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"Charting the Visual Cortex"

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10 Charting the Visual Cortex

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1. Introduction

Progress in understanding the visual cortex was punctuated, in the past decades, by a series of discoveries from Hubel and Wiesel's laboratory, beautifully narrated in Hubel's Nobel lecture (1982). This and Hubel and Wiesel's Ferrier lecture (1977a) provide a good starting point for the analysis which I have in mind. I will not repeat their statements, but rephrase them in my own terms, in order to show the difference in point of view which is the main justification for a separate discussion of quantitative aspects of the visual cortex. Mainly, my emphasis as well as that of some other modelists is on the formulation, at all costs, of an efficient neuronal model compatible with general ideas about the cortex, and apt to enrich them. By an efficient model I mean one which could be immediately translated into a computer program in order to make a computer perform exactly those operations which we ascribe to that piece of cortex. I am emphasizing *at all costs,* for that is probably the main difference, the costs in the case of the models including unwarranted assumptions and possibly even exclusion of facts which are generally considered to be well established. Such bending of the official picture is justified if no other way has yet been found of arriving at an efficient model, the efficiency and hence most fundamental agreement with the existing mechanism (which is also, obviously, efficient) constituting the supreme criterion for the validity of any model.

The facts which I will consider refer almost exclusively to the monkey. As the aim is quantitative comparison of a variety of data, it is safer to stick to one species first.

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2. Receptive Fields: General Remarks

Similar to what had already been established for retinal ganglion cells by Barlow *et al.* (1957), visual cortical neurons act as differential analyzers of the visual input in the following sense. Typically, diffuse illumination at any intensity produces little or no output of these elements, whereas local maxima or minima will. Only, in the case of most cortical neurons, the most effective input configuration is anisotropic, not circularly symmetric as in the case of retinal ganglion cells. This anisotropy is sometimes apparent in a static analysis, when an elongated region of maximum or minimum illumination is much more efficient in activating a certain neuron if it is presented in a certain place at a certain orientation in the visual field than at any other place or orientation. Sometimes, the anisotropy is more readily seen when such elongated maxima or minima move through the visual field, the response again depending on orientation. Most of the time, the effective input configuration is defined only for a limited region of the visual field, the so-called receptive field, although influences of the visual input outside this region are not systematically investigated in the common type of experiment.

2.1. Balance of Excitation and Inhibition

The lack of response to uniform illumination is a remarkable property. One obvious consequence is good response to contours irrespective of the level of illumination. This apparently carries great selective advantage as long as animals move among objects with well-defined boundaries, i.e., solid and liquid bodies. But we are not too impressed by this property in the context of a theory of the cortex, as cortical neurons apparently inherit it from neurons upstream, i.e., in the retina and geniculate body. Also, we notice, but do not particularly emphasize, the fact that some cortical neurons are detectors for maxima of illumination and others for minima, as this again is probably simply inherited from previous stages of elaboration of the visual input. The retinal elements described by Barlow *et al.* come in two varieties, one a detector of black dots on white and the other vice versa.

2.2. Rules Governing the Layout of Excitatory and Inhibitory Regions

In some cortical neurons, the lack of response to diffuse illumination is clearly related to the structure of the most effective input configuration. These are the cases in which the receptive field is composed of one or more regions where light enhances the spike activity of the neuron, and one or more regions in which light inhibits the neuron. Hence, if these influences are separate and independent and if their effects are balanced, illumination of the entire receptive field has no effect on the neuron. Now, from the published evidence it is apparent that the configuration of such inhibitory and excitatory subfields of receptive fields follows a certain pattern. To my knowledge, no checkerboard configurations have been described, no polka dot patterns, and no strongly curved boundaries between excitatory and inhibitory subfields. It is as if most receptive fields were cut out of a basic striped pattern with alternate stripes of excitation and inhibition (Fig. 1). Depending on the relative position of the field boundary, we may get fields with an excitatory stripe flanked by two inhibitory ones, or an inhibitory stripe flanked by two excitatory stripes, or just an excitatory and an inhibitory one side by side. Receptive fields do not seem to span much more than three or four of the basic stripes.

We cannot decide yet how seriously we want to take this idea of a basic striation underlying the structure of receptive fields in the visual cortex. I will provide a hypothetical justification later on. For the time being, we have to be careful. The underlying periodic structure would be interesting if it reflected some basic periodicity of the architecture of the cortex. But this cannot be deduced from the plots of receptive fields as they are usually presented, namely, in the coordinate frame of a screen situated in front of the experimental animal. The transformation of the coordinates of the visual field into those of the cortex

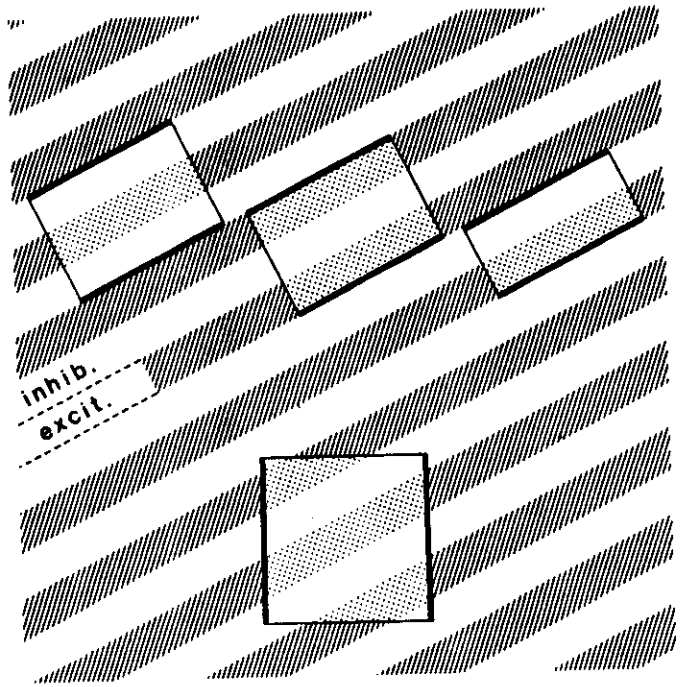

Figure 1. A grossly simplified diagram illustrating how receptive fields with inhibitory and excitatory subregions may be related to an underlying periodic pattern of parallel inhibitory and excitatory strips (above). When the preferred orientation (heavily outlined sides of the rectangles) is not parallel to the pattern (below), the excitatory and inhibitory subfields may not be so easily detected. This diagram is related to the hypothesis made explicit in Figs. 8–13.

implies knowledge of the optics of the eye and of the distortions in the nervous pathways. These are of various kinds and are very considerable. In particular, the scale of the projection varies enormously as a function of position in the visual field. For most plots of receptive fields, the position is not given with sufficient accuracy to permit reliable reconstruction of the corresponding region of the cortex. In a rough way, however, the size of the receptive fields is proportionate to the scale (the so-called inverse magnification factor) of the projection, so that both the size of the fields and the size of their excitatory and inhibitory subregions remain fairly constant over the whole extent of the cortex when they are expressed in cortical coordinates. If there is an underlying striation of the cortex, it may be uniform throughout.

2.3. "Simple" and "Complex" Characteristics

The idea of a periodic organization of the cortex being responsible for the structure of receptive fields, with their alternating excitatory and inhibitory subregions, would not have presented itself if only one class of cortical neurons had been considered, the so-called complex cells of Hubel and Wiesel. They are said to be homogeneous throughout the extent of their receptive fields, with no special subregions in which a light stimulus, punctiform or elongated, would produce special effects. They are, however, sensitive to particular orientations of lines in the visual field as are the others, the "simple" cells, and are particularly receptive for lines moving through the field either in one or the other direction or in both directions.

It is interesting that not only in the case of the striped "simple" cell field, where the explanation is obvious, but also in that of the homogeneous "complex" cell field, the optimal stimulus is a line much narrower than the receptive field itself. One gains the impression that somewhere between the input fibers to the cortex and the cortical neurons there is a level of signal transformation which is common to all types of cortical neurons, "simple," "complex," and "hypercomplex."

The "hypercomplex" characteristic of some cortical neurons is related to the main property of "simple" cells, namely, a nonhomogeneity of the response in different portions of the receptive field. Only in the case of simple cells are the inhibitory subfields elongated and parallel to the preferred stimulus orientation, whereas in the hypercomplex cells they are located in regions which continue the long axis of the optimal stimulus. This makes them respond to the input configuration "end of a line."

Any attempt at reducing simple, complex, and hypercomplex cells to just three geometrical variations of a common principle (I believe that this is possible) will have to consider an interesting distinction which was provided by Hammond and MacKay (1975), Orban (1975), Hoffmann and von Seelen (1978), and Burr *et al.* (1981). Simple cells which respond vigorously to a moving pattern of lines hardly respond when a pattern of dots is used instead. Complex cells, on the other hand, respond about as well to dots as to lines. The idea which this suggests is that in the simple cells the underlying elementary mechanisms are somehow aligned, whereas in complex cells they are not.

3. The Basic Layout of Elements in Area 17 of the Macaque

As soon as we try to translate the preceding sketch of "feature detectors" as seen by physiologists into a neuronal model compatible with the known anatomy, we run into unexpected difficulties. These are mostly due to the unquantitative or scarcely quantitative nature of some of the facts from either discipline, as well as to some contradictions in the data when they are expressed in quantitative terms. In trying to solve these difficulties, the first step is to establish a map of the visual cortex in which the relative sizes and positions of the various elements become apparent (Braitenberg, 1984).

For the surface of the monkey striate cortex, we have the old, unchallenged figure of 1320 mm^2 on either side (Daniel and Whitteridge, 1961).

The number of geniculocortical input fibers is not known with precision. We have, however, counts of fibers in the monkey optic tracts. The figure given by Bruesch and Arey (1942) is 1,210,000 in each tract. What is not known is whether the geniculate relay is divergent or convergent or whether it leaves the number of fibers unchanged. Quite arbitrarily, we set a limit of a factor 3 up or down, and assume that the visual cortex on each side receives between 400,000 and 3,600,000 "specific afferent" fibers. This gives us a rough estimate of the spacing of geniculocortical afferents, of the raster of input points, the pixels as they are called in the machine analog.

