two cases surgical and recording procedures were carried out with one hemisphere of the brain, and the animal allowed to recover for a minimum of 3 weeks; then the other hemisphere was prepared. Thus 16 recording sessions were involved, each from a different hemisphere and most from different monkeys.

Surgical preparation

Each animal was anesthetized with an intrathoracic injection of 35 mg/kg of pentobarbital sodium (Nembutal). The anesthetized animal was placed in a stereotaxic instrument, and a cannula was inserted into the lesser saphenous vein for intravenous administration of anesthetic. The skull was bared and a trephine opening was made over the expected location of the LGN. The dura was cut away and the cortical surface was

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protected with agar. A second opening was made over the visual cortex, dura intact, in which a 2 cm² thermoelectric cooling device (Cambion, #3954-1) was placed. The cooling device was located immediately posterior to the lunate sulcus, and immediately lateral to the falx cerebri. Transferred heat was removed from the device by means of cool water circulating through an attached aluminium reservoir. The device was battery-powered and caused no electrical interference. A Yellow Springs Instrument #421 thermistor was placed against the dura directly beneath the cooler and attached to a Y.S.I. Thermistemp Controller. This permitted continuous monitoring of cortical temperature.

The animal’s eyes were refracted, dilated with Cyclogyl, and fitted with corrective or plano contact lenses. Polyethylene rings attached to the stereotaxic frame held the eyelids open. The animal’s rectal temperature was held at 34° by a heating pad placed under the animal’s body and connected to a second Yellow Springs Instrument Thermistemp Controller and a rectal thermistor probe.

**Optical system**

The system by which photic stimuli were administered is diagrammed in Fig. 1. Light originated from a tungsten source and passed through a Bausch and Lomb mono-
After exiting from the monochromator, the beam was passed through a small box containing a thin, optically neutral sheet of glass, diagonally placed to reflect a small amount of light to two photocells. Readings from these photocells were used to make settings of a variac connected with the monochromator in order to obtain an equal energy spectrum. The beam was then collimated and passed through two filter wheels: the first contained 0.0 through 0.9 units of Inconel neutral density filters in 0.1 log units steps; the second contained 0.0 through 4.0 units of Inconel filters in 1.0 log unit steps, and a beam block. From the filter wheels the light passed through an iris diaphragm and a beam splitter, and was brought to a focus in the plane of a shutter activated by a solenoid. The light was again collimated and then reflected by a beam splitter through an angle of 90°. Here it was again focused to traverse the base leg of the system, and recollimated before passing through a field aperture and striking a moveable silvered prism. The prism reflected the beam 90° into a focusing lens. The animal's left eye was then placed at the focal point (i.e. in Maxwellian view). The moveable prism was next adjusted so that the right eye was in Maxwellian view, and the position of the prism was marked. In this way the light could be shifted from one eye to the other with minimal difficulty.

**Recording system**

Electrical activity of single LGN units was detected by extracellular glass micropipettes filled with 3-M KCl. Tip diameters were approximately 0.5–1 μm, and resistances varied from 2–20 MΩ. Signals were fed through a conventional Medistor cathode/follower, and amplified by an a.c. amplifier. After analog-to-digital conversion, responses of single units were relayed to the recording head of an incremental 8-channel Precision Instrument magnetic recorder and later analyzed by a CDC 3400–3600 computer. On line-monitoring was accomplished by means of Inovac decade counters, a Tektronix 504 oscilloscope and a Heath kit audio amplifier. Background noise was eliminated from the data by means of an adjustable criterion line, visible on the oscilloscope, which was set to exclude low voltage background activity. Electrical interference was not automatically rejected by the system. However, when encountered, its waveform and/or amplitudes were obviously different from single cell responses. Individual sources of interference were located and eliminated. An example of the digital output can be seen in Fig. 2.

