Composition of Geniculostriate Input to Superior Colliculus of the Rhesus Monkey

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SUMMARY AND CONCLUSIONS

1. In order to examine the composition of the geniculostriate input to the superior colliculus, microelectrode recordings were undertaken in this structure of the rhesus monkey while parvocellular or magnocellular laminae of the LGN were reversibly inactivated by injecting minute quantities of lidocaine or MgCl₂.

2. The inactivation of magnocellular laminae disrupted the visually driven activity of most cells in the topographically corresponding areas of the colliculus, but not in the superficial retinotectal recipient zone. The inactivation of parvocellular lamina had no effect on the visually driven activity of collicular cells.

3. Several controls were carried out to rule out the possibility of intervention with fibers of passage. We ascertained that the LGN injections did not affect the direct retinotectal pathway by comparing the effect of such inactivation with the effect produced by reversibly cooling visual cortex. These two manipulations yielded similar results: cells in the most superficial regions of the superior colliculus were unaffected by both cortical cooling and by magnocellular injections, while below this region the response of collicular cells was reduced or eliminated in both cases.

4. These results suggest that the indirect visual pathway to the superior colliculus via cortex is activated selectively by the broad-band system, which is relayed through magnocellular LGN. The color-opponent system does not appear to have a corticotectal input sufficient to drive collicular cells independently.

INTRODUCTION

The superior colliculus in mammals receives a visual input both directly from the retina and indirectly from the visual cortex (5, 7, 8, 14, 20, 21, 23). The direct retinal input in the monkey is selective in that only two of the three functionally distinct classes of retinal ganglion cells project to the colliculus (26). These three classes in the monkey are the color-opponent, the broad-band, and W-like cells (3, 26). The retinotectal pathway is comprised of the broad-band and the W-like cells. This is similar to what has been reported for the cat. Of the three major classes of retinal ganglion cells discerned in this species, the X, Y, and W, it is the Y- and W-cells that project to the superior colliculus (1, 6, 8).

The lateral geniculate nucleus (LGN) of the rhesus monkey receives input from both the color-opponent and broad-band cells of the retina. The projection sites of these two classes of cells appear to be spatially segregated in the LGN (4, 27, 31). The color-opponent cells terminate in parvocellular laminae (layers 3–6); consequently, most cells in these laminae have color-opponent properties. The broad-band cells of the retina terminate in the magnocellular LGN layers (layers 1–2), whose principal cells projecting to cortex also have broad-band receptive-field properties. The parvo- and magnocellular terminal arborization zones remain segregated in the visual cor-
tex: the parvocellular input terminates predominantly in layers 4A and 4\(_{c,\beta}\), while the magnocellular projection terminates predominantly in layer 4\(_{\alpha}\) (12, 16).

The corticotectal pathway from visual cortex plays an essential role in the control of collicular activity. In the monkey, disruption of this pathway eliminates all visually driven activity in the intermediate and deeper layers of the superior colliculus, but has little or no effect on cells in the most superficial portions of this structure (28). In striate cortex, the corticotectal cells have complex-type receptive-field properties and reside in layer 5 (5, 24).

The aim of this study was to determine the extent to which the indirect visual pathway to the superior colliculus via visual cortex is specialized. Specifically, we wanted to find out to what degree the color-opponent and broad channels contribute to the cortically driven activity of the superior colliculus in the rhesus monkey. In order to examine this question, we reversibly inactivated either the broad-band or color-opponent pathways at the level of the LGN while recording from visually driven cells in the superior colliculus. Hoffman and Straschill (11), Hoffman and Sherman (9), and Toyama, Matsunami, and Ohno (30) have suggested that in the cat the indirect visual input is supplied predominantly by the Y channel, which is believed to be analogous to the broad-band channel of the monkey.

**METHODS**

Three juvenile rhesus monkeys were used to obtain the data for this study. In addition, the methods employed for the reversible inactivation of the geniculostrate pathways were tested in several other animals. For all recordings the animals were paralyzed and anesthetized with \(\text{N}_2\text{O}\) in a manner described elsewhere (26, 27, 28).