Assuming an arrangement such that neighbors form equilateral triangles (the closest packing of pennies on a plane, familiar from honeycombs and insect eyes), the 400,000 to 3,600,000 input fibers distributed uniformly over the 1320 mm^2 of the striate cortex turn out to be spaced at distances of 62 μm for the lower number and 21 μm for the higher one.

There is another, more indirect, way of determining the spacing of input points in the visual cortex from the optical resolution in perception. The acuity of visual perception can be tested in various ways, and the number of picture elements which are elaborated by the brain can be derived from this. One method is two-point resolution: the smallest angular distance at which two stars can be seen as separate can be taken as twice the distance between neighboring picture elements, for there must be an unexcited element between two excited ones in order to distinguish two stars from one. Another method is to determine the highest (space) frequency of a periodic pattern which can be distinguished from uniform gray: again the spacing of picture elements should be one-half that of the wavelength of the pattern according to a well-known theorem of information theory. The angle of resolution determined by these methods roughly corresponds to the angle subtended by two neighboring cones of the central portion of the retina, and when in some ingenious perceptual experiments the perception of small displacements appears to be more accurate than that, the phenomenon is rightly called hyperacuity (von Volkmann, 1865; Westheimer, 1979). But to what distance on the plane of the visual cortex does the angle of resolution correspond? The answer is straightforward when the scale of the representation of the visual cortex is known. Unfortunately, the scale (as I prefer to call it, using a term from cartography), or inverse magnification factor M^{-1} [as it is usually called, the magnification factor (Daniel and Whitteridge, 1961), being defined as the ratio of a stretch of cortex in millimeters to the corresponding angle in

Table I. Density and Number of Afferents to Area 17, Derived from Visual Resolution and Cortical Magnification Factor[a]

Foveal inverse magnification		Minimal angle of resolution: δ = 0.1' ["Hyperacuity" (Westheimer and McKee, 1977; Wülfing, 1892)]	δ = 0.67' (Grether, 1941)	δ = 1' (Folklore)	δ = 1.57' [Dow *et al.*, 1981 (from max. space frequency 19/deg)]
M^{-1} = 10'/mm	(Daniel and Whitteridge, 1961)	d = 10 μm N = 15,200,000	d = 67 μm N = 339,000	d = 100 μm N = 152,000	d = 157 μm N = 61,000
M^{-1} = 6.54'/mm	(Hubel and Freeman, 1977)	d = 15.3 μm N = 6,500,000	d = 102 μm N = 146,000	d = 153 μm N = 65,000	d = 240 μm N = 26,000
M^{-1} = 5'/mm	(Guld and Bertulis, 1976)	d = 20 μm N = 3,800,000	d = 134 μm N = 84,000	d = 200 μm N = 38,000	d = 314 μm N = 15,000
M^{-1} = 2.4'/mm	(Dow *et al.*, 1981)	d = 42 μm N = 800,000	d = 279 μm N = 19,000	d = 417 μm N = 8000	d = 654 μm N = 3500
M^{-1} = 2'/mm	(Talbot and Marshall, 1941)	d = 50 μm N = 600,000	d = 335 μm N = 13,000	d = 500 μm N = 6000	d = 785 μm N = 2500

[a] Surface area of monkey striate cortex: 1320 mm² (Daniel and Whitteridge, 1961). Number of fibers in the optic tract: 1,210,000 (Bruesch and Arey, 1942).

the visual field in degrees], varies in different portions of the visual field and is least well known in the region for which the angular resolution is best known, namely, in the fovea. There, the data are obtained by extrapolation from measurements made in parafoveal regions, the closest to the center of the field being the measurements of Dow *et al.* (1981). The resulting uncertainty is summarized in Table I.

In Table I, five different values for the scale or inverse magnification factor M^{-1} are combined with four different but all equally creditable values for the foveal resolution, δ. All data refer to the monkey. Each pair of values results in a value for separation of input fibers in the cortex, d, and in the corresponding number N of input fibers under the assumption that they are uniformly distributed over the 13.2 cm² of visual cortex.

The number of possibilities shown on the matrix can be drastically reduced by eliminating those entries in which N is either too large or too small. Remember that we had set ourselves the limits for the number of fibers in the optic radiation between three times less and three times more than the fibers in the optic tract. This eliminates everything on the right half of Table I, where the values for N are far too low. Apparently, no matter what magnification factor is assumed, the density of input fibers in the cortex affords a representation of the visual environment with a grain much finer than the 1 min of arc which is often assumed.

On the left half of Table I, we find some values (underlined) which are in reasonable agreement with anatomy. The entries for N which hover around 1 million, the value which would seem to be most acceptable, surprisingly are all in the hyperacuity column. Because of the potential importance of this statement, I will rephrase it in other terms: If the values for magnification factor provided by the most recent measurements of Dow *et al.* (1981) are correct, then 1 million input fibers spread evenly over the visual cortex provide a resolution of the visual environment (in the fovea) which correspond to what is usually called "hyperacuity."

This would indicate that no cortical mechanism of interpolation must be assumed to explain the astonishing accurancy in the alignment of two marks on a vernier, or in the differentiation of the sizes of very small figures, or in the perception of very small displacements. The raster of cortical input fibers will do. But by assuming this, we have only shifted the problem to another level. Ultimately, we must face the question of why the cortical input has a finer grain than the arrangement of cones in the retina (which just corresponds to ordinary acuity, not to hyperacuity). It seems unavoidable to postulate a mechanism of interpolation, somewhere between the retina and the cortex. Could it be the task of the lateral geniculate body, for the existence of which up to now no very convincing explanation has been advanced, to make hyperacuity out of acuity?

There is of course an alternative interpretation of the data in Table I: the values for the foveal magnification factor of Dow *et al.* (1981), Talbot and Marshall (1941) may be wrong. They may be due to an illicit extrapolation of a function describing magnification in the rest of the visual field into a special region which may not continue the general law. But the alternative is not more appealing. If we want the input fibers to come in at such distances as to represent roughly the input from individual cones of the retina, even if we stick to the

foveal magnification factor of Daniel and Whitteridge (1961) or Hubel and Freeman (1977) (Table I), the total number of input fibers turns out to be much too low, and we would have to assign to the lateral geniculate nucleus the even more exotic role of a compression of the input from the optic tracts onto a bundle of fibers by an order of magnitude less numerous.

The entire question will have to be solved by anatomical techniques. It is not beyond technical feasibility to stain the geniculocortical fibers in order to determine their spacing in the cortex. It will then be seen whether the basic assumption (Hubel and Wiesel, 1974b) underlying my preceding speculation is correct, namely, the uniformity of the weave of the entire visual cortex independently of the area of the retina represented there.

Because of the questions which Table I leaves open, we prefer to draw not one, but two alternative maps of the layout of input fibers on the cortical surface in area 17 (Fig. 2). In Fig. 2a the spacing of inputs (dots) is based on the pair of values $\delta = 0.1'$ and $M^{-1} = 2.4'/\text{mm}$, resulting in an average separation of neighboring input points of 42 μm (see Table I). On the other hand, in Fig. 2b the average separation of $d = 67$ μm is obtained from the pair of values $\delta = 0.67'$ and $M^{-1} = 10'/\text{mm}$. Thus, Fig. 2a is compatible with the assumption that the grain of input points is sufficiently fine to explain "hyperacuity," while in Fig. 2b the raster of input points corresponds to the sampling of visual space which is apparent in ordinary two-point resolution experiments.

4. Ocular Dominance Columns

It will be noticed that in both Fig. 2a and b the points are not placed exactly in the vertices of a hexagonal lattice. The reason for the distortion is connected with the well-known "ocular dominance" columns (Hubel and Wiesel, 1972). This unfortunate term ("columns" suggests a different shape) denotes a pattern of elongated regions of the visual cortex, each about 0.4 mm wide, in which input from one eye alternates with input from the other eye. The vertical lines in Fig. 2a and b are the borders between neighboring ocular dominance columns or stripes separating input from one and the other eye. The pictures from both eyes, different only in their stereoscopic discrepancies, are not superimposed but are sliced into long thin strips which are laid out on the cortex, always one strip belonging to one eye side by side with the corresponding strip belonging to the other eye. To be precise, it seems that the cuts in one picture are halfway between the cuts in the other picture, so that each strip has overlapping information with the neighboring strips on either side, belonging to the other eye (Hubel *et al.*, 1974; Blasdel and Lund, 1982).

The separate projection of the pictures from both eyes onto the plane of the cortex must result in a distortion of the projection in one of two ways, or possibly in a combination of the two (Hubel *et al.*, 1974): (1) if the projection remains metrically correct on a small scale, i.e., if neighboring picture elements keep their correct distances in every direction, the overall picture is enlarged by a factor of two in the direction perpendicular to the stripes compared to the