**Procedure**

When a light-responsive unit was isolated, its responses to diffuse flashes of monochromatic light (1 sec duration) were recorded. On the basis of its response to different wavelengths it was classified as +Red—Green, +Green—Red, +Blue—Yellow, +Yellow—Blue, broad-band inhibitor, or broad-band excitor, according to criteria established previously (DeValois, et al., 1966). Then either of 2 procedures was followed: (1) responses to 6 wavelengths were obtained for several intensities of light; (2) responses to the single wavelength evoking the greatest number of spikes were recorded during at least 6 and at most 20 separate 1-sec presentations of that stimulus. At this point cortical cooling was initiated, and either procedure (2) was repeated immediately or procedure (1) was repeated after cortical temperature had been reduced to 15°C or less (but not lower than 3°C). Next the cooler was turned off and the cortex was allowed to warm naturally to at least 20°C (about 5 min). Then the whole procedure was repeated. This was continued either until sufficient data could be obtained or until the cell was lost. Recording from another
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Histological examination was performed on all brains, and all probes were located.
Results

Responses of 45 cells were recorded long enough to be assessed adequately during cooling. Many additional cells were encountered in the course of these experiments, but could not be held long enough for complete warm and cool cycles. All units reported were determined to be LGN cells on the basis of their biphasic waveform (see Bishop, et al., 1962). Seventeen of the 45 probably projected to non-cooled areas of the visual cortex, as determined by location in LGN and known projections of areas of LGN to areas of visual cortex (Polyak, 1957). None of the 17 showed any response alteration during cortical cooling. Fifteen of the 28 cells which appeared to project to cooled areas did show response alteration. Eleven of these cells are classified basically as "decreasers". That is, their most dramatic response alteration is a decrease in firing to excitatory stimuli. The decreasers could be further divided into two subgroups: (1) those whose responses fell to the spontaneous rate during cortical cooling; and (2) those whose responses during cortical cooling still showed excitation but were of obviously lower frequency than during normal cortical temperature. (Excitation here refers to an increase in firing rate; inhibition, to a decrease. These terms do not necessarily imply any given synaptic mechanism.)

Four decreaser cells fell into the first subgroup, i.e. their responses to diffuse light flashes could not be distinguished from spontaneous firing. The data from one of these four cells can be seen in Fig. 3. Four complete cycles of warming and cooling were carried out, with high reliability from one cycle to the next. It should be noted that both excitatory and inhibitory responses were lost, but that since the excitatory response was originally the more prominent, its loss was more noticeable. It should also be noted that the cell appears to require varying amounts of time on the several cycles to decrease or increase its response rate. However this apparent difference is due to differing lengths of time between onset (or offset) of cooling and onset of recording on the several cycles. For example, in cycles 1 and 2, the cooler was on for several min before recording began, while in cycle 3, the two procedures were accomplished almost simultaneously. As a result, recording during cycle 3 progressed for several min before the cortex was cool enough for the effect to occur. The change in response rate is not gradual in either direction, although it is continuous. The other three cells in this subgroup responded in essentially identical

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Fig. 3. Photically induced responses (above and below spontaneous rate) of a +Y-B decreaser cell. Responses to wavelengths across spectrum to left of vertical line. Responses to one wavelength (640 nm) to right of line. Stimuli presented for 1 sec at 22 sec intervals. Dotted line represents spontaneous rate, averaged every six trials. ●●●Warm. ▲▲▲Cool.
fashion. One cell was an ipsilateral +B–Y cell; the other three were contralateral +Y–B cells.

Seven cells are classified in the second subgroup of decreasers. Excitatory responses of these cells did not fall to spontaneous level during cortical cooling, although response rate was clearly diminished. A typical example of responses of these cells can be seen in Fig. 4. Excitatory responses during cooling to even the brightest stimuli (0·0 neutral density) were lower than those to the dimmest stimuli (1·0 neutral density) during normal cortical temperatures. Furthermore there seems to be a slight decrease in the strength of the inhibitory response. All seven cells followed this same pattern of response alteration; the cell depicted in the figure is truly typical. Of the seven, five were +Y–B and two were +B–Y cells. They ranged in geniculate location from the caudal extremity to the large middle portion, and were found in contralateral and ipsilateral layers.