**Superior colliculus recording**

Glass-coated platinum-iridium electrodes were used to record in the superior colliculus. Penetrations were made at a 30\(^\circ\) angle relative to the Horsley-Clark AP plane. This angle made it possible to place the penetrations almost at right angles to the superior colliculus. Although it would have been desirable to increase this angle a little, we did not do so because the guard tube containing the microelectrode would have had to pass through the medial portion of striate cortex. We used a mechanical microdrive, which provided a reliable assessment of the location of the electrode tip in depth. The microdrive was designed and built by J. Chubbuck.

**Selective disruption of geniculostrate pathway**

In order to disrupt selectively the broad-band and color-opponent channels to visual cortex, small amounts of 2% lidocaine or 40 mM MgCl\(_2\) were injected into either the magnocellular or parvocellular laminae of the LGN. The lidocaine was pressure injected through a recording-injection pipette developed by Malpeli and Schiller (22), which is illustrated in Fig. 1A. This is a metal-coated pipette insulated with varnish except for a small ringlike exposure at a location 50–150 \(\mu\text{m}\) from the tip. This area serves as a recording surface that is used to determine 1) proper electrode placement in the LGN, and 2) the effects of injections.

The consequences of lidocaine and MgCl\(_2\) injections within the LGN were examined in several animals prior to this experiment. The area affected was assessed 1) by recording from the electrode at various distances from the injection site along a penetration, and 2) by placing several other recording electrodes near the injection site. Data obtained in this fashion showed that 25 nl of lidocaine or MgCl\(_2\) inactivate an area of approximately 300 \(\mu\text{m}\) in diameter. With such small regions of inactivation it is possible to confine the block to a single lamina. The duration of the block varies somewhat from animal to animal and tends to decrease slightly with repeated injections. The range for lidocaine is 3–15 min, and for MgCl\(_2\) it is 1–5 min.

We have made up to 30 repeated injections at the same LGN site without discernible damage as assessed by return of unit activity and by light microscopic examination of tissue stained with cresyl violet.

**Procedure for selective inactivation**

Since the reversible inactivation of the LGN is restricted to a small area, it is necessary to use procedures that assure us that the cells that are to be blocked in the LGN project, via cortex, to those cells in the superior colliculus that are under study. In order to achieve this we aligned the receptive fields of the LGN cells, where we were injecting, with the receptive fields of the cells from which we were recording in the superior colliculus. Repeated penetrations were undertaken in these two structures until an overlap was obtained for the receptive fields. Once this was accomplished, the response properties of the collicular cells were examined before, during, and after inactivation of the LGN region in
FIG. 1. A: schematic diagram of injection pipette tip. Crosshatchings show region that is metal coated and varnished. Solid black area is the recording surface. Lidocaine or MgCl₂ is exuded at the tip by applying pressure with a syringe that is connected to the upper end of the pipette with polyethylene tubing. The amount injected is determined by watching the meniscus move against a millimeter scale at the upper region of the pipette (not shown), which is not coated with metal. B: schematic drawing of the experimental procedures and the pertinent wiring diagram of the visual system. The color-opponent (C-O) retinal ganglion cells relay their messages to cortex via the dorsal parvocellular geniculate laminae 3–6. The broad-band (B-B) cells reach cortex via the magnocellular laminae (1 and 2) and also project to the superior colliculus (S.C.), which in addition receives input from the W-like (W) retinal ganglion cells. Unit recordings in the superior colliculus are carried out with glass-coated platinum-iridium electrodes. Injection electrodes (A) are placed into a parvocellular, a magnocellular, or into both LGN laminae. Repeated penetrations are undertaken until the receptive fields of the cell sites in the LGN and the S.C. are superimposed on the tangent screen facing the animal. Cross-hatched area above cortex represents a silver plate, which is attached to a thermoelectric probe (not shown) with the aid of which cortex can be reversibly cooled. Holes in the plate permit unit recordings to circumscribe the area affected by the cooling.

The vicinity of the electrode tip. The collicular cells were generally activated through that eye whose inputs were disrupted by the LGN lidocaine injection. This, in most cases, was the eye contralateral to the recording sites as we typically placed electrodes into the top and bottom laminae of the LGN (layers 1 and 6). Exceptions to this were controls, which are described in the RESULTS section.

