Dimensions and Properties of End-Zone Inhibitory Areas in Receptive Fields of Hypercomplex Cells in Cat Striate Cortex

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SUMMARY AND CONCLUSIONS

1. Subregions in the receptive fields of hypercomplex cells have been examined by a variety of quantitative methods with particular reference to the dimensions and properties of the end-zone inhibitory areas. These data have made it possible to construct detailed maps of the receptive-field organization of the two types of hypercomplex cell (I and II).

2. The spatial extent of the end-zone inhibitory area is much greater than that responsible for discharge-region excitation. End-zone inhibition is, however, position dependent, the part of the area causing maximal inhibition lying precisely along the line of the most responsive part of the discharge region and just beyond its lateral border. Spatial summation of end-zone inhibition takes place along the line of its optimal stimulus orientation.

3. Some simple and complex cells may have hypercomplex-type length-response curves in the nonpreferred direction of stimulus movement and vice versa for some hypercomplex cells. Whether these response patterns are due to the presence of direction-selective end-zone inhibition or not remains to be determined. While end-zone inhibition may be direction selective, it appears that it is usually nondirectional. Even when discharge region excitation is itself completely direction selective, the end-zone inhibition may be equally effective in both directions. Hence end-zone inhibition appears to be independent of the mechanism responsible for the direction selectivity of the discharge region.

4. End-zone inhibition is stimulus orientation dependent, being maximal when the orientation is the same as the orientation that is optimal for the discharge region. When the stimulus is rotated away from the optimal, the strength of the inhibition progressively declines, falling to zero at 90° to the optimal. This property distinguishes end-zone inhibition from side band inhibition since the latter is not orientation sensitive.

5. There may be considerable, or even total, spatial overlap between discharge-region excitation and end-zone inhibition, the spatial summation required for excitation being much less than that required to produce an inhibitory effect. The onset of inhibition in the length-response curve indicates that the effects of the spatial summation of inhibition now exceeds those of discharge-region excitation.

INTRODUCTION

This is the third of four planned papers devoted to a detailed examination of the properties of hypercomplex cells in the striate cortex of the cat. Our first paper (9) was largely concerned with the problem of cell classification and we gave particular attention to those cell properties by which hypercomplex cells are to be distinguished from other orientation-sensitive cells. The analysis of end-zone inhibition in that paper was based solely on the responses to optimal stimuli moving in the preferred direction. In the paper immediately preceding the present report (11, the second in the series) we studied the properties of hypercomplex cells using a much wider variety of stimuli including sta-
tionary flashing bars as well as nonoptimal orientations and nonpreferred directions of movement. The present paper (the third in the series) completes our analysis of the general properties of end-zone inhibition. We provide a detailed description of the receptive-field organization of the two types of hypercomplex cell (I and II), giving particular attention to the dimensions of the end-zone inhibitory areas in relation to the other subregions in the receptive field. In addition, we examine the orientation dependence and direction selectivity of the inhibition. The present investigation, the variable delay method has since been used extensively in laboratory though not previously used in the present series. We shall refer to the two procedures described in detail in earlier papers from this laboratory though not previously used in the present series. We shall refer to the two procedures as the conditioning method (2, 5) and the variable delay method (5, 10), respectively. Both methods were originally introduced as a way of studying the nondominant eye receptive fields of monocularly discharged striate neurons. The conditioning method has since been used extensively in monocular studies in order to reveal inhibitory subregions in the receptive field. In the following account we will refer to the curve relating the strength of the inhibition at closely spaced positions across the width of the end zone without undue interference from the response variability of the neuron. In the following account we will refer to the curve relating the strength of the inhibition to position across the end zone as a width activity profile. The strength of the inhibition is taken as the amount by which the response from the discharge region is reduced below the mean control level expressed as a percentage of that control level. The control level, indicated by the dashed line in Fig. 1, is given by the amplitude of the response to the fixed stimulus acting on its own.

RESULTS

The presence of inhibitory side bands (2) in the receptive fields of hypercomplex I cells
Disch

Fixed stimulus Variable stimulus

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
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<th>5</th>
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% change in response

FIG. 1. Diagram of the variable delay method used for preparing width activity (inhibitory) profiles from an end zone of a hypercomplex cell. The fixed stimulus moves over the discharge region, while the variable stimulus, starting at different times before or after the fixed stimulus, moves over an end zone. Given this difference in starting time, the variable stimulus crosses different regions of the end zone at the same time as the fixed stimulus moves over the discharge region. The reduction in the response to the fixed stimulus gives the strength of the inhibition at the different parts of the end zone. The profile is given as a function of variable stimulus position by converting differences in starting time to spatial coordinates.

(9) complicates the analysis of the properties of end-zone inhibition. The extent to which these two inhibitory regions—side bands and end zones—are spatially distinct is a matter that is difficult to decide, particularly since the two regions have some inhibitory properties in common. In this respect hypercomplex II cells have a simpler organization in that they lack inhibitory side bands. For this reason we have selected a hypercomplex II cell for our initial description before going on to examine the organization as it is found in hypercomplex I cells. The subsequent general descriptions given in this paper are based on the quantitative analysis of the responses of the same 60 striate neurons that were used in the previous paper (11). We stress once again that the dimensions of the various regions in the receptive field and the general outline of a field are somewhat arbitrary since they depend very much on the stimuli and procedures that were used to determine them (cf. DISCUSSION in previous paper, Ref. 11).