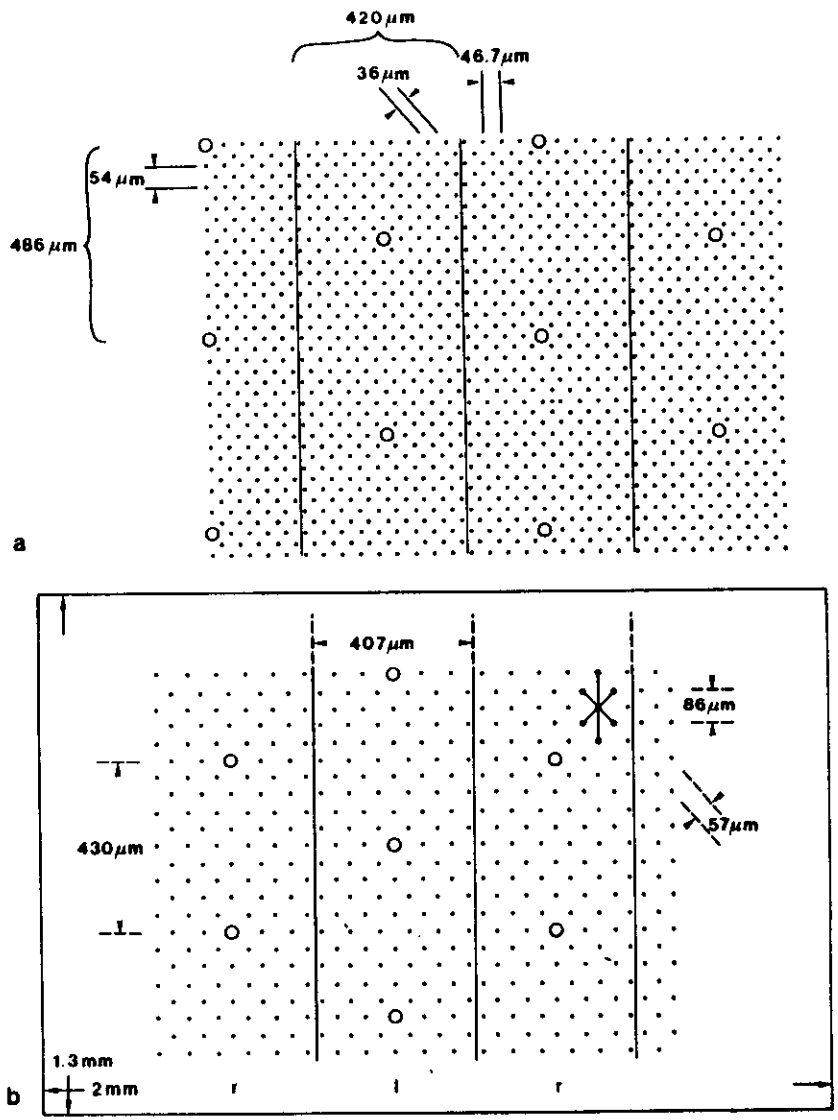

Figure 2. Alternative charts displaying input points, ocular dominance stripes, and cytochrome oxidase blobs, obtained by assuming (a) average separation of input fibers $d = 42$ μm, corresponding to a foveal resolution $\delta = 0.1'$ with the scale $M^{-1} = 2.4'/\text{mm}$; (b) $d = 67$ μm corresponding to $\delta = 0.67'$ and $M^{-1} = 10'/\text{mm}$ (see Table I). The different distances of the input points in different directions of the array result from a compression of the ocular dominance stripes by a factor 2 (see Fig. 3). Due to this, the pattern formed by one point and its six equidistant neighbors goes over into the elongated pattern shown in the upper right corner of (b). The frame around (b) shows the size of an average "receptive field" drawn to scale.

direction parallel to the stripes, because of the doubling of each strip in the cortical representation (Fig. 3, I). This is the case of microscopic isometry combined with macroscopic anisometry. If, on the other hand (Fig. 3, II), we want the overall picture to be undistorted, we must assume a compression of indiviudal strips to half their original width before they are recomposed into a common picture: macroscopic isometry at the expense of microscopic anisometry (the square in the lower left corner turns into a rectangle).

The maps of Fig. 2 are drawn under the assumption that in the visual system of the macaque, alternative II is valid. The pattern of input points is derived from an assumed originally hexagonal arrangement (like that, roughly, of the cones in the fovea) by a compression in the direction perpendicular to the ocular dominance strips. The reasons for this assumption come from two sources. First, Daniel and Whitteridge (1961) in their careful measurements of magnification factor, which are macroscopic measurements if we read their methods correctly, explicitly approach the problem of isometry and find it is present. Second, in psychophysical experiments on the perception of periodic striped patterns (Rovamo *et al.*, 1982) or on the threshold of detection of curvature (Fahle and Braitenberg, 1983), a dependence on position and orientation in the visual field becomes apparent, which admirably matches the arrangement of ocular dominance columns in the monkey when they are projected out into the visual field (Hubel and Freeman, 1977). These experiments, especially the ones on the detection of curvature, seem to indicate that hyperacuity reaches a higher degree of resolution in the direction along the ocular dominance strips than in the direction perpendicular to them. This may be explained on the basis of the idea that hyperacuity is due to intracortical computation of interpolated values between input points: the further they are apart, the greater is the amount of cortical tissue to do the computation and the higher the accuracy of interpolation. Hence, with microscopic anisometry as in Fig. 3 (II), hyperacuity depends on ocular dominance columns.

However, since these maps were first drawn, first-hand evidence on the metrics of the projection of visual space onto the visual cortex of monkeys was provided by Tootell *et al.* (1982). Their [^{14}C]-2-deoxy-D-glucose photographs of geometric patterns in visual space on the monkey visual cortex among other things demonstrate a macroscopic anisometry related to ocular dominance columns (see Crawford, this volume). The anisometry amounts to less than the ratio 2 : 1 postulated in Fig. 3 (I). Apparently, the cost of the doubling of visual space by ocular dominance columns is carried jointly by a micro- and a macroscopic distortion. This makes it still possible for the uneven spacing of input points to make itself felt in psychophysical experiments on hyperacuity.

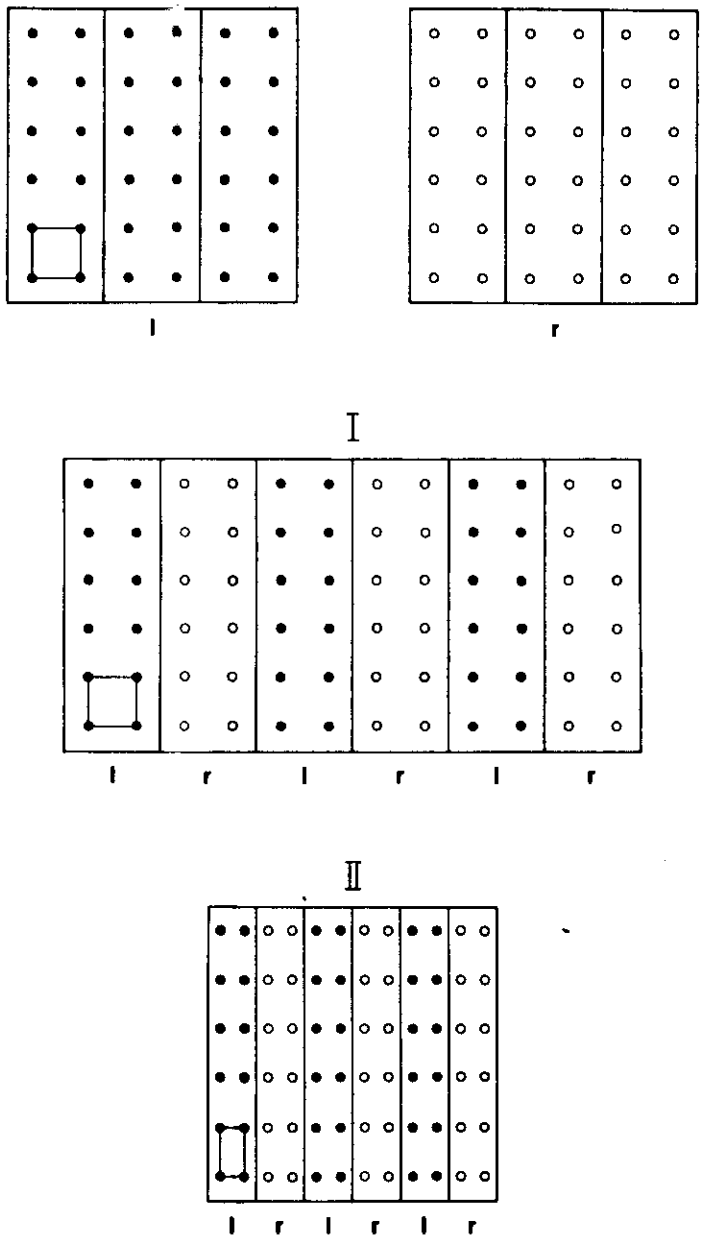

Figure 3. A square region of the visual field, seen by the left and right eye (l, r). The cortex receives a picture of the square composed of alternate strips from both eyes. This implies some distortion, either global (I)—the elementary array is undisturbed at the microscopic level, but macroscopically the square turns into a rectangle—or local (II)—the square is represented macroscopically in its correct proportions, at the expense of a microscopic anisometry, as the horizontal distances of neighboring points are reduced to one-half of their vertical distances.

5. Structure of the Cerebral Cortex

A detailed account of visual cortex neuropil would be doubly redundant here, because of contributions of other authors in this series (see Valverde, this volume), and because only a very small part of the available descriptive neuroanatomy can be utilized in functional models of vision. I will limit myself to a few general remarks.

Uncertainty about the level of description: It is often held that the great variety in the shape of individual cortical neurons makes it practically impossible to offer a description which could be turned into a manageable mathematical model. This is indeed true if what the model maker asks for is a complete catalog of all the 5000 to 10,000 synapses of every single neuron in the cortex. It is possible, however, that the genetic processes which made the tissue were subject to restrictions in information capacity quite similar in nature and extent to those of the neuroanatomist who analyzes the tissue. If it could be decided up to what level of specification a rigid plan was prevailing, and what morphological details were left to chance, it might be possible to use the limited channel capacity of neuroanatomy for a meaningful description of cortical structure. A glance at "cortical architectonics," the variation of neuron size, shape, number, and layering which correlates with areal functional specialization, suggests the following. Within a certain area, in any specified layer of the cortex the neurons of a certain type have fairly uniform characterisitics if only the outlines of their dendritic and axonal territories and the density of dendrites and axons in these territories are considered. The detailed pattern of ramification varies a great deal from neuron to neuron and may therefore be assumed to be beyond genetic specification. On the other hand, territories and densities themselves vary from area to area even in neurons which belong to the same (e.g., pyramidal) type, indicating the functional importance of these magnitudes. Thus, we feel justified in describing neurons as clouds of postsynaptic sites (on the dendrites) connected to clouds of presynaptic sites (on the axon) neglecting the detailed geometry of the ramifications and assuming the distribution of synapses within each cloud to be random and sufficiently well described by the shape of the cloud and by the density of synaptic sites in it. Synaptic connections between neurons can then be calculated by the rules of the calculus of probability under the assumption that no specific affinities between various types of neurons exist. This approach essentially goes back to Sholl (1956) and was exemplified in some work on the mouse cortex (Braitenberg, 1978a,b, 1981) where numbers are actually derived from the relevant densities of pre- and postsynaptic sites. The assumption of a purely statistical nature of the interneuronal connections is particularly convincing in the case of the connections between pyramidal cells, less so for some of the axons of stellate cells which seem to have special affinities to certain parts of other neurons ("baskets" surrounding pyramidal cell bodies, etc.).