Controls

It was essential to be certain that the LGN injections had their effects only on the geniculo-striate pathway that projects from the region of LGN that was to be inactivated. Therefore, several controls were instituted to make sure that fibers of passage were not disrupted. Of particular concern are three pathways: those fibers projecting from the retina directly to the superior colliculus, those retinal fibers that project to the parvocellular laminae and could be interrupted by magnocellular injections, and those geniculo-striate axons that originate in the magnocellular laminae and could be disrupted when a parvocellular lamina is injected.

The controls we employed to test for these possibilities will be described in detail in the RESULTS section. The major methodological addition was the placement of a thermoelectric cooling probe over striate cortex. This probe permitted reversible interruption of the corticotectal pathway by lowering the temperature of cortex
to the level that stopped cellular activity. The methods for this were described in an earlier study (28). The portion of the visual field represented by the area to be cooled was determined by recording through several small holes around the perimeter of the cooling plate and represented approximately a 4°-diameter area in the lower visual field, the center of which was approximately 3° from the fovea.

**Procedures for each animal studied**

The basic procedures for this study are shown in Fig. 1. Figure 1A, as already noted, depicts the tip of the injection electrode. Figure 1B is a diagram of the experimental manipulations. Somewhat different procedures were employed in each of the three animals:

**Monkey 1.** A recording metal electrode was placed in the superior colliculus. Injection pipettes were placed into both lamina 1 (magnocellular) and lamina 6 (parvocellular). This procedure permitted assessment of superior colliculus cell activity following inactivation of 1) layer 1 (magnocellular), 2) layer 6 (parvocellular), or 3) both of these regions simultaneously.

**Monkey 2.** A recording electrode was placed in the superior colliculus. An injection electrode was lowered into LGN magnocellular lamina 1 or 2 and a cooling probe was placed on visual cortex. Before terminating the experiment, the cortex was destroyed by placing crushed dry ice on the dura. Both of these procedures enabled us to compare the effects of LGN inactivation with visual cortex inactivation on cell activity in the superior colliculus.

**Monkey 3.** The procedure was similar to that employed for monkey 2. The injection electrode in the LGN was placed either into magnocellular or parvocellular lamina. For the parvocellular inactivation we examined cells in the four-layered region of the LGN, (beyond 20° eccentricity from the fovea) where there is only one parvocellular lamina for each eye.

The receptive fields of collicular cells, particularly in the lower layers, were larger than the LGN receptive fields. Therefore, the light stimulus was generally confined to the receptive field of the LGN site. This was most commonly a flashing spot of 0.25 - 1° in size or a rapidly moving stimulus with a small excursion. To test the

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**FIG. 2.** Effects of lidocaine injection in the LGN on the visually driven activity of collicular cells. Histograms on the left were obtained from a group of collicular cells before, during, and after inactivation of a topographically corresponding region in the dorsalmost parvocellular LGN lamina. This manipulation had no effect. Histograms on the right were obtained from another group of collicular cells when the magnocellular lamina 1 was injected. In this case the inactivation obliterated the visually driven activity in the colliculus. Each histogram shows the initial 300 ms of the on- and off-response to 20 repeated stimulus presentations of a 0.5° square stimulus, which was on for 1 s and off for 1 s. The ordinate shows the total number of spikes per bin. Bin width: 7.8 ms.
spread of the LGN block, in a number of cases the stimulus was flashed at several locations relative to the center of receptive fields of LGN cells at the tip in the injection pipette. Injections were also made in a few cases where the alignment between the LGN and superior colliculus sites was off by 1 or 2°.