The second basic type of corticofugal effect shown by geniculate cells is an increased rate of firing to excitatory stimuli during cooling. Four cells were affected in this manner. The responses of one of these are shown in Fig. 5. Repeat measures were obtained on two of the increasers, showing the increase to be reliable. T-tests were performed on responses of the two which were subjected to complete cooling before recording began. The difference between pre- and during-cooling responses were significant well beyond the 0·01 level (t=5·72**, t=7·33**). The other two were ill-suited to this test since recording began while the cortex was just beginning to be cooled, and the observed increase was fairly gradual. Only the peak excitatory responses of these cells were observed, so any possible effects on inhibitory responses cannot be determined. Of the four increasers, one was +R–G, two were +Y–B, and one was +G–R. All were contralateral and were located in the most dorsal layer (layer 4 in the caudal extremity and layer 6 in middle sections).

Almost all of the 30 cells not affected by the cooling procedure were quite constant in their responses to light stimuli. It was in contrast with the response stability in cell after cell that the corticofugal effects stood out so clearly.

If the cells showing response alteration are separated according to opponent type, it can be seen that +Y–B and +G–R cells are affected more frequently than +B–Y and
Ten of 13 +Y–B cells and 3 of 4 +G–R cells showed response alteration, as compared with 1 of 6 +B–Y cells and 1 of 4 +R–G cells. Perhaps the clearest separation of effects on the different cell types can be seen by comparing cells on the same probe. Whenever no cell on a given probe was affected, the questions can be raised whether these cells did in fact project to the cortical area being cooled, and whether the cooling did effectively ablate the prescribed area of visual cortex. However, when some cells on a given probe were affected by cortical cooling and nearby cells on the same probe were not affected, it is likely that these cells differentially receive corticofugal influence. Fifteen cells on four probes were differentially affected in this manner. Three out of the seven affected cells were +Y–B, three were +G–R, and one was +R–G. Of the eight non-affected cells, four were +B–Y, two were +R–G, one was +G–R, and one was +Y–B. The responses of +Y–B and +G–R cells then, are altered by cortical cooling more frequently than +B–Y and +R–G cells. The significance of this distribution of effects is not presently understood.

Table 1 shows the spontaneous firing rates during normal and cooled states for each

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<th>Avg. spontan. rate of cells showing no effect on photic responses (spikes/sec)</th>
<th>Avg. spontan. rate of cells showing decrease of photic responses (spikes/sec)</th>
<th>Avg. spontan. rate of cells showing increase of photic responses (spikes/sec)</th>
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<td>Warm</td>
<td>10.1</td>
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<td>Cool</td>
<td>10.0</td>
<td>8.4</td>
<td>9.6</td>
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of the following three groups: decreasers, increasers, and no-effect cells. For no-effect cells there is a difference of only 0.1 spike/sec between measurements during warm and cooled cortical temperatures. The decreasers show a reduction during cooling in average spontaneous rate of 0.6 spikes/sec. The increasers increased their spontaneous rate an average of 1.1 spikes/sec during cooling. T-tests were used to compare pre- and during-cooling spontaneous firing rates for decreasers and increasers. In both cases the difference was non-significant (t = 0.44 and t = 0.46 respectively). Thus corticofugal influence has little if any effect on spontaneous firing.

DISCUSSION

As noted above, the excitatory response rates of 11 cells were decreased during cortical cooling. In the case of four decreaser cells all responses were indistinguishable from spontaneous levels of firing. If a cell loses its ability to respond at all when cortical efferents are functionally ablated, then obviously such afferents are essential for any response it may give. Responses of seven other decreasers did not fall to the spontaneous firing level, and therefore seem to be in a somewhat different category than those discussed above. Clearly the optic afferents are capable of firing the cells in the absence of efferent influence, although not with as high a frequency as before the influence is removed. Inhibitory responses are also still present during cortical cooling, but are diminished in strength. In the case of these cells, corticofugal influence can be inferred to amplify both excitatory and inhibitory responses to light.