Hypercomplex II cell: receptive-field plot

The receptive fields of hypercomplex II cells have three main areas, a discharge region and two end zones (9), and Fig. 2 illustrates the procedures we applied to such a cell in order to plot these three regions. The properties of this cell were examined in considerable detail and various aspects of the cell's investigation are illustrated in Figs. 2, 6, 7, 8, and 9. The upper half of Fig. 2 concerns the width (A) and the length (B) of the discharge region and the lower half of Fig. 2, the length (C) and the width (D) of the end zones. In the lower portion, the discharge region has been redrawn from the upper section and only one of the two end zones has then been added. All the spatial details in Fig. 2 have been drawn to a common scale as shown on the lower right. In the interests of simplicity the optimal stimulus orientation, which was 60°, has been set to the vertical and the preferred direction of stimulus movement is taken as being from left to right across the receptive field. Doubtless the various receptive field regions have irregular outlines but since, in general, we had knowledge only of their length and width, both in Fig. 2 and in most of the remaining figures in this paper, we have represented these regions as rectangles.

DISCHARGE REGION. The hand methods we
used to define the borders of the discharge region were described in an earlier publication (9) and our concern here is solely with quantitative determinations. For our present purposes we shall regard the discharge region as that area of the receptive field from which firing of the cell can be obtained by a narrow moving bar stimulus.

*Width.* The width of the discharge region is defined by the primary borders ab and dc in Fig. 2A. These borders were given by the width excitatory profile A (average response histogram) in response to an optimally 1.14 x 0.29° light bar (Sx) moving from left to right across the receptive field. The two vertical dashed lines at a and d mark the beginning...
and end of the discharge, the firing outside these bounds being regarded as the maintained discharge of the cell.

Length. Unless we have specifically indicated otherwise, the length of the discharge region has always been given either by a bilateral (9) or two unilateral (11) length-response curves. In the present description, however, the length, defined by the lateral borders ad and bc (Fig. 2B), has been determined by reference to a length excitatory profile B. The reason why we have chosen such a length criterion on this occasion will become clear when we discuss the spatial overlap of the discharge region and inhibitory end zones (see below). Nevertheless unilateral length-response curves were also recorded so that the data are available for the determination of the lateral borders by both methods. A length excitatory profile represents the responsivity of a cell to a short bar stimulus moved across the receptive field but with its line of movement successively shifted laterally to stimulate different parallel strips of the receptive field. In the present instance (Fig. 2B), the data points defining the profile were given by the responses to the movement of a 1.14" x 0.29' light bar (S₉) moved along a series of 10 traverses across the discharge region, each traverse being displaced from its neighbor as indicated by the abscissa scale. The location of the data points along the abscissa correspond to the midpoint of the light bar in each case. As noted previously (7) the excitatory profile obtained in this way approximates a Gaussian curve. The two horizontal dashed lines at d and c mark the lateral borders of the discharge region that have been arbitrarily set to correspond to response levels X and Y on the excitatory profile that are 25% of the peak value, taking the level of the maintained discharge as 0%. The level of the maintained discharge in this and subsequent figures is indicated by a horizontal arrow located at the beginning of the curve and directed toward the ordinate scale. By the two procedures described above we found the discharge region of this cell to be 4.2" wide and 4.4' long. Had we used length-response curves to define the lateral borders, the value we ascribed to the length of the discharge region would have been significantly smaller. The reason for this discrepancy will be considered below.

End-Zone Inhibitory Region. Length. The length of an end-zone inhibitory region is measured along the line of the optimal stimulus orientation. The horizontal dashed lines at h and g in Fig. 2C show how the inner and outer borders, respectively, of one of the end-zone inhibitory regions have been derived from the unilateral length-response curve C to give an end-zone width of 3.2". The inner border (eh), marking the onset of end-zone inhibition, is given by the downturn of the length-response curve while the outer border (fg) corresponds to the beginning of the terminal plateau phase of the curve. It should be noted that the length-response curve locates the inner border of the end zone well inside the lateral border of the discharge region as given by the excitatory profile (see below). Only the end zone with the stronger inhibition (98%) is shown in Fig. 2, but the data for the other end zone are given by the second length-response curve in Fig. 9.