The model of orientation columns in the visual cortex which I will present is specified in terms of such statistical neuroanatomy. If the model proves successful, it may actually help spread the feeling that a too detailed analysis of fiber patterns as if they were electronic circuits, misses the point of neural connectivity.

The other piece of neuroanatomy which we need for the model is the distinction between two classes of neurons, pyramidal cells and inhibitory stellate cells. The taxonomy of cortical neurons which was inaugurated by Ramón y Cajal's (1911) account had seemed to lead over the years to an ever-increasing catalog when suddenly a very strong principle of classification emerged from electron microscopic studies (among others: Colonnier, 1968; LeVay, 1973; Parnavelas *et al.*, 1977; Peters and Fairén, 1978; Peters and Proskauer, 1980; Somogyi and Cowey, 1981; Winfield *et al.*, 1981; see chapters in Volume 1) cutting across various previous distinctions. There are two prototypes of cortical neurons, each characterized by a set of properties which have a very strong correlation among each other, in the sense that when a neuron has one or two of these properties, it is likely to have the others as well (see chapters in Volume 1). These properties are for the first neuron type:

1. Most of the synapses (a few thousand) for which the neuron is postsynaptic are excitatory and are localized on dendritic spines.
2. Synapses on the soma are inhibitory.
3. Synapses formed by the axon of the cell are excitatory.
4. The main axon is directed toward the white substance and almost always enters it.
5. Loose, fairly straight axon collaterals.

(here we identify excitatory synapses with the electron microscopic characteristic of Gray type I synapses, and inhibitory synapses with type II.)

For the other neuron type the characteristics are:

1. No spines (or much less than 1 spine per micrometer of dendrite).
2. Mixed excitatory and inhibitory afferent synapses on the soma.
3. Synapses formed by the axon of the cell are inhibitory.
4. Dense axonal ramification overlapping the dendritic tree.

Because of the strong coherence of each of these sets of attributes, we tend to give less weight to some other chracterisitics which have sometimes been used as defining properties, such as the presence of an apical dendrite. Spiny stellate cells, which are said to lack apical dendrites, have all of the attributes which characterize our first type and none of those of the second type (see Lund, Volume 1 of this treatise). If we call the first type pyramidal cells, we are almost, but not quite, in agreement with the current usage.

I am not claiming that the two types characterized by such well-defined properties cover the entire variety of cortical neurons, although my guess is that the neurons of the two types together represent about 90% of the population. There are some other neurons, most of which I consider to belong to the traditional class of Martinotti cells, in which the synaptology is still quite obscure. They are certainly "nonpyramidal" according to various criteria, nor do they seem to belong to the other type. I shall not make use of this type in my model.

We can now mentally (not graphically) complete the picture of the visual cortex as represented in Fig. 2. The input fibers meet with a population of

intrinsic cortical neurons whose average separation (computed as the inverse of the cubic root of their density in the tissue) is about one-half or one-third of the separation of input points. Most of the neurons are of the pyramidal type. They have excitatory synapses on their axons and mostly excitatory synapses on their somatodendritic membrane. For sheer numerical reasons (Braitenberg, 1978a,b), the conclusion is inescapable that most of the cortical synapses connect pyramidal cells to other pyramidal cells. This diffuse network of pyramidal cells, rich in positive feedback, is locally molded by a variety of inhibitory (stellate) cells, whose shape and distribution and mode of action reflect the specific task of any particular area.

6. Periodicity of 0.4–0.5 mm in the Visual Cortex

In the monkey (Hubel and Wiesel, 1968), as in the cat (Hubel and Wiesel, 1962), the orientation of the elongated maxima or minima of luminance to which the cortical neurons respond changes in a regular, or almost regular way when the electrode proceeds tangentially through the cortex. In many electrode paths, there are stretches a millimeter or (rarely) more in length, for which the angles of orientation seem to vary as a linear function of distance in the cortex. The slope of this linear dependence is about 180°/0.4 mm (from the plots in Hubel and Wiesel, 1974a). As orientations 180° apart are identical, this seems to reflect something in the cortical network which repeats every 0.4–0.5 mm. This period is interesting, especially as it matches the width of the alternating stripes of input from the right and left eye. It must be remembered, however, that in the case of the ocular dominance columns, the periodicity across the direction of the stripes is twice that, if the afferents are also considered, because of the alternation of the input from the two eyes. We will discover soon that the distance of 0.4 mm which turns up in the two aspects of cortical architecture, is no accident.

The idea of long narrow bands of neurons with equal orientation specificity extending through wide stretches of the cortex, perhaps at right angles to the ocular dominance strips, is not very satisfactory because it implies a strong dependence of the steepness of orientation change on the direction of the penetration. If the slope is 180°/0.4 mm in one direction, it should be zero or close to zero in the perpendicular direction. This has never been demonstrated. On the contrary, all published records of orientation specificity as a function of position in the cortex seem to show about the same slope. This led us (Braitenberg and Braitenberg, 1979) to assume a simple dependence of orientation, not from the position of the neuron in a system of Cartesian coordinates of the cortex, but from its position in polar coordinates centered around special points, the "hypercolumn centers" and valid for the surrounding hypercolumn. In order to account for the prevailing periodicity of 0.4 mm, one would have to assume that the hypercolumns are about 0.4 mm wide in every direction.

Figure 4 illustrates two variants of this idea already proposed by von Seelen (1970). Each of the charts represents a piece of visual cortex seen from above. The lines on the diagrams represent orientation specificity of neurons in the corresponding positions, specified in cortical coordinates. The corresponding orientation in the visual field could be determined if the magnification factor and the orientation of the visuocortical projection were known (for small portions of the visual fields, we may assume the angles in the visual field to be correctly represented on the plane of the cortex). In Fig. 4a the orientations are arranged radially around hypercolumn centers, in b concentrically. The two schemes are identical except for a difference of 90° in the orientations. It can be seen that in both cases an electrode (arrows) traversing the tissue would encounter sequences of smoothly changing orientation, with occasional reversals of the sense of rotation and occasional discontinuities in the transition from one hypercolumn to the next. All of this is quite typical for the experimental plots.

More precisely, the model imposes certain quantitative restrictions on the sequences of orientation sensitivity which can be expected with straight tangential progressions through the cortex (Fig. 5). Traversing the hypercolumn off center, the plot would be S-shaped (Fig. 5a), with the steepest part corresponding to an orientation perpendicular to the electrode track (this is assuming the radial arrangement of Fig. 4a, while with the concentric arrangement of Fig. 4b the steepest part of the curve would correspond to an orientation parallel to the track). When the track goes through two adjoining hypercolumns (Fig. 5b,c), the plot would have two maxima of steepness, corresponding to the same orientation (or, when the plot is like in Fig. 5b and in many of Hubel and Wiesel's illustrations, to two orientations 180° apart). The transition is smooth when the

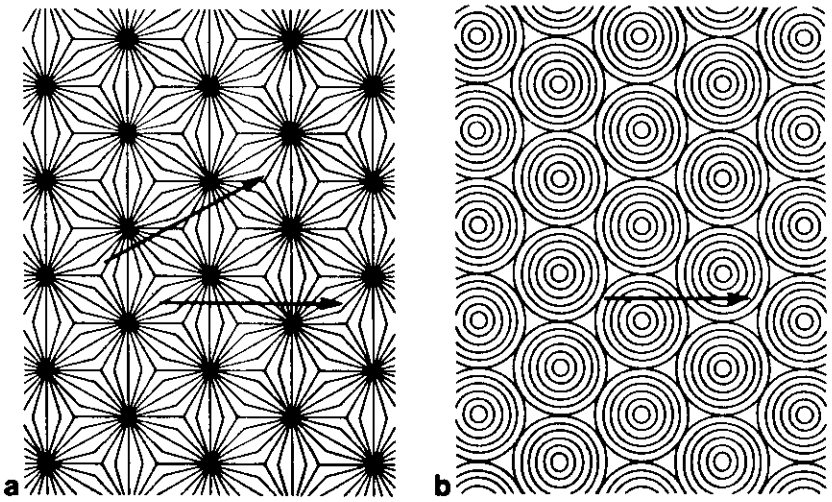

Figure 4. Alternative ways in which cortical neurons with different orientation specificity may be arranged in the visual cortex around centers spaced at distances of about 0.4–0.5 mm from each other. The radial lines emanating from the centers in (a) and the concentric circles of (b) represent orientation of receptive fields projected onto the plane of the cortex. The set of different orientations around each center is called an orientation hypercolumn. Arrows: electrodes advancing through the cortex encounter regular successions of orientation specificity. From Braitenberg and Braitenberg (1979).

plot changes from clockwise to counterclockwise between one hypercolumn and the next (Fig. 5c) while in the other case there is a jump (Fig. 5b) in the plot.

Keeping these rules in mind, it is possible to fit the experimental data with theoretical curves by assuming a distribution of hypercolumn centers in the vicinity of the electrode track (Fig. 6). Actually, the positions of these centers can be rigorously determined if the orientations are given with sufficient accuracy and further, the direction of the electrode path (in its projection onto the visual field) can also be unequivocally reconstructed. The method, a geometric construction, is simple and straightforward (Braitenberg and Braitenberg, 1979). The position of the centers relative to the electrode track is the same whether the underlying hypothesis is that of Fig. 4a or b, although the projection of the electrode track onto the visual field is at right angles in the two cases. This fact could be used to decide between a radial and a concentric arrangement of orientation specificities, but the progression of the position of the receptive fields which accompanies the changes in their orientation is not recorded in the experiments of Hubel and Wiesel (1974a) which served as a basis for this analysis.

We tend to favor now the scheme of Fig. 4b for reasons which will become apparent soon. Figure 7 is based on a concentric arrangement of orientations around hypercolumn centers. It is designed to show that very long progressions in which orientation angle varies almost linearly (within ± 15°) with distance along the electrode track are compatible with the present hypercolumn scheme.