RESULTS

In three monkeys 141 collicular sites were examined with microelectrode recordings. Of these, 18 were sites where isolated, single units were obtained, and 123 consisted of multiple-unit activity, which we estimate to have generally consisted of three to eight cells. The effects of lidocaine injection into a parvocellular geniculate lamina alone were assessed for 28 collicular sites, while the effects of magnocellular geniculate injections were determined at 126 sites. For 16 collicular sites alignments were made both for a magnocellular and parvocellular location; at 9 of these 16 sites injections were made simultaneously into both LGN laminae. The magnocellular layer was also injected alone for all 16 of these collicular sites, and the parvocellular lamina was injected alone for 13 of the 16 sites. Such successive sets of injections were always interspersed with sufficient time for LGN recovery. Lidocaine was used for the majority of these injections but for the examination of 54 locations in colliculus, MgCl₂ was injected in magnocellular LGN. Visual cortex was cooled while recording from 25 sites in the colliculus, and the effects of this cooling were compared with successive or simultaneous injections of lidocaine (or MgCl₂) into the magnocellular geniculate.

Effects of parvocellular and magnocellular blocks on superior colliculus

The results obtained with parvocellular and magnocellular LGN injections were straightforward: in almost all cases blocking the parvocellular pathway had no effect on the response properties of collicular cells. By contrast, when the magnocellular geniculate was blocked, most cells in the superior colliculus, with the exception of those located in the most superficial portion of this structure, became either totally unresponsive to visual stimuli or were dramatically reduced in their responsivity. This occurred without a significant reduction of the spontaneous firing rate of these cells. Figure 2 demonstrates these results for two sites in the superior colliculus. At both of these sites multiple-unit activity was recorded. Parvocellular LGN was injected for one site and magnocellular LGN was injected for the other site. Each histogram is the result of 20 repeated stimulus presentations. The stimulus was a 0.5° stationary square presented for 1 s at a cycle rate of 2 s (1 s on, 1 s off). The first 500 ms of the on- and off-responses are shown for each of three conditions: a) response before 25 nl of lidocaine were injected into the LGN, b) the response immediately following such an injection, and c) the response after the LGN recovery from the injection.

The figure shows that the parvocellular injection had no effect on the activity of superior colliculus cells. We observed no significant reduction in activity in any of the 28 cases in which a single parvocellular injection was made.

The magnocellular injection demonstrated in Fig. 2 completely abolished the visually driven activity of the cells. Of the 126 superior colliculus sites examined with magnocellular injections, complete elimination of the visually driven response was produced in 50 cases. The mean depth of these sites from the collicular surface was 380 μm. Dramatic but partial reductions were obtained at 36 sites. The mean depth for these locations was 225 μm. No effect was observed in 40 cases. Of these, 15 represented locations where effects were tested with a significant misalignment between the LGN and collicular sites (1–3°). The remaining 25 unaffected sites were mostly superficial, with an average depth of 124 μm below the surface.

In 16 cases we also examined whether the reductions in superior colliculus cell activity as a result of magnocellular injection could be augmented by simultaneous injection into parvocellular LGN. For three of the sites the reduction in responsivity was somewhat greater with paired injections, although in none of these cases did parvocellular injections alone have any effect.

The results suggest that the superior colliculus receives its major activating input from cortex via the broad-band system, transmitted through the magnocellular LGN.
However, a small contribution from the color-opponent system via cortex cannot be excluded.

Controls

DIRECT RETINOTECTAL PATHWAY. Since the results we obtained were so clear cut and consistent, our main concern was that we might be interrupting not only the geniculostriate pathways but also, as a result of the magnocellular injection, the direct retinotectal projection. Five different procedures were employed to exclude this possibility:

1) Recordings in superficial colliculus. In this area the direct retinal input is pronounced and, therefore, injection into the magnocellular LGN should be fully effective only if the direct pathway is interrupted. This control assumes that we know that we are recording from a site that receives no effective cortical input; otherwise, interruption of the indirect geniculostriate-colliculus pathway might be responsible for the effect on collicular activity. In fact, injections generally had little effect on superficial collicular activity. However, in some cases multiple-unit recordings did show a reduction in activity. In such cases we could not determine, with this control, whether the effects were due to blocking the direct or indirect pathways. Therefore, this control was not entirely satisfactory.