Width. The width (fg) of the lower end-zone inhibitory region in Fig. 2 was 5.9' as determined from the width activity profile D prepared by the variable delay method (see METHODS). For this profile we used the light bar S₉-F (2" x 0.29") as the fixed stimulus and the bar S₉-V (5.5" x 0.29") as the variable stimulus. The length of the fixed stimulus (2") was made approximately equal to the length of the discharge region (1.9") as given by the two unilateral length-response curves in Fig. 9. The fixed and variable stimuli were so located that the site where they abutted one another end to end approximately coincided with the inside border (eh) of the end zone as given by the downturn of the length-response curve C. The length of the variable stimulus was chosen so that it extended beyond the outer border (fg) of the end zone as set by the onset of the plateau phase of the length-response curve. For the activity profile C we tested the strength of the inhibition at 27 positions spaced 0.24" apart across the width of the end zone over a total range of 6.2". The maximum end-zone inhibition, calculated from the length-response curve C (98%), was approximately the same as that given by the variable delay method D (90%). Using similar methods, we found the length of the end zone on the other side to be rather less (2.7") and the inhibition less intense, being 51% on the basis of the uni-
lateral length-response curve in Fig. 9 and 43% using the variable delay method (not illustrated). The vertical dashed line PQ in Fig. 2 shows that the part of the end zone causing maximal inhibition is precisely in line with the most responsive part of the discharge region.

**Hypercomplex I cell: receptive-field plot**

The receptive field of a unimodally responding hypercomplex I cell usually has five main subdivisions: a centrally located discharge region, two inhibitory side bands, and two inhibitory end zones (9). Figure 3 illustrates the procedures we adopted to determine the boundaries of these areas.

**DISCHARGE REGION.** The borders of the discharge region abcd were determined in the same way as for the hypercomplex II cell in Fig. 2. The primary borders were derived from an average response histogram to a narrow light bar moving over a 10° traverse (not illustrated). As pointed out above, we
normally define the lateral borders by reference to length-response curves but, for our present purposes, these were again set by the 25% amplitude levels on a length excitatory profile (not illustrated). By these methods the discharge region in Fig. 3 was 1.3° wide and 2.7° long.

**Inhibitory Side Bands.** Because hypercomplex I cells have little or no spontaneous discharge, we used the conditioning method (see METHODS) to determine the inhibitory side bands as shown at B in Fig. 3. For the test stimulus we used a light bar of optimal length as given by a bilateral length-response curve. The vertical dashed lines extending down to the response histogram B mark the width of the proximal (jk) and distal (no) side bands, respectively. The side band on the proximal side, though only 1° wide, was nevertheless powerful enough to suppress the background maintained firing completely. The side band on the other side, though broader (2°), was rather less powerful. The length of the two side bands was also determined by the conditioning method, retaining the conditioning stimulus as before but setting the orientation of the test stimulus at 90° to the optimal. In its new setting, the test stimulus ($S_C$) was made long enough (3.5°) to cover the full width of the proximal side band as well as half that of the distal one. Portion of the resulting length activity profile is shown at C. End-zone inhibition is orientation sensitive (see below) and so the test stimulus ($S_C$), being at 90° to the optimal, had no effect on the background maintained discharge in profile C until it reached the line timpl, by which time it had passed over the lateral two-thirds of the end zone. Hence the inhibition in profile C can reasonably be ascribed to the side bands rather than the end zone, particularly since it is known that side band inhibition is not orientation sensitive (2). The above observation was confirmed by the preparation of the length activity profile D using the variable delay method.

The horizontal dashed lines at p and o mark the lateral borders of the full extent of the two side bands. Assuming that the two side bands were of equal length, each measured 3.8°. The determination of the separate lengths of each side band would have required a more detailed analysis using a much shorter test stimulus moved over a series of parallel strips along the length of the receptive field. Earlier observations from this laboratory (2) indicate that, for the stimulus at 90° to the optimal, the whole of the area...
TABLE 1. Subregions in hypercomplex cell receptive fields

<table>
<thead>
<tr>
<th></th>
<th>Length of Discharge Region,* deg</th>
<th>End-Zone Inhibition,* %</th>
<th>End-Zone Length, deg</th>
<th>End-Zone Width, deg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypercomplex I</td>
<td>1.4 ± 0.94 (1.6)†</td>
<td>70 ± 19.3</td>
<td>1.9 ± 0.93</td>
<td>5.4 (5 cells)</td>
</tr>
<tr>
<td>24 cells</td>
<td>(0.3–3.6)</td>
<td>(27–100)</td>
<td>(0.3–4.0)</td>
<td>(3.1–6.6)</td>
</tr>
<tr>
<td>Hypercomplex II</td>
<td>1.6 ± 1.02 (1.9)†</td>
<td>67 ± 21.3</td>
<td>3.0 ± 1.12</td>
<td>6.1 (2 cells)</td>
</tr>
<tr>
<td>10 cells</td>
<td>(0.7–3.6)</td>
<td>(39–100)</td>
<td>(1.6–5.5)</td>
<td>(5.9–6.7)</td>
</tr>
</tbody>
</table>

Values are means ± SD. Figures in parentheses are ranges. * From Ref. 9. † Values have been adjusted for the difference between the unilateral and bilateral curves.