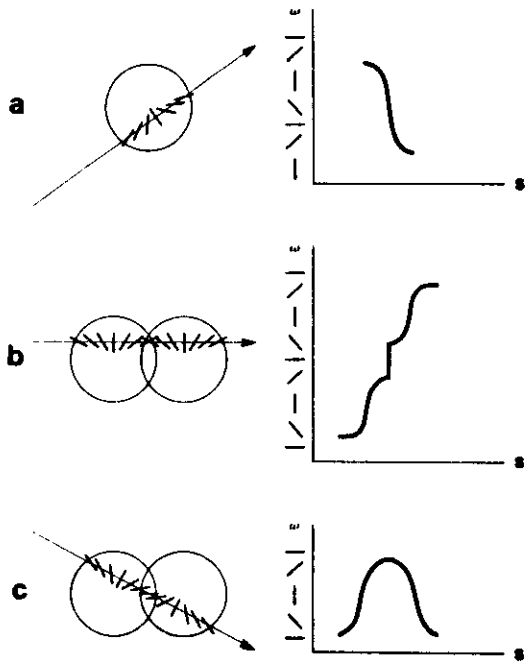

Figure 5. The sort of successions of orientation specificity that are to be expected in a typical Hubel and Wiesel plot according to the hypothesis embodied in Fig. 4a. The alternative hypothesis of Fig. 4b predicts the same curves, only shifted 90° along the ordinate. From Braitenberg and Braitenberg (1979).

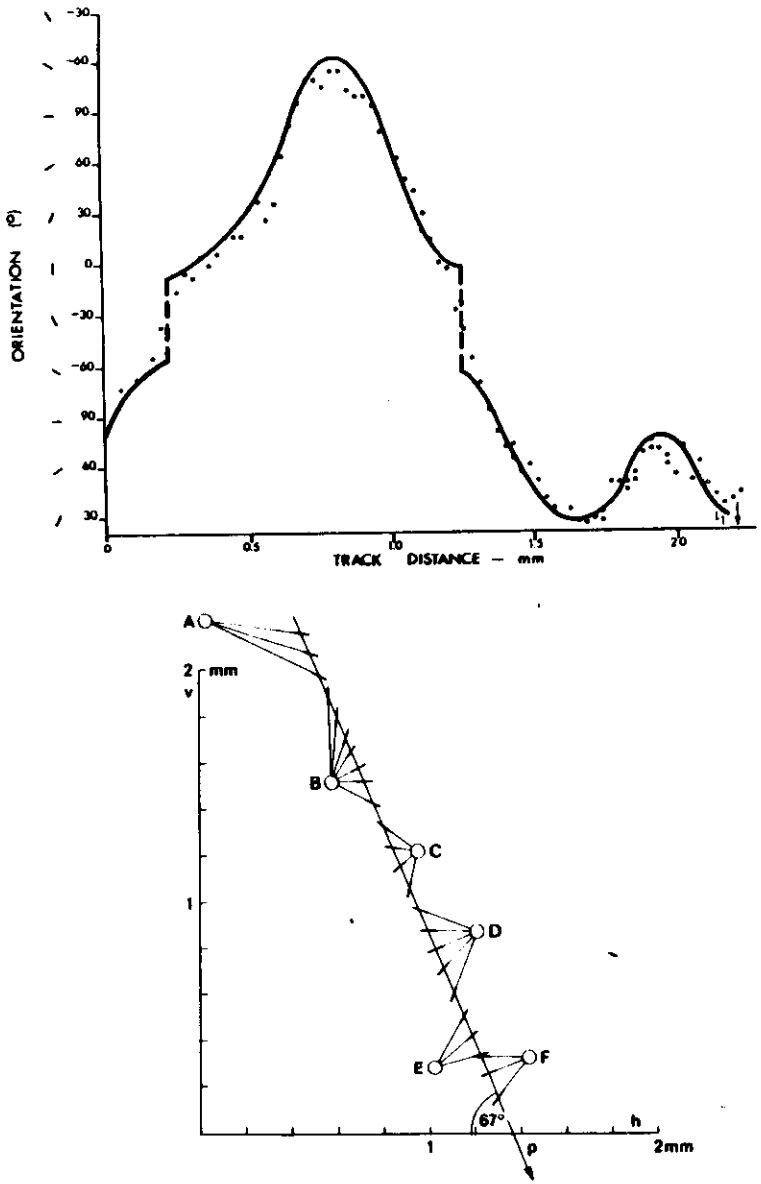

Figure 6. (Top) One of the plots in Hubel and Wiesel (1974a) (dots) interpreted according to the scheme of Fig. 5. (Bottom) A–F are the hypercolumn centers in the vicinity of the electrode track (p) around which the orientations are assumed to be radially arranged to produce the plot above. The coordinates of the lower diagram are the vertical and horizontal directions of the visual field, projected onto the visual cortex. From Braitenberg and Braitenberg (1979).

From all our reconstructions based on the Hubel and Wiesel (1974a) plots, a typical spacing of 0.4–0.5 mm between neighboring hypercolumn centers emerged. Unfortunately, there was nothing in the anatomy of the visual cortex, as it was known at the time, that could be identified with the hypercolumn centers, except perhaps the Meynert cells which are said to keep distances of about 0.4 mm between each other (Chan-Palay *et al.*, 1974). But it was difficult to see how these large pyramidal cells could organize the wiring of the hypercolumns around them in such a way as to produce all the known effects.

7. The Cytochrome Oxidase Blobs

A direct demonstration of the singularities predicted by our analysis of orientation–distance plots had to wait for the introduction of a cytochemical method. Horton and Hubel (1980, 1981) and Humphrey and Hendrickson (1980), using a technique proposed by Seligman *et al.* (1968) for the demonstration of cytochrome oxidase in the tissue, found a patchy distribution of the enzyme, with the patches (blobs) arranged in a fairly regular array at distances of 0.4–0.5 mm from each other. Horton and Hubel (1981) showed that single rows in the array of blobs correspond to single ocular dominance strips, with the blobs aligned in the middle of the strip. The arrangement is that of the circles in Fig. 2a and b.

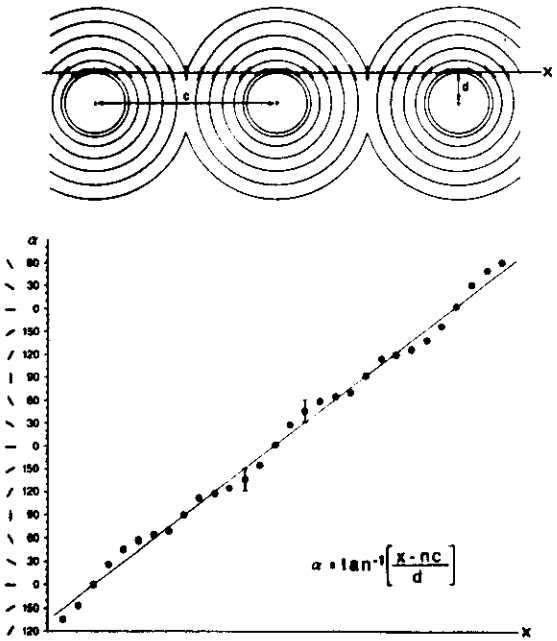

Figure 7. Example of how an almost linear progression of orientation angles (± 15°) along a straight electrode track is compatible with the arrangement illustrated in Fig. 4b. The mathematical expression gives orientation angles as a function of distance traveled by the electrode (x), distance of the hypercolumn centers from the electrode track (d), and distance between hypercolumn centers (c).

Contrary to a previous report (Carrol and Wong-Riley, 1982) which seemed to indicate stellate neurons with type II presumably inhibitory synapses on their axons as the preferential site of cytochrome oxidase activity, a more extensive study by the same authors (Carrol and Wong-Riley, 1984) showed the reactive mitochondria to be housed almost equally in pyramidal and nonspiny stellate cells both within and around the blobs. The impression of these authors was that the cytochrome oxidase stain reveals the level of activity of the neurons independently of their excitatory or inhibitory action. This makes it possible to uphold the interpretation which their first report suggested, namely, that of the blobs as regions from which inhibition emanates, reducing the level of activity in the surrounding regions. This supposition is supported by the recent finding of a higher density of glutamic acid decarboxylase in the blobs (Hendry *et al.*, 1984). If the blobs are the sites in which inhibitory neurons are concentrated, they could indeed organize the wiring of the column around themselves to produce all the known effects (Braitenberg, 1983).

8. An Efficient Model of Orientation Hypercolumns

Let the blobs be the sites of inhibitory neurons with dendritic fields slightly narrower than the hypercolumn and with dense axonal ramifications strongly inhibiting all the pyramidal cells within their reach. We assume the dendritic fields of the pyramidal cells to be about as large as those of the stellate cells in their projection on the cortical plane. The axon collaterals of the pyramidal cells are longer than those of the stellate cells and ramify less. They make a loose network of random excitatory connections with other pyramidal cells. The geniculocortical afferent fibers make point-to-point contacts indiscriminately with the stellate cells and with the pyramidal cells.

Observe the activity of a pyramidal cell (black cell body in Fig. 8) as a function of the input. Diffuse input excites both the pyramidal cell and its neighboring inhibitors (white cell body in Fig. 8; all the dendritic fields are outlined by large circles in Fig. 8) and will consequently be unable to activate the pyramidal cell. An elongated region of input activation (dashed rectangle) may activate the pyramidal cell if it is so oriented as not to affect the neighboring inhibitors (Fig. 8a) but will be unable to do so with other orientations (Fig. 8b). Thus, each pyramidal cell has a receptive field (Fig. 8c and d, cross-hatched) which is the region of its own dendritic tree minus the overlapping regions of dendritic trees belonging to the neighboring inhibitors which influence it through their axons. We may suppose that a pyramidal cell situated halfway between two inhibitory cells is inhibited by both of them. Consequently, its receptive field will be bicon-

cave (Fig. 8c). A pyramidal cell with its dendritic tree overlapping only with that of one neighboring inhibitory cell, or one inhibited only from one side, has a crescent-shaped receptive field (Fig. 8d).

It is important to distinguish these receptive fields or microfields of individual pyramidal cells from the much larger simple, complex, etc. receptive fields of Hubel and Wiesel, for reasons which will become apparent in the next section.