It is impossible to determine at present whether the two groups of decreasers represent one quantitative continuum of effects or two qualitatively different patterns. In other words, the ratio of corticofugal to optic influence may vary among cells on a continuum of 0-1. Decreasers whose response rate falls to spontaneous levels during cortical cooling would be at one extreme. Other decreasers would fall in the middle, while no-effect cells would fall at the other extreme (Increasers must be considered separately.) On the other hand, two discontinuous types of corticofugal influence may be involved here. In this case some decreasers would respond (under present experimental conditions) only while corticofugal influence was intact. Other decreasers would respond to optic impulses without corticofugal influence, but these responses would be amplified by the corticofugal impulses.

As noted in the Results section, four other cells increased their peak firing rates during cortical cooling. Inhibitory responses of these cells were not examined during cooling. Therefore corticofugal effects on such responses cannot be determined at present.

The relative paucity of during-cooling increases in response rate (signifying removal of inhibitory effects) is somewhat at variance with several previous experiments (WIDÉN and AJMONE-MARSAN, 1960; AJMONE-MARSAN and MORILLO, 1961; IWAMA et al., 1965; KWAK, 1965; and SUZUKI and KATO, 1965), which demonstrated a predominantly inhibitory effect of cortical stimulation on geniculate neurons. However, the present results are in the same direction as those of VASTOLA (1967). Furthermore, Vastola found both excitatory and inhibitory effects on the same unit in most cases, as did the present experiments. There seems to be no simple explanation for the differences in predominant effect on the basis of different preparations, since the same type of preparation (unanesthetized, with mid-collicular transection) showed predominantly inhibitory influence in two studies (WIDÉN and AJMONE-MARSAN, 1960, and AJMONE-MARSAN and MORILLO, 1961) and a predominantly excitatory influence in another (VASTOLA, 1967). However, it should be
noted that those authors at variance with the present results used either single shocks or trains of shocks to the visual cortex. This is a very artificial kind of stimulus.

**Spontaneous activity**

There was little, if any, effect of cortical cooling on spontaneous levels of firing (see Table 1). The slight differences observed were in the expected directions, i.e. spontaneous rates of decreasers decreased slightly during cooling, while those of increasers increased slightly. There was much variability in spontaneous activity, however, and the observed differences were non-significant according to t-tests performed on the data.

**Location of cells in the LGN**

A composite map of probes located histologically is shown in Fig. 6. The location of

![Composite map of probes through LGN](image)

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Fig. 6. Composite map of probes through LGN. Caudal to rostral.
of depth from the first cell encountered, as well as the number of changes from contra-
lateral to ipsilateral cells, were compared with the histological slides in order to place
cells on the composite map. There is a slight bias toward sampling in the caudal end, and
a more considerable bias toward sampling from the top two layers. The latter is under-
standable in the light of the topography of the LGN: these layers are proportionately
larger than the others and are the first to be contacted by the descending electrode. Fur-
thermore, the top layers bend around so that only a medially placed electrode will eventually
contact the lower layers. Also, the proportion of cells in the lower two layers which are
responsive to diffuse light may be somewhat smaller than in the upper layers (WIESEL
and HUBEL, 1966).

There is a topographical distribution of retinal projections to the LGN and of geniculate
projections to visual cortex. The area centralis of the retina projects to all of the caudal-
most LGN as well as to a wedge-shaped area of the central LGN, the large part of the
wedge falling at the bend of the "knee." The wedge then tapers to a point at the hilum,
and is flanked on both sides by projections from the more peripheral areas of the hemi-
retinae. (The left half of each retina projects to the left LGN and the right half to the
right LGN.) The center of fixation projects to a line along the uppermost part of the bend
of the "knee", along the center-back of the foveal wedge (POLYAK, 1957).