Dimensions of receptive-field subregions

Using the variable delay method we measured the width of one of the end zones from each of five hypercomplex I cells (mean, 5.4°) and width of both end zones from each of two hypercomplex II cells (mean, 6.1°). The results from three of the hypercomplex I cells are shown in Fig. 4. For each of the cells, average response histograms (A, C, and E) were prepared in response to the movement of a narrow light bar optimally oriented for the discharge region. Then, for recording the end-zone width activity profiles (B, D, and E), the orientation that was optimal for the discharge region was used both for the fixed and the variable stimuli. In Fig. 4 each activity profile has been placed in accurate register underneath its respective average response histogram. Finally the abscissa scales have been arbitrarily arranged so that, in each case, the 2° mark coincides with the peak of the average response histogram. The important point to note is that, in every case, as indicated by the vertical dashed lines, the site of maximum inhibition is fairly accurately in line with the most responsive part of the discharge region.

The dimensions of the various subregions in hypercomplex cell receptive fields are given in Table 1. The lengths both of the discharge regions and of the end zones have been obtained from bilateral length-response curves (9), the length of the end zones being taken as half the distance from the onset of end-zone inhibition to the beginning of the plateau phase (cf. Fig. 1 in previous paper). Using mean values for the various dimensions, the scale diagrams in Fig. 5 represent the receptive-field organization of cells in the simple and complex families as revealed by an optimally oriented narrow light bar moving in the preferred direction. The inhibitory end zones (dotted) are clearly much larger than their respective discharge regions (crosshatched). The responses to the narrow light bar by the simple and hypercomplex I cells are both considered to be of the unimodal type (9, 12) and data from an earlier publication (2) have been used to add the inhibitory side bands to the diagram of the simple cell receptive field. The side bands of hypercomplex I cells have yet to be studied systematically so that, for the purposes of Fig. 5, we have assumed that they have the same mean dimensions as for simple cells (2). In view of the uncertainty regarding the extent of the overlap between the side bands and the end zones, only a vague indication of this overlap is intended in Fig. 5.
Stimulus orientation dependence of end-zone inhibition: hypercomplex II cell

The scale diagram in Fig. 6A shows how the variable delay method was used to demonstrate the stimulus orientation dependence of end-zone inhibition for the same hypercomplex II cell as in Fig. 2. We prepared a series of seven end-zone activity (inhibitory) profiles (Fig. 6B, a–g) using exactly the same stimulus configuration as for the activity profile D in Fig. 2 with the one additional feature that, for each successive profile, the variable bar stimulus S-V in Fig. 6A was rotated about its midpoint by 15° to a new angular setting. Thus the seven orientation settings (a–g) in Fig. 6A covered a total angular range of 90°. The fixed (S-F) and variable (S-V) stimuli in Fig. 6A are shown at the positions they would occupy if both were at the midpoint of their respective sweeps (corresponding to the center of the discharge region), the preferred direction of movement being from left to right as indicated by the arrow at S-F. For the purposes of diagram A, the full sweep of the variable stimulus has been foreshortened unequally at its two ends so that the sweep from position 1–position 2 at orientation a represents only part of the full sweep, similarly for the sweep from positions 3–4 at orientation b, and so on. The arrows associated with each orientation setting indicate the direction of bar movement. For each profile in B, the strength of the end-zone inhibition was tested at 13 variable stimulus positions (cf. Fig. 1) in steps of 0.48° over a total range of 5.76°, virtually just enough to cover the full width of the end zone. The control level in B (dashed line) was given by the amplitude of the response from the discharge region in the absence of the variable stimulus. Just as in Fig. 2D, the strength of the inhibition was taken as the percentage reduction in the response below the control level.

The series of inhibitory profiles in Fig. 6B clearly demonstrates that end-zone inhibition is stimulus orientation dependent. This dependence is further attested in Fig. 6 by the observation that maximal inhibition was produced when little more than half the length of the variable stimulus covered the end zone, whereas at 90° to the optimal, the variable stimulus produced little, if any, inhibition even though the whole length of the bar was now able to influence the end-zone region.

End-zone inhibitory orientation tuning curve

The series of inhibitory profiles in Fig. 6B provides the data for one limb of an end-zone inhibitory orientation tuning curve as shown at B in Fig. 7. Using the same abscissa and ordinate scales, we have also added to Fig. 7 the (excitatory) orientation tuning curve (A) for the discharge region of the same cell prepared with a bar stimulus of length approximately optimal for that region (2.17°; cf. Fig. 9). The optimal orientation for the discharge region is given by the peak of the excitatory tuning curve and, as with Figs. 2 and 6, this has been arbitrarily set at 0°. The orientations of the variable stimulus given in Fig. 6B are in keeping with the above decision regarding the zero setting. Figure 7 shows that the end-zone inhibition was maximal (96%) when the
FIG. 6. End-zone activity (inhibitory) profiles prepared by the variable delay method for seven different stimulus orientations on the same hypercomplex II cell as for Figs. 2, 7, 8, and 9. The configurations used for the fixed (S-F) and variable (S-V) stimuli are shown at A in relation to the discharge region and end zone. The lower-case letters indicate orientations of the variable stimulus and the numerals, 1 → 2, 3 → 4 . . . , etc. show the corresponding direction of movement at each orientation.