Whether crescent-shaped or biconcave, these fields have an "orientation," a long axis perpendicular to the line which connects them to the nearest hypercolumn center (Fig. 9). If we sample pyramidal cells within a hypercolumn and pay attention to the orientation of their receptive fields, we expect to find a rotation of the orientations along a straight electrode track quite like in Fig. 4b, or in Fig. 7.

9. Receptive Fields as Cell Assemblies

The assumption of centers of inhibition spaced at distances of 0.4–0.5 mm explains the distribution of orientation specificity on the cortical plane in a surprisingly simple, yet satisfactory way. But it does not explain the size of the receptive fields of the "simple" and "complex" kind described by Hubel and Wiesel and many other authors. What rotates in the visual field when the electrode samples a succession of neurons along a straight track are not the tiny crescents of Fig. 8, or the compass needles of Fig. 9, which are small compared to the distance of 0.4 mm over which a rotation of 180° is completed, but the much larger receptive fields of cortical neurons (Fig. 10). This becomes clear when the receptive fields, as they are plotted by the physiologist on the screen in front of the monkey, are referred to the system of coordinates of the cortex by means of the known laws of the projection (e.g., magnification factor, orientation of the visual field on the cortex).

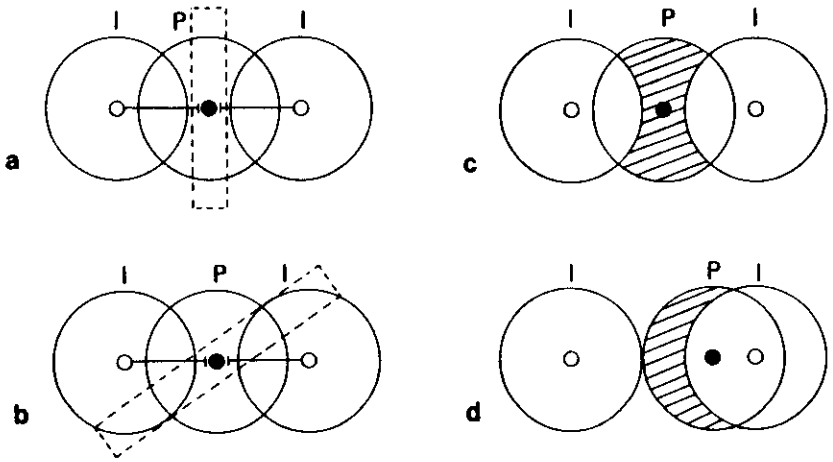

Figure 8. (a, b) How a cortical pyramidal cell (P) may become sensitive to oriented line segments (dashed rectangles) due to the influence of nearby inhibitory cells (I). The drawing is in the cortical plane. Large circles outline the dendritic fields. Small white circles: inhibitory neurons; small black circle: pyramidal cell. The stimulus must be so oriented as to hit the dendritic field of the pyramidal cell but not that of the neighboring inhibitory cells. P would respond in case (a) but not in case (b). (c, d) The receptive fields of individual pyramidal cells, because of the mechanism illustrated in (a, b), are either biconcave (c) or crescent-shaped (d), depending on whether they are under inhibitory influence from both sides or from one side only.

Several authors provide data on the sizes of receptive fields of cortical neurons in degrees of visual field (Wurtz, 1969; Poggio, 1972; Hubel and Wiesel, 1974b; Dow *et al.*, 1981). Where the eccentricity has been recorded, the size of the corresponding region in the cortex can be calculated by means of the function relating eccentricity and magnifiction factor (Daniel and Whitteridge, 1961; Hubel and Wiesel, 1974b; Dow *et al.*, 1981). When this is done, it turns out that the cortical field size varies considerably, perhaps by a factor of 2, but at least one of the sides of the (rectangular) field is always larger than 1 mm.

An example is shown in Fig. 2b in which the outer rectangular frame outlines a region of cortex whose projection onto the visual field corresponds to a medium-sized simple or complex receptive field. It will be seen that this field is much larger than the hypercolumn distance of 0.4–0.5 mm.

There is a problem when we try naively to imagine the neuronal circuits connecting the input points (several hundred, or a thousand) comprised by this huge field to the particular neuron from which we are recording. In the case of a "simple" field, we would suppose excitatory connections to our particular neuron from all the inputs belonging to some of the subregions of the field, and

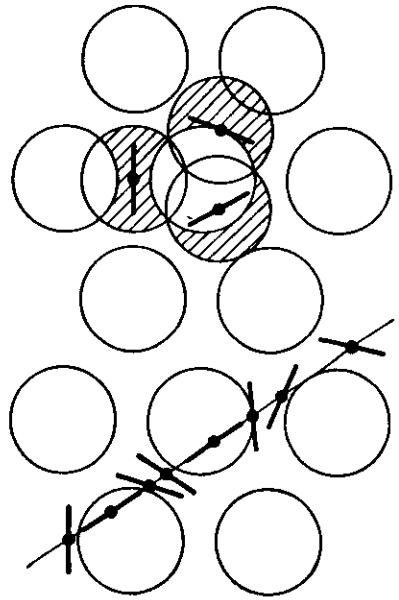

Figure 9. The white circles represent the inhibitory centers, schematically arranged in a regular hexagonal array. In the upper part of the figure, the receptive fields of three pyramidal cells (cross-hatched) belonging to one hypercolumn derive their orientation (compass needle) from their position relative to the hypercolumn center. In the lower part of the figure, a succession of orientations along a straight path is shown.

inhibitory connections from the other subregions. In the case of a "complex" cell, we would first have to construct a set of line—or movement detectors—all with the same orientation, distributed throughout the field, and then specific connections from these detectors to our neuron. This is complicated, and requires a great deal of very specific wiring, but the situation becomes absolutely unweildy when we picture the complete wiring for several neurons of one neighborhood. The real difficulty is in imagining the same wiring scheme, a few millimeters in overall size, repeated at a very slight angle for neighboring neurons about 20 μm apart, in order to produce over a distance of 0.4 mm the whole cycle of rotation which is apparent in the records. Of course, we could use selected sets of input fibers of the ON-center and of the OFF-center kind to explain at least the simple cell fields in a fairly simple manner, but this does not help much in the case of the complex fields.

I propose a more realistic solution. The keys to orientation sensitivity are the small crescent-shaped or biconcave fields of Fig. 8c,d. They belong to individual pyramidal cells of the cortex. As I have pointed out elsewhere (Braitenberg, 1978a,b), the system of pyramidal cells, the skeleton of the cortex, is characterized by a high degree of internal connectivity. For most of the axon terminals of any pyramidal cell, another pyramidal cell is the postsynaptic element, and presumably the synapses are excitatory. This makes the system of pyramidal cells an even better candidate for the associative network implicit in the theory of cell assemblies (Hebb, 1949). Also, we know now that Hebb was correct in his assumption of "plasticity" in synapses whose strength is adapted

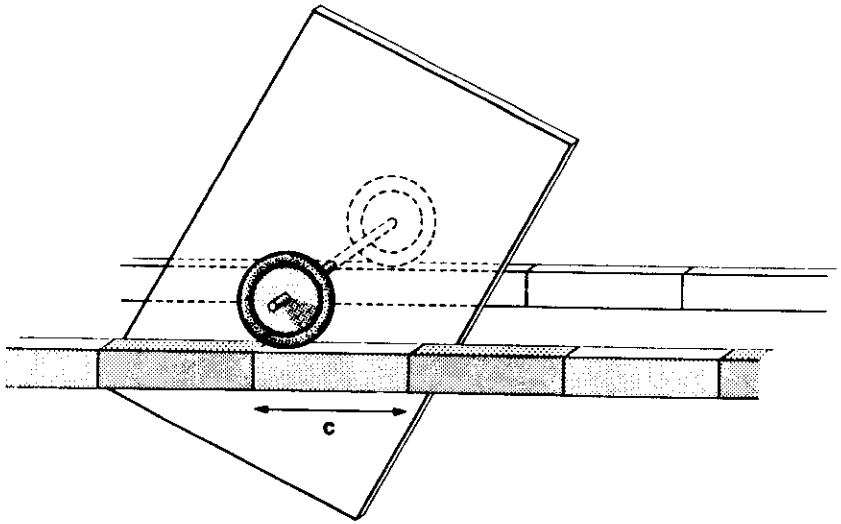

Figure 10. A mechanical model illustrating the size of a (Hubel and Wiesel type) receptive field compared to the length c which corresponds to a full 180° cycle of orientations. The comparison of these magnitudes emphasizes the problems connected with the idea of a preformed cortical "wiring" responsible for orientation columns.

to the frequency of coincident activity between pre- and postsynaptic neuron, for such synapses must be postulated in order to explain some effects (Hubel and Wiesel, 1965; Wiesel and Hubel, 1965; Rauschecker and Singer, 1979) in visual physiology.

My proposal is that pyramidal cells of different hypercolumns unite in a cell assembly when they respond to the same orientation (Fig. 11). Such cells are either located on the same or on opposite sides of the same hypercolumn center, or in corresponding positions of neighboring hypercolumns, or even of hypercolumns farther apart. There is enough divergence in the system of pyramidal cells [from one to thousands of pyramidal cells (Braitenberg, 1978a,b)] and their axon collaterals are long enough for sufficient reciprocally connected assemblies to form by chance. More importantly perhaps, contours sweeping the visual field will produce synchronous or almost synchronous activation of pyramidal cells tuned to the same orientation and will strengthen or even create the connections within such assemblies by some Hebbian learning process. The upshot is that we expect every pyramidal cell in the visual cortex to be under strong excitatory influence from several other pyramidal cells with identical orientation characteristics.

Note that when the physiologist maps the receptive field of a neuron in the cortex, what he really sees is the conjoined field of all the neurons making up the assembly to which the particular neuron belongs. This means, of course, that there are always several neurons having exactly the same receptive field, but if they are far enough apart, it is very unlikely that any neurophysiologist will ever discover this fact, as there is little probability of his recording from two or more neurons of the same assembly.