2) Visual cortex ablation. This control was carried out, as was the first one, to be certain that the direct retinotectal pathway was not affected by the LGN injection. When the geniculostriate input to the superior colliculus is disrupted by destruction of visual cortex, injection into magnocellular LGN could have an effect on superior colliculus cells only by influencing the direct retinotectal pathway. We ablated visual cortex at the end of one experiment, and subsequent injections into the LGN had no discernible effects in the superficial sites of the superior colliculus (no visually driven activity was observed at sites deeper than 100–300 \( \mu \)m following visual cortex ablation). An example appears in Fig. 3. Since this recording yielded mostly an off-response, only that part of the record is shown. The data demonstrate that the response of super-

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FIG. 3. Off-responses of a group of collicular cells before, during, and after inactivation of LGN layer 1 in an animal whose visual cortex was destroyed by freezing prior to collection of these data. These histograms show that after cortex is destroyed collicular responses are not affected by lidocaine injection. Stimulus: 0.5\(^\circ\) square, 1 s on, 1 s off; 20 trials per histogram. Bin width: 7.8 ms.
ficial collicular cells after cortex ablation is not affected by lidocaine injection into layer 1 of the LGN.

3) Cortical cooling. If the direct retinotectal pathway is unaffected by lidocaine injection, the results of the LGN block on collicular cells should be similar to what is obtained when cortex is cooled (28). This expectation was fulfilled for most of the 25 occasions when this was tested, where lidocaine injections and cortical cooling were both tested on the same collicular cells. An example for a single cell in the superior colliculus appears in Fig. 4. This figure demonstrates that for cells in the intermediate layers of the superior colliculus, both cooling of cortex and lidocaine injection eliminate the response of cells to light stimulation. The two manipulations yielded comparable results for most of the cases tested.

![Graph showing the effect of magnocellular LGN injection or cortical cooling on a single cell in the superior colliculus located 830 μm from the surface.](image)

**Fig. 4.** Effect of either magnocellular LGN injection or cortical cooling on a single cell in the superior colliculus located 830 μm from the surface. Either of these manipulations produces complete inactivation of the collicular cell. Stimulation and data collection parameters as in Fig. 2.
The results for successive cooling and lidocaine injections for 25 collicular sites are shown in Table 1. In this table the effects of either manipulation are placed into three categories: 1) wipeout of visually driven response, 2) a reduction in responsivity, and 3) no effect. The table shows that cooling of visual cortex tended to yield results similar to those observed when lidocaine is injected into magnocellular LGN. At most of these sites multiunit records were obtained.

We also tested a few sites by injecting lidocaine into the LGN while cortex was blocked by cooling. An example of this appears in Fig. 5 for multiple-unit activity 100 μm from the surface of the colliculus. Cortical cooling produced a slight reduction in activity. Injection into layer 1 at this time produced no further change at this site. The data secured 6 min after rewarming cortex show a full return of the response. Had the direct pathway been affected, the lidocaine injection should have altered the response further.

4) On 54 occasions we used 40 mM MgCl₂ instead of lidocaine. MgCl₂ should reduce synaptic transmission and perhaps calcium inward current at cell bodies, but it should have little or no effect on fibers of passage (2). The results we obtained were similar to what was found with lidocaine, but the duration of the block was typically shorter. In earlier experiments we also examined the effects of saline injections that produced no changes when administered in comparable quantities.

5) All the injections described so far were placed into either layers 1 or 6. We examined 10 sites in the superior colliculus while injecting into LGN layer 2, which has its inputs from the ipsilateral eye. This manipulation increases the distance between the retinotectal pathway and the injection site as a result of the intervening first LGN layer. The responses elicited in collicular cells by stimulating the ipsilateral eye were significantly reduced or completely blocked when LGN layer 2 was blocked. The effects, in other words, were the same as those obtained when LGN layer 1 was blocked while stimulating the contralateral eye.

To test the spread of injection-induced inactivation we performed three tests: in the first one we injected into layer 1 of the LGN (contralaterally innervated) while assessing the response through the ipsilateral eye and found that 25-nl injections usually had little or no effect on the adjacent LGN lamina. In the second the spread of the block was assessed by placing a flashing visual stimulus at several sites on the tangent screen relative to the location of the magnocellular receptive fields. These controls showed that the effects of the injection were highly lo-
calized. When the stimulus was placed 1–2° from the edge of the magnocellular fields at the injection site, the block was no longer effective. In the third test injections were made in the LGN where this site and the collicular site were misaligned 1–3°. Such injections had little or no effect.