Orientation of the variable stimulus was nearly the same (−10°) as the optimal orientation setting of the stimulus used for the discharge region (0°). When the end-zone stimulus was rotated away from its optimal setting, the inhibition progressively weakened until, at 90° to the optimum setting, there was no inhibition at all. Had the bar been rotated through 90° in the opposite direction, the inhibition would presumably have weakened progressively in the same way. On the assumption of symmetry, the resulting tuning curve's half-width at half-strength would have been about 40°, approximately the same as the corresponding value for the excitatory curve (37°).

Stimulus orientation dependence of end-zone inhibition: hypercomplex I cell

In hypercomplex I cells, interference from inhibitory side bands is clearly a problem in deciding whether end-zone inhibition is orientation dependent or not, particularly since side band inhibition is not sensitive to stimulus orientation (2). However, profile C in Fig. 3 already indicates that end-zone inhibition is probably stimulus orientation dependent. As described above, profile C was used to determine the length of the inhibitory side bands, the orientation of the test stimulus $S_c$ being 90° to the optimal. The important observation in the present context is that the bar $S_c$ had reached the line ilmp without producing any inhibition, whereas when the

FIG. 7. Stimulus orientation tuning curve (A) for the discharge region and inhibitory orientation tuning curve (B) for an end zone from the same hypercomplex II cell as for Figs. 2, 6, 8, and 9. The abscissa and ordinate scales are common to both curves. The optimal stimulus orientation for the discharge region has been arbitrarily set at 0°. Data for curve B come from the end-zone inhibitory profiles in Fig. 6B.
stimulus orientation was optimal, the corresponding unilateral length-response curve showed spatial summation of end-zone inhibition right to the outer border (eh), the inhibition by that time reaching 85%. On this basis, we ascribed the onset of inhibition in profile C to the side bands and not to the end zone.

We confirmed the above conclusion by preparing end-zone activity profiles (Fig. 3) both at the optimal stimulus orientation (A) as well as at 90° to the optimal (D) using the variable delay method. The fixed stimulus (S_A,F) was the same for both profiles A and D. We first carefully positioned the two stimuli, fixed and variable, so that, at its optimal orientation setting, we judged the path of the variable stimulus (S_A-V) to lie outside the lateral borders of the side bands, although subsequent detailed analysis in Fig. 3 showed that there might have been some slight side band involvement. As mentioned above, the resulting activity profile A was used to define the width (eh) of the end zone. Presumably because the path of the variable stimulus lay outside the powerful medial portion of the end zone, the inhibition due to the variable stimulus was now much reduced, being only 37% as opposed to the 85% given by the corresponding length-response curve. Two observations indicate, however, that the inhibition was due to the end zone and not the side bands. The site of maximal inhibition is directly in line with the center of the discharge region and not with either of the side bands, and the shape of the activity profile A gives no indication of any significant inhibitory contribution from either side band.

Next we rotated the variable stimulus through 90°, repositioning it to a site corresponding to S_P-V in Fig. 3. We then tested for inhibition at the variable stimulus positions given by the abscissa in D. The activity profile (D) shows that the response due to the fixed stimulus remained essentially unchanged at variable stimulus positions across the greater part of the end zone, only becoming significantly reduced at positions inside the lateral borders of the side bands as indicated by the dashed line limp. As with the hypercomplex II cell in Fig. 6, the inhibitory effect of the bar S_A-V at the optimal orientation is much greater than when it is at 90° to the optimal, even although, when optimally oriented, only about two-fifths of the bar passed over the inhibitory zone, whereas in the 90° position, the whole of the length could be effective. Thus we can conclude that end-zone inhibition is stimulus orientation dependent in both kinds of hypercomplex cell, having its maximum effect both when the orientation is the same as the optimum setting for the discharge region and when the stimulus is in line with that region. There appears to be a fair degree of overlap between the side bands and the end zones and we confirm earlier observations (2) that side band inhibition is not orientation dependent.

Orientation-dependent inhibition: evidence from other cells

We were able to obtain fairly detailed maps of the receptive-field organization of eight cells along the lines indicated above. For two of the eight cells, both hypercomplex I, the data were insufficiently detailed for a decision to be made, but in the remaining six cells (hypercomplex I, four; hypercomplex II, two) the evidence was clear cut that end-zone inhibition was stimulus orientation dependent, being always maximal when the orientation was the same as the optimal setting for the discharge region. The variable delay method was used for all six cells and two were additionally tested by the conditioning method. Only one cell (Fig. 6) was tested in sufficient detail to prepare an inhibitory tuning curve, but the remaining five cells were tested at two orientations, optimal and 90° to the optimal, and one of these, a hypercomplex I cell, was also tested at 45° to the optimal. At 45° to the optimal, the result was intermediate between the other two orientations.