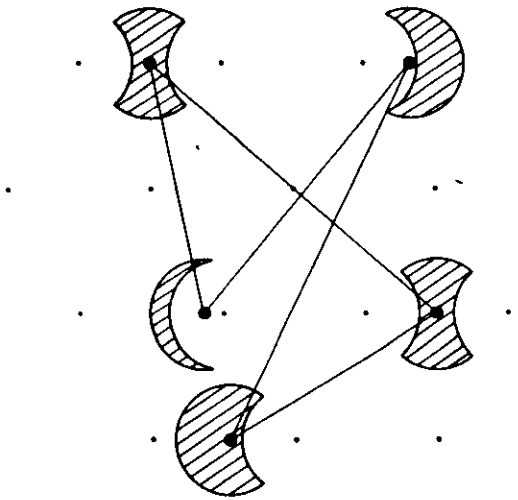

Figure 11. Illustration of the idea of a Hubel–Wiesel-type receptive field as the sum of many microfields of individual pyramidal cells with a similar orientation joining in a Hebbian cell assembly.

10. Consequences of the Cell-Assembly Concept

The idea of Hubel–Wiesel-type receptive fields as cell assemblies provides an easy answer to the puzzling question of the large size of the fields compared to the periodicity of the mechanism (0.4–0.5 mm) of orientation selectivity. We need not worry about the cumbersome wiring and its gradual variation from place to place within a hypercolumn. All we need is enough of a network of randomly arranged axon collaterals and dendrites of pyramidal cells for the corresponding partners to find each other and to associate into cell assemblies. Orientation specificity arises separately for each of the component neurons, in the local interaction of inhibitory cells and pyramidal cells.

The fact that all the neurons of one cell assembly have the same receptive field, in the sense in which this term is used by Hubel and Wiesel, has an interesting consequence. The position of the receptive field relative to the recording site is of course different for a neuron which is in the center of the assembly than for another which is in the lower left corner, for example. If

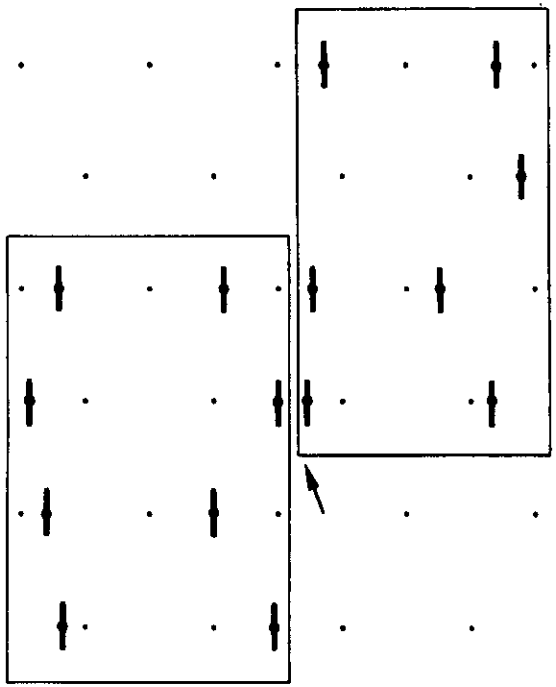

Figure 12. The dots represent the array of hypercolumn centers. The compass needles are microfields of the same orientation belonging to two different cell assemblies. The macro-receptive fields corresponding to these two cell assemblies occupy distinct positions in the visual field. Thus, neurons lying side by side in the cortex but belonging to different assemblies (arrow) may have receptive fields in the Hubel and Wiesel sense which are subject to considerable scatter.

neurons are selected at random, at one time the receptive field (translating the coordinates of the screen into those of the cortex) will be centered on the position of the neuron, and at another time it will be to one side or to the other. The physiologist will notice that when he is recording from a set of neurons in a small area of the cortex, the position of the receptive fields scatters by almost as much as the size of the fields (Fig. 12). This had indeed been found by several authors, notably Hubel and Wiesel (1974b). Implicit in this explanation is the assumption that neighboring neurons often belong to different cell assemblies even if they have the same, or almost the same, orientation. This is quite plausible because of the statistical characterisitics of the axon-collateral–basal-dendrite network (Braitenberg, 1978b): the axonal trees of pyramidal cells are so loose, and the synapses which they form are so widely dispersed, that even for two pyramidal cells in close proximity the probability that they are not connected by a synapse is greater than the probability that they have at least one synaptic connection. This statement is only in apparent contradiction with the other important fact of pyramidal-cell-pyramidal-cell connectivity, that each one is pre- and postsynaptic to several thousand different pyramidal cells. In fact, the direct synaptic partners are dispersed among a much more numerous population of neurons within a radius of up to 1 mm from the pyramidal cell in question.

The varying size of the receptive fields is also easily explained by the statistical nature of the wiring between pyramidal cells. Even within a small cortical region (to exclude the variation due to varying magnification factor) the fields pertaining to different neurons, whether simple or complex, quite regularly differ in size by a factor of 2, and sometimes twice as much, if I read the pictures in Hubel and Wiesel (1974b) and the plots in Dow *et al.* (1981) correctly. This is to be expected, if the accident of reciprocal connections among a small number of neurons in a random network is involved.

Another early finding of Hubel and Wiesel (1968), the narrowness of the optimal stimulus compared to the width of the receptive field, was later confirmed by many (e.g., Schiller *et al.*, 1976a,b; Dow *et al.*, 1981) but remained puzzling, especially in the case of the complex cells in which the preference for narrow stimuli is not related to any obvious fine structure of the receptive field. The idea which I introduce, that all large cortical receptive fields, simple, complex, etc., are really composites of many small crescent-shaped or biconcave fields of individual neurons, makes this finding quite understandable. We only have to realize that a stimulus, in order to excite one of these microfields without touching the fields of neighboring inhibitors, must be quite narrow.

11. Differences between Simple and Complex Cells

The original idea of a hierarchy—concentric (geniculate), simple, complex cell, each field of one level composed of several of the preceding level—has generally been abandoned for various experimental reasons (Hoffmann and Stone, 1971). The tendency nowadays is to assume that both simple and complex cells receive direct connections from the input fibers. What is the difference then? In the present view, both derive their receptive field characteristics by

being part of small cell assemblies connecting neurons of similar orientation housed in different hypercolumns. I propose the following explanation for the fact that such assemblies at one time have the characteristic of the "simple cells," at another that of the "complex cells" (Fig. 13).

When the orientation of the microfields of neurons forming an assembly corresponds to one of the axes of the array of hypercolumns (Fig. 13a), a narrow elongated stimulus presented at the right orientation may hit several inhibitors of a row, or none at all, depending on its position. At one time its effect on the neurons of the assembly will be purely inhibitory, at another time purely excitatory. When testing the receptive field in the usual manner with such a "slit" or "bar," parallel bands or subfields will be discovered with alternating excitatory and inhibitory effects on the neurons in question. The physiologist will then make the diagnosis "simple cell."

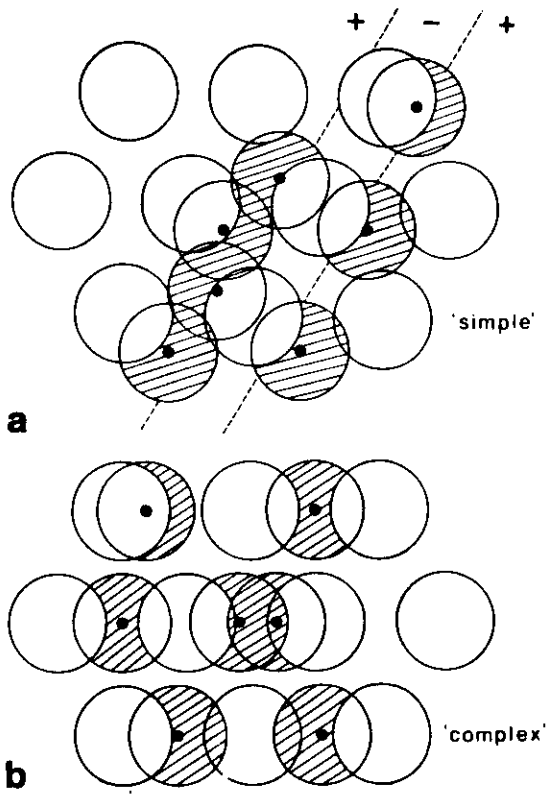

Figure 13. Simple and complex receptive fields depending on the orientations of the microfields relative to the axes of the array of hypercolumns. In the simple cell, a line stimulus of the appropriate orientation may hit only excitatory or only inhibitory subfields depending on the position. In the complex field, no such positions can be found because the orientation of the microfields does not correspond to any of the axes of the array.

If, on the other hand, the orientation of the microfields of an assembly does not correspond to any of the axes of the array of hypercolumns (Fig. 13b), a slit or bar of the correct orientation cannot hit only inhibitory hypercolumn centers, or only excitatory pyramidal cells situated between them, but will always hit elements of the two kinds. The physiologist will not be able to find a position in which the elongated stimulus produces only inhibitory, or only excitatory effects. He will find, however, that for any position of the stimulus, the neurons of the assembly are activated, for the stimulus will always hit some of the microfields making up the receptive field, and the corresponding neurons will ignite the rest of the assembly. Of course, in order to fit into the crescent-shaped or biconcave microfields without activating the neighboring inhibitors, the stimulus has to be narrow, just as narrow as in the previous case. It is clear that in the conventional terminology, what has just been described is the receptive field of a so-called "complex" cell.

This interpretation of simple and complex cells has an immediate consequence. If neurons are sampled in a narrow region of the cerebral cortex, simple cells should have some orientations, and complex cells the others. Depending on the geometry of the array of hypercolumn centers ("blobs"), the preferred orientations of simple cells could be one (if the blobs form rows but have random spacing within the rows) or two (if the blobs in neighboring rows are in register) or three (if the blobs form a regular hexagonal array, as in our diagrams). Two preferred orientations for simple cells, vertical and horizontal, have sometimes been claimed for the center of the visual field (Mansfield, 1974; Blakemore *et al.*, 1981). But I know of no evidence for or against the idea of complementary orientations of simple and complex cells except perhaps some of the plots in Leventhal (1983) which for certain (not all!) orientations show a complementary distribution of simple and complex cell frequency. In any case, Leventhal shows a dependence of orientation on the intrinsic coordinates of the cortex, which is much more evident for simple cells than for complex cells. This is in accordance with our model.