MAGNO- AND PARVOCELLULAR PATHWAY CONTROLS. Since parvocellular injections alone had basically no effect on the activity of cells in the superior colliculus, it is unlikely that such injections could have affected the geniculo-cortical fibers that originate in layer 1 of the LGN. However, the possibility still remained that injections into magnocellular LGN disrupted the retinal fibers coming to LGN layer 6. This was tested by recording in layer 6 on several occasions in association with this and other experiments while injecting in the topographically corresponding area of layer 1. In no case was there even the slightest evidence that layer 1 injection had any effect on layer 6 cells. Therefore, we are certain that the retinogeniculate fibers of layer 6 were unaffected by our magnocellular blocks.

The last control, which turned out to be unnecessary for this experiment, was one in which the possible disruption of the magnocellular fibers by injection into layer 6 was determined. We recorded in layer 1 and activated cells antidromically from cortex. Lidocaine injection into layer 6 had no effect on this antidromic activation.

DISCUSSION

In this study we have found that those cells in the superior colliculus that depend on the cortical downflow for their visual activation, as determined by cooling striate cortex, became unresponsive when the magnocellular LGN was blocked; parvocellular blockage, on the other hand, had no significant consequences. Superficial collicular cells unaffected by disruption of the corticatctal pathway, presumably because of direct retinal activation, were not affected by the LGN blockage either, demonstrating that our LGN injection procedures did not influence the retinotectal pathway. The results of this study suggest, therefore, that the indirect geniculostriate input to the superior colliculus is predominantly a magnocellular, broad-band pathway.

There are two inferences that can be drawn from these results. The first is that the corticatctal cells of area 17 are driven only by the magnocellular geniculate input. In light of anatomical findings, this conclusion is surprising. Using retrograde transport of horseradish peroxidase from the superior colliculus, Lund et al. (20) identified the pyramidal cells of cortical lamina 5B as the source of striate cortical input to area 17. On the basis of earlier Golgi studies (18, 19) the layer 5B pyramidal cells were regarded as receiving information from both magnocellular and parvocellular laminae.

The second inference is that the color-opponent and broad-band pathways remain segregated, in part, in cortical areas beyond area 17. Thus, it is conceivable that corticotectal cells of extrastriate visual areas might also be selectively driven by the broad-band pathway. Indeed, Lund et al., (20) trace a direct projection of striate lamina 4B—which receives predominantly magnocellular input—to superior temporal sulcus, and this extrastriate cortical region projects to deep portions of the superior colliculus (13).

Several hypotheses have been advanced regarding the functional significance of having two distinct pathways in the geniculo-striate system (17, 29). It has been suggested that the X (color-opponent) system is involved in fine pattern analysis in focal vision, while the fast Y (broad-band) system is involved in the analysis of spatial location, thereby playing a central role in directing attention and the eyes to targets in visual space. The fact that the superior colliculus, which is involved in the control of saccadic eye movement in monkeys, receives its direct input from the broad-band system is in consonance with this view. It has also been suggested that in striate cortex of the cat the X system activates predominantly simple cells, while the Y system feeds into complex cells, though the evidence at this stage is fragmentary (10, 15, 23, 25). In conflict with this suggestion, in the monkey we have found some simple cells which are driven by either magnocellular geniculate input, and complex cells which can be driven by either magnocellular or parvocellular input. It is true, however, that the corticatctal cells in striate cortex are complex (5, 24). The resolution of the issues concerning the way the color-opponent and broad-band systems contribute to the organization of striate cortex awaits further study.
ACKNOWLEDGMENTS

The authors are indebted to Denice Couch-Helwig and Cathy McAnsh for their help.

This research was supported by National Institutes of Health Grant 5 R01 EY00676, National Science Foundation Grant BNS 76-82543 A01, National Institute of Neurological and Communicative Disorders and Stroke Grant 5 F32 NS05799, and Alfred P. Sloan Foundation Research Fellowship BR-1895.

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