Direction selectivity of end-zone inhibition

Using only optimally oriented stimuli, the direction selectivity of end-zone inhibition can still present itself in a number of different forms. When a length-response curve is used to uncover the inhibition, the conditioning and testing stimuli are necessarily combined in the one stimulus and both move in the same direction. From a study of length-response curves in the previous paper (11), we concluded that, although the end-zone inhibition in an occasional hypercomplex I cell may be direction selective, in the great majority of cases it is not. The direction
selectivity of end-zone inhibition can, however, be tested in ways that do not depend on a discharge region response in the nonpreferred direction. By separating the conditioning and testing stimuli, with the former passing over the discharge region and the latter over the end zone, it is possible to test whether movement over the end zone in the nonpreferred direction can inhibit firing from the discharge region produced by movement in the preferred direction and, of course, the reverse situation can also be tested when the cell fires in both directions. Here the terms preferred and nonpreferred refer to firing from the discharge region and not to inhibition from the end zone.

Two cells (hypercomplex I and II), tested by the conditioning method, showed clear-cut inhibition when the test stimulus moved over the end zone in both directions (Fig. 8, hypercomplex II). Since the discharge region of one cell (hypercomplex II) was completely direction selective and that of the other cell almost so (94%), this result means that, for these two cells, the inhibition was not direction selective, at least in relation to discharge region firing in the preferred direction. For both cells the above observations were confirmed in other ways. The hypercomplex I cell still fired sufficiently in the nonpreferred direction to show that its length-response curve was of the hypercomplex type in both

FIG. 8. The use of the conditioning method to demonstrate non-direction-selective end-zone inhibition from a hypercomplex II cell whose discharge region was completely direction selective. Same cell as for Figs. 2, 6, 7, and 9. The background discharge (condition only) was produced by repetitive stimulation of the discharge region. The test stimulus in A moved over an end zone both in the direction preferred by the discharge region as well as the nonpreferred direction.
directions, and the variable delay method used on the hypercomplex II cell also indicated that its inhibition was not direction selective. Figures 2 and 8, being from the same cell, also show that the width of the end zone and the magnitude of the inhibition as revealed by the conditioning method are about the same as those given by the variable delay method. Our observations, therefore, indicate that, while end-zone inhibition may be direction selective, it is usually nondirectional. Furthermore, end-zone inhibition appears to be independent of the mechanism responsible for the direction selectivity of the discharge region.

Overlap of discharge region excitation and end-zone inhibition

There may be considerable, or even total, spatial overlap between discharge region excitation and end-zone inhibition, the spatial summation required for excitation being much less than that required to produce an inhibitory effect. We have already drawn attention to this overlap when describing the plotting procedures in Fig. 2, the inner border of the end zone (eh) given by the length-response curve (C) lying well inside the lateral border of the discharge region (bc) derived from the length excitatory profile (B). Figure 9 examines this observation in more detail. The length excitatory profile (A) has been redrawn from Fig. 2 and the two unilateral length-response curves (B) are accurately located in register beneath it, the abscissa for B applying to both A and B. The bar stimuli a, b, and c are drawn to scale, bar c (1.14" x 0.29") being the bar used for the length excitatory profile A (cf. Fig. 2B). The lengths of the bar stimuli a and b are those that gave maximal responses in the respective length-response curves. The vertical dashed lines, marking the onset of end-zone inhibition in the two length-response curves, cut the length excitatory profile at points M and N, respectively.

Analysis of the data in Fig. 9 shows that, for this cell, the overlap is almost complete between discharge region excitation and end-zone inhibition. All parts of the discharge region can show both excitatory and inhibitory effects. Thus bar c on its own at the location indicated in Fig. 9 gives a response (14.7 spikes/response) that is more than half the peak value of the length excitatory profile, but when bar a is added to produce a longer bar (a + c) the response (8.4 spikes/response) is smaller than either a or c on their own. This reduction in response occurs despite the fact that bar a is passing over the most sensitive part of the discharge region.

An overlap of excitation and inhibition is indicated whenever the length of the discharge region as given by a length excitatory profile (e.g., XY in Fig. 2B) is longer than that given by the two unilateral length-response curves (Fig. 9B). An analysis along these lines is, however, complicated by the fact that the lateral spread of the length excitatory profile depends on the length of the bar stimulus that is used. The spread of the profile increases with increasing bar length up to some maximal value, and thereafter the spread decreases as summation of end-zone inhibition begins to take effect. Length excitatory profiles and unilateral length-
 TABLE 2. Overlap of end-zone inhibition and discharge region excitation

<table>
<thead>
<tr>
<th>Cell Type</th>
<th>Cortical Cell Lamina</th>
<th>Length of Discharge Region,° deg (From Length Excitatory Profile)</th>
<th>Length of Discharge Region, deg (From Unilateral Length-Response Curves)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Stimulus length</td>
<td>Profile length</td>
</tr>
<tr>
<td>Hypercomplex I</td>
<td>2–3</td>
<td>0.5</td>
<td>0.82 (1.5)</td>
</tr>
<tr>
<td></td>
<td>2–3</td>
<td>1.0</td>
<td>1.03 (2.1)</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>0.46</td>
<td>0.86 (1.5)</td>
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<td></td>
<td>6</td>
<td>0.52</td>
<td>1.6 (2.9)</td>
</tr>
<tr>
<td>Hypercomplex II</td>
<td>1.14</td>
<td>4.4 (7.9)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>1.0</td>
<td>2.51 (4.5)</td>
</tr>
</tbody>
</table>