The geniculate "foveal" fibers project to the temporal-most area of the striate cortex.
Fibers representing the zone 10° from the center of the fovea project along the caudal,
medial portion of the flat dorsal visual cortex. Fibers from intermediate zones, in order
of distance from the center, project between the lateral foveal zone and the medial 10°
zone (TALBOT and MARSHALL, 1941). More peripheral portions of the retina are represented
in the Calcarine sulcus and along the central sulcus (DANIEL and WHITTERIDGE, 1961).

The cooling device in these experiments was placed in a position to cover the largest
flat area of visual cortex possible. Thus, the cortical zones representing approximately
2–10° from the center of the fovea were cooled.

Specificity of location of corticofugal influence

There is a topographical pattern of cells whose firing rates were altered by the cooling
procedure. These cells were located in the caudal portion of the LGN and along the edges
of the foveal wedge of the central portion. Of the 17 geniculate cells which fairly clearly
did not project to the cortical area being cooled, none were affected by the cooling pro-
cedure. Six of these were located in the center of the foveal wedge, four were considerably
outside the wedge, and seven were in the anterior portion of the LGN. If corticofugal
influence were diffusely directed to large areas of the LGN, a given geniculate cell would
likely show response alteration during cortical cooling even though its axon did not
project to the area being cooled. The lack of any response alteration of cells not projecting
to cooled areas indicates a more discreetly directed influence. In other words, if a cell's
axon does not project to the area of cortex being cooled, the cortical cooling will have
no effect on its responses. Thus a relatively specific "loop" seems to be involved. Since
the cooled area was slightly more than 2 cm^2 and since it is impossible to determine the
exact projection of a geniculate cell, no estimate of discreteness can be made from the
present data. Some spread of the influence seems likely on the basis of observations of
SZENTÁGOTHAI et al. (1966). Cooling an axon terminal has no direct effect upon the
initiation of spikes at the axon hillock (C. D. BARNES, personal communication). Thus
the specificity is not due merely to an experimental artifact.
Discussion of cooling technique

A number of studies have investigated effects of hypothermia on electroencephalogram (EEG) and electrocorticogram (ECG) activity (Byck and Dirlik, 1963; Callaghan, et al., 1954; Massopust, et al., 1964a and b; Pasztor, et al., 1965; Ten Cate, et al., 1949; Woodhall and Reynolds, 1958). The usual finding is that EEG and ECG activity become steadily lower in amplitude and slower in frequency, until activity disappears at about 20–21°C. When the cooling is stopped and the brain allowed to warm naturally, activity reappears in the form of small bursts of low amplitude activity at about 21°C, and fast, normal activity reappears at 30°C. No long-term deleterious effects of the cooling have been observed either on cortical activity (Callaghan, et al., 1954; Woodhall and Reynolds, 1958; Donald and White, 1962; Bick and Dirlik, 1963), behavior (Mark, 1961; Bigelow, et al., 1953), or on performance of tasks previously learned (Callaghan, et al., 1954).

When whole animals were cooled, electroretinogram (ERG) a and b waves dropped out at about 24°C esophageal temperature (Massopust, et al., 1964b), and cortical and subcortical evoked potentials were flat at 18–20°C rectal temperature (Massopust, et al., 1964a). Activity of pulmonary stretch receptors is blocked at 10°C (Dawes, et al., 1951).