Values in parentheses are the estimated full lengths of the excitatory profiles. * Length measured at 25% excitatory profile height.

response curves were prepared from six cells (Table 2) and, on the arbitrary assumption that the length of the discharge region is set by the 25% level of the length excitatory profile, four cells (hypercomplex I, two; hypercomplex II, two) showed an overlap. One of the cells that failed to show an overlap on this basis is illustrated in Fig. 2D and E in the previous paper (11). However, the length of the bar used to prepare the length excitatory profile for this cell was rather long (1.0°) and, had a shorter bar been used, it is possible that even this cell would have shown an overlap. The overlap is naturally much more extensive if the full length of the discharge region is taken into consideration (Table 2, values in parentheses).

DISCUSSION

Receptive-field dimensions

We have already stressed (9) that the dimensions of the various subdivisions of a receptive field are critically dependent on the nature of the stimulus and the plotting procedure that is used. This observation is clearly exemplified by the marked differences that may occur between the dimensions obtained by hand methods and those obtained by quantitative procedures. In principle the various plotting methods fall into two categories: those using a differential measure of receptive-field dimensions and those using an integrative measure. The differential method measures the responsiveness at each point in the receptive field, or along separate parallel strips across the field, and corresponds to what we call an excitatory or activity profile. The hand method we use to plot the receptive field of hypercomplex cells (9) is of this type. The receptive-field organization of hypercomplex cells lends itself to this method both because these cells respond well to very short bars and because the use of long bars brings in the endzone inhibitory mechanism. By contrast, an integrative procedure is better suited to endfree cells, and simple cells in particular prefer long stimuli over spots. The various integrative procedures are based on the preparation of length- (or width-) response curves in one form or another, and they have in common that the extent over which there is spatial summation of responses is a measure of the receptive-field dimension. Most of our quantitative estimates of length use either unilateral or bilateral length-response curves. Another form of length-response curve starts with a short bar outside the receptive field and tests for spatial summation by progressively extending the bar along the receptive field until it covers the whole of its length. We have not used this method quantitatively, but it has been used in other studies (7, 13). The hand method used for plotting the length of the minimum response field (1, 3) is another type of integrative procedure. However, this plotting method is rather different from the other integrative procedures in that the lateral border of the discharge region is given, not by the extent over which there is spatial summation, but rather by the point at which spatial summation from the periphery inward just gives rise to a threshold response.

If comparisons are to be made of the dimensions of the receptive fields of cells in different cortical laminae or in the same lamina under different conditions, the plotting pro-
procedure used for each type of cell should be uniform throughout the study and should subsequently be described in proper detail. Unfortunately, Gilbert's (4) account is lacking in both these respects. He variously estimated the length of the discharge region by two different methods—from length-response curves and from length excitatory profiles. He also estimated the width by two methods—by stationary flashing bars and by calculation from response histograms to a moving bar. In each case the two methods may give quite different results. Even the one method may give different results depending, for example, on the spatial dimensions of the stimulus (e.g., length excitatory profile).

Spatial overlap of discharge region excitation and end-zone inhibition

In describing the higher order hypercomplex cells in area 19, Hubel and Wiesel (8) showed that the spatial extent of the receptive field giving rise to excitation when tested with a short bar may be overlapped by that giving rise to inhibition when the bar is lengthened. Recently the same phenomenon has been reported in area 17 (4, 7, 13, 16) and used by Gilbert (4) as the basis for differentiating standard complex cells from special complex cells. Sillito (16) described the phenomenon in some detail, observing that a clear subdivision of the receptive field into inhibitory and excitatory zones appeared to be the exception rather than the rule for the superficial-layer hypercomplex cells. He regarded his observations as consistent with the presence of an excitatory region showing a graded change in responsiveness, decreasing from the center to the edge of the field, that is overlapped by inhibitory zones showing a similar variation in responsiveness over the field and possibly also extending from the center outward. The possibility of a complete overlap was suggested to him by the observation that a long bar only produced its maximal inhibitory effect when the path of the stimulus included the excitatory center of the receptive field. In addition, long slits produced a strong suppression of the hypercomplex cell resting discharge when it was artificially maintained by iontophoretically applied DL-homocysteic acid, indicating that the motion of the bar was eliciting a postsynaptic inhibitory input to the cell.

If the onset of inhibition in a length-response curve is used to determine the length of the discharge region then, by definition, the discharge region and the end zones must be nonoverlapping. Since it leaves open the possibility of demonstrating an overlap between the excitatory and inhibitory areas, we have used the length excitatory profile to define the extent of the discharge region. In this way we have confirmed the presence of an overlap in the receptive fields of area 17 hypercomplex cells. Henry, Goodwin, and Bishop (7) have also provided a convincing demonstration of the overlap in hypercomplex I cells by starting a length-response curve at one end of a length excitatory profile and extending the bar progressively along the length of the discharge region (cf. their Fig. 3C). They showed that end-zone inhibition can become manifest when the end of the bar has progressed no further than the central part of the discharge region corresponding to the peak of the length excitatory profile. Hence, overlap is not a property reserved for the higher order cells of area 19 as was originally thought by Hubel and Wiesel (8). In addition, it is clear that the overlap commonly occurs in both types of hypercomplex cell (I and II). Furthermore, the onset of inhibition in a length-response curve is not to be regarded as indicating a border between the discharge region and the end zone, but rather the site at which spatial summation for inhibition becomes greater than that for excitation.

Position and orientation sensitivity

We have confirmed Hubel and Wiesel's (8) original observations that end-zone inhibition is both stimulus position and stimulus orientation sensitive. Their hand method for testing position sensitivity is the same in principle as our variable delay method. It involved stimulating the discharge region and an end zone simultaneously with two edges separated by a step of varying height. The position sensitivity of the end zone was tested by varying the height of the step. The position sensitivity of the end zone was tested by varying the height of the step. Hubel and Wiesel's (8) evidence for orientation sensitivity was, however, not conclusive since, in their tests, they varied stimulus position and stimulus orientation at the same time. A non-orientation-sensitive end zone with higher position sensitivity in its central part might be sufficient to account for their result since their method of varying orientation also led to a more peripheral part of the end zone.
FIG. 10. Two models for the receptive-field organization of hypercomplex cells, one (A) without and one (B) with overlap of discharge-region excitation (E) and end-zone inhibition (I). The two length-response curves (L-R) represent the interaction of hypothetical length excitatory (Ea) and length inhibitory (Ia) curves. Eb, length excitatory profile; Ib, length inhibitory profile.

being stimulated. Furthermore, for the edge stimulating the end zone, the relationship of the axis of movement to the orientation of the edge changed as the orientation varied. In our tests, position and orientation were varied independently and the axis of movement of the end zone stimulating bar was always at right angles to its orientation. Nevertheless, in every case we found the end zone to be both position and orientation sensitive.

Though opposite in their effects on the cell, the properties of the discharge region and the end zones are always closely matched. Not only do both regions have the same optimal stimulus orientation, but also both appear to have much the same orientation specificity. Furthermore the most sensitive parts of the two regions line up with one another in the direction of the optimal orientation. The two regions must also match in another way. We have observed that the end zones of a given hypercomplex cell have the same spatial extent and degree of inhibition whether the stimulus is stationary or moving. This is remarkable since cortical neurons vary considerably in the extent to which they respond to stationary and to moving stimuli. Nevertheless, the relative responses to the two kinds of stimuli by the two regions in a given cell would have to be approximately equal for the degree of end-zone inhibition to be about the same under the two conditions of stimulation.

Models for end-zone inhibition

Hubel and Wiesel (8) proposed two models for the organization of the receptive fields of lower order hypercomplex cells, the inhibitory afferents responsible for the end-zone inhibition being from complex cells in both cases. In one model, the inhibitory cells or sets of cells have their receptive fields abutting the discharge region but not overlapping it, whereas in the second model, the inhibitory input is supplied by a single cell with a large receptive field covering not only the discharge region but also regions beyond its lateral borders. The latter model requires that a bar restricted to the discharge region be too short to affect the inhibitory cell. Hypercomplex cells of higher order have a dual preferred stimulus orientation, one at right angles to the other. Hubel and Wiesel (8) proposed that the simplest mechanism for these cells would involve convergence on the cell of two sets of afferent hypercomplex cells having their preferred orientations 90° apart. The two models for lower order cells are still valid, though the second seems much the more likely in view of the extensive, or even complete, overlap of excitatory and inhibitory elements that obtains in lower order cells. Figure 10 shows updated versions of the two Hubel and Wiesel models drawn in terms of length excitatory and length inhibitory profiles and unilateral length-response curves.

Schiller, Finlay, and Volman (14) have suggested that the end-zone inhibition ap-
pearing at cortical level may simply be a reflection of inhibition at the level of the lateral geniculate nucleus, the majority of geniculate neurons being strongly inhibited by their surrounds when long bars or edges are used. Another possibility they propose (15) is that, in end-stopped cells, the inhibitory side bands extend around the ends of the discharge region to form the end-zone inhibitory areas. There are strong arguments against both possibilities. If the inhibition is generated at geniculate level, why are there end-free cells in the cortex? As Schiller et al. (14) point out, their existence would require that the significant transformation at the cortical level would be the elimination of end-stopping rather than the opposite. Furthermore, we have found that end-zone inhibition is always orientation sensitive and may be direction selective, properties quite unlike the surround inhibition at geniculate level. Again, as we have mentioned above, Sillito (16) has provided evidence that end stopping is based on a postsynaptic inhibitory input to the hypercomplex cell. The suggestion that side band inhibition and end-zone inhibition have a common basis also has its problems. Side

band inhibition is neither orientation sensitive or direction selective (2). Furthermore, simple cells have inhibitory side bands but no end-zone inhibition, whereas hypercomplex II cells have end-zone inhibition but no side bands.

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