In light of this wide variability of effective temperatures, the present experiments included recording of photically evoked potentials from the visual cortex of two monkeys during cortical cooling. The stimuli were intense diffuse flashes of light. The results of these records are presented in Fig. 7. Before cooling, the animal's rectal temperature

![Fig. 7. Evoked potentials from visual cortex during cortical cooling and natural warming. Computer of Average Transients: records are average of 30 responses; duration of 1 sec from onset of flash. Stimulus: 6W fluorescent tube, 10 msec, 110 V.](image-url)
was 37°C and cortical temperature was 35°C. As can be seen, the evoked potential decreased steadily in amplitude. The evoked potential was approximately flat at 15°C, and the cortex was then allowed to warm naturally. The amplitudes of evoked responses during warming increased steadily until they assumed approximately their pre-cooling levels. In addition cortical temperature was monitored during recording of single units. These records are in general agreement with the evoked potential data. Most of the affected cells showed a fairly sharp, though continuous alteration, which began at about 17°C and was complete at 13–15°C cortical temperature.

**SUMMARY**

1. The activity of single units in the lateral geniculate nucleus was recorded before and during diffuse monochromatic stimuli. Corticofugal influence was measured as the difference in firing rate between responses to the same stimuli recorded before and during reversible cortical ablation by cooling.

2. The predominant effect on 11 of the 28 cells projecting to cooled cortical areas was a decrease in response rate to excitatory stimuli, probably implying a removal of corticofugal facilitation.

3. The predominant effect on 4 of these 28 cells was an increase in response rate, implying removal of corticofugal inhibition.

4. There was no response alteration of any of the 17 cells projecting to non-cooled cortical areas, and little or no alteration of spontaneous activity in any cell.

5. The cell types most often altered were +Green—Red and +Yellow—Blue. These cell types also showed higher response rates than their “mirror-image” types, i.e. +Red—Green and +Blue—Yellow.

**Acknowledgements**—The author expresses sincere appreciation to R. L. DeValois, G. P. Frommer and J. I. Boles for their encouragement and assistance throughout the course of this study.

**REFERENCES**


Corticofugal Influence in the Macaque Lateral Geniculate Nucleus


Abstract—Responses of single cells in the macaque lateral geniculate nucleus were recorded through extracellular micropipettes. Spectral sensitivity of each cell was determined by recording its responses to 6 diffuse monochromatic photic stimuli. Activity in a predetermined limited area of striate cortex was then temporarily removed by cooling and responses to the same photic stimuli were recorded. Responses of approximately half the units projecting to the cooled cortical area were altered during cooling. A decrease in responses to excitatory stimuli was predominant, implying removal of corticofugal excitation. Some cells increased their responses to excitatory stimuli during cortical cooling, implying removal of corticofugal inhibition.

Résumé—On enregistre avec des micropipettes extracellulaires les réponses de cellules isolées du corps latéral genouillé chez le macaque. On détermine la sensibilité spectrale de chaque cellule en recueillant les réponses à 6 stimuli monochromatiques diffus. Ensuite on supprimait temporairement l’activité dans une aire limitée du cortex strié en la re-
froidissant et on enregistrait les réponses aux mêmes stimuli lumineux. Durant le refroidissement, les réponses d'environ la moitié des unités qui se projetaient dans l'aire corticale refroidie se trouvaient altérées. En majorité on constatait une diminution dans les réponses aux stimuli excitants, ce qui implique la suppression d'une excitation corticofugue. Quelques cellules augmentaient leurs réponses aux stimuli excitants durant le refroidissement cortical, ce qui implique la suppression d'une inhibition corticofugue.


Резюме — Реакции отдельных клеток в ядрах латеральных коленчатых тел макаки были зарегистрированы экстралекточно с помощью электродов — микропипеток. Спектральная чувствительность каждой клетки была определена путем записи ее реакций на 6 диффузных монохроматических световых стимулов. Затем активность предварительно определенной ограниченной области стриатной коры была временно выключена охлаждением и снова записаны ее реакции на те же самые световые стимулы. Реакции приблизительно половины клеток, имеющих проекцию в охлажденной кортикальной области, были изменены во время охлаждения. Преобладало понижение реакций на возбуждающие стимулы, что предположительно означало выключение кортикофугального возбуждения. Некоторые клетки увеличивали свои реакции на возбуждающие стимулы, что могло означать выключение кортикофугального торможения.