It will now be clear what was meant by the "underlying striation" of Fig. 1. The implication is that in the parallel subregions of excitation and inhibition which characterize simple cells, the rows of inhibitory hypercolumn centers with the intercalated pyramidal cells become visible. This could be tested by a quantitative analysis. The projection of simple cell fields onto the cortex taking into consideration the locally varying magnification factor, should result in a constant width of the subfields. The published evidence does not prove or disprove this. However, the question touches on some of the problems which emerge when cortical neurons are considered as space frequency filters.

12. Spatial Filters versus Space Frequency Filters: General Remarks

The reader of the neurophysiological literature not conversant with Fourier analysis may have difficulties in bringing together the view of the visual cortex as a set of spatial filters, as I have described it up to now, and that of "space

frequency analysis" of the visual input. This is not difficult in principle, and the polemic which has occasionally arisen about this point may be largely resolved at a didactic level.

We are all familiar with applications of Fourier analysis to acoustics. A reasonable question about an acoustic signal—certainly not the only one, and not even the most relevant one in most physiological contexts—refers to the frequency components of that signal. This is the question of which pure sounds, if any, added together produce the acoustic signal in question. Given an acoustic signal that lasts long enough, we may determine this by letting the signal intereact with long stretches of test sounds, e.g., sinusoids of different frequencies. An arrangement which takes the product of the signal and the test sound, and the average of that product for a long stretch of time, provides a measure of the degree of relation between the two. If we want to be finicky, we may shift the test sound a little with respect to the signal in order to find out in which relative position we obtain the best fit, and call that relative position "the phase."

Now, this is all very theoretical and not even very useful, as there are few signals that last long enough without a change to make this sort of analysis possible. Usually, in addition to the frequency components, we want to know the beginning and the end of the signal or the times when its changes. So we have to use very short test sounds, to be ready for the changes, and make the comparison over brief stretches of time. The trouble is that this introduces a fundamental difficulty: the shorter the test sounds, the less information we get about the frequency content of the signal (or about the test sound itself).

We end up with two machines, one for the frequency analysis and one for the time of occurrence of the signals, or with a compromise machine, called a sonograph, which provides some information about both.

We may now rewrite the same story for vision. A reasonable question about a picture—certainly not the only one and not even the most relevant in most physiological contexts—is whether it contains periodic elements and at what spacing they occur. We may do this by comparing the picture with various test patterns, say blurred black-and-white stripe patterns of varying periods which we lay over the picture (think of the picture and the test patterns as transparencies) and shift around a little to find the best fit. Only in this case we have one more thing that we may vary, namely, the orientation of the test patterns, for our picture may be vertically or horizontally or obliquely striped. Again, this sort of analysis is not very interesting in general, for in most cases the picture is not uniformly striped but may be striped one way in one place and another way in another place and perhaps not at all in some places. So we are driven to use test patterns which are much smaller than the picture and apply them everywhere in every possible orientation, well knowing that they give less information about the periodicity of the whole picture but some information about where the details are. Again, we may build the two extreme machines, one with the blurred stripe patterns for frequency analysis and one with very small test patterns, say small holes, for local analysis, or a compromise machine, with small periodic stripe patterns at all possible orientations. This is the analog of the sonograph for pictures, or if you wish, a model of the visual cortex.

In fact, receptive fields composed of parallel inhibitory and excitatory regions (Fig. 1) are good approximations to the sort of test patterns which represents the spatial analog of the filter in the sonograph. The two kinds—inhibitory–excitatory–inhibitory and excitatory–inhibitory–excitatory—represent the cosine phase, while those composed of only one excitatory and one inhibitory region stand for the sine phase. We are not surprised to find many of those test patterns of all possible orientations within any small region (= hypercolumn) of the cortex, for this was indeed the theoretical requirement.

However, there is one difficulty with the "sonograph" theory of the visual cortex, a contradiction with the idea of the periodicity of hypercolumns underlying the structure of individual receptive fields. If the fields of simple cells are related to a basic periodicty (in one, two, or three directions of the cortical plane) in the manner illustrated (for one direction only) in Fig. 1, one would expect all simple cells in a given region to be tuned to the same space frequency. But to make the sonograph analogy work, we would like a range of space frequencies to be represented in every position of the visual field. Thus, we must turn to those workers (Maffei and Fiorentini, 1977) who specifically investigated the distribution of neurons with different frequency selectivity both on the plane and in the depth of the (feline) cortex. I select three facts from their papers which seem relevant:

First, as the average size of receptive fields varies as a function of retinal eccentricity, the average space frequency to which they respond best varies inversely. This very strongly suggests that the period of the periodic pattern which is best seen by the neuron (the inverse of space frequency) is related in a simple way to the structure of the receptive field.

Second, in (mostly oblique) penetrations, preferred space frequency seems to vary in discrete jumps.

Third, simple cells have narrower preferences for space frequency than complex cells.

13. Wilson's Discrete Channels

Effects pointing to a discrete set of space frequencies have been discovered in human perception (Wilson, 1978, 1983; Wilson and Bergen, 1979). It seems that a small number of fixed space frequencies, first four, in later reports six, each dominate a region of the frequency spectrum centered around it or shifted somewhat toward the lower frequencies. The effect is noticeable in masking experiments: the influence of a certain masking frequency on the threshold of perception of varying test frequencies remains the same for a certain range of test frequencies. Outside this range, other masking frequencies become more effective. For the frequencies which are maximally effective in each range, Wilson (1983) gives the values 0.8, 1.7, 2.8, 4.0, 8.0, and 16.0 cycles/degree.

The tendency to have these discrete channels tuned in octaves (i.e., neighbors differing by a factor of 2) on a space frequency scale is obvious in these numbers. This is a natural way in which discrete acoustic frequencies arise in a trumpet or on a string, but I am unable to find a scheme by which octaves on a space frequency scale arise out of some periodicity of an underlying structure. Rather, I could imagine that multiples of an elementary space constant in the

structure correspond to certain space periods which in turn correspond to a discrete set of space frequencies.

This is an attractive idea when it is applied to the periodicity of the hypercolumn array in the visual cortex (Fig. 14). Taking 0.4 mm as the elementary period, we get the set of values 0.4 mm, 0.8 mm, 1.2 mm, 1.6 mm, corresponding to the angles in the visual field [assuming the magnification factor M of 6 mm/deg (Daniel and Whitteridge, 1961)]: 4′, 8′, 12′, 16′. The corresponding space frequencies are 15, 7.5, 5, 3.75 cycles/degree, which are well compatible within the experimental uncertainty with the upper four of Wilson's discrete space frequency channels (16, 8, 4, 2.8 cycles/deg).

But this again may seem strange to physicists, who are not wont to see subharmonics arise in periodic structures such as crystals. In nonlinear systems of interactions such as nerve nets, we are not surprised to find resonances for periodic patterns with maxima spaced at multiple distances of an elementary distance. If receptive fields are composed of microfields belonging to different hypercolumns, a periodic pattern with a period equal to one hypercolumn distance will maximally excite a receptive field composed of microfields housed in neighboring hypercolumns. But if, for instance, the cell assembly has formed

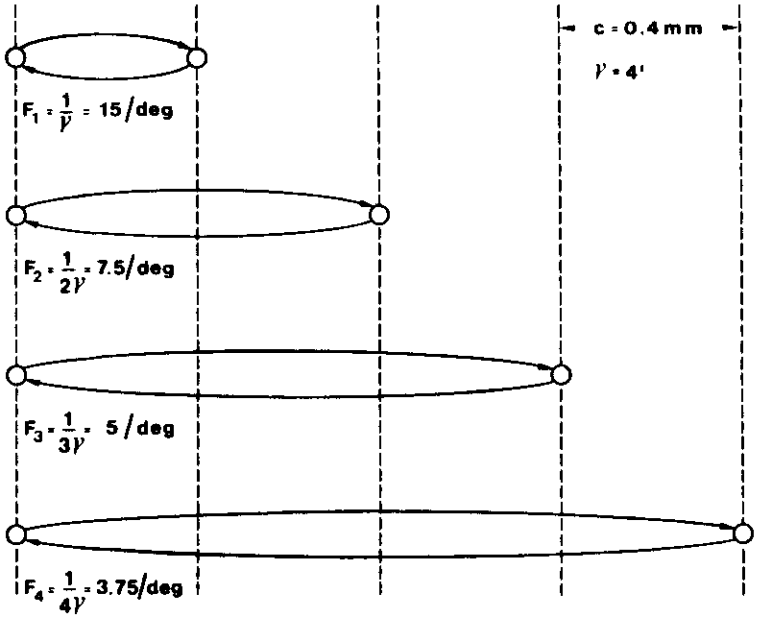

Figure 14. If receptive fields are composed of elements one, two, three or more hypercolumns apart, they may respond to figure elements spaced at the corresponding distances. If the hypercolumn distance in the cortex is taken as c = 0.4 mm, the corresponding angle (foveal magnification factor M = 6 mm/deg) in the visual field is 4′. Multiples of that distance are 8′, 12′, 16′. The corresponding space frequencies are 15, 7.5, 5, 3.75 cycles/deg. These values are not too different from Wilson's (1983) discrete space frequency channels: 16, 8, 4, 2.8 cycles/deg.

between elements in two hypercolumns separated by another hypercolumn, the periodic function to which it responds best will have twice that period, etc.

14. Epilogue: What I Did Not Discuss

As I mentioned in the beginning, the theorist may easily sacrifice some of the data to the coherence and efficiency of his model. Insofar as he is aware of this, he must talk himself out of the ensuing clash with other opinions.

A strong statement in the present story is the new interpretation of simple and complex cells. The most serious discrepancy is with the finding (Hubel and Wiesel, 1977a; Gilbert, 1977, for the cat) of a differential distribution of simple and complex cells in different layers: simple cells mainly in layer IVb, complex and hypercomplex cells in the other layers (see Martin, Volume 2 of this treatise). As simple cells, in our view, derive their properties from their alignment with respect to an underlying lattice, a possible explanation is that the geometry of this lattice is perhaps best defined in layer IV, and less in other layers.

Another finding which is not against our model, but does not immediately follow from it, is the different sensitivity of simple and complex cells to moving patterns of dots rather than lines. This finding may actually strengthen our model once the exact configurations of dots or other picture elements which excite complex cells maximally are defined.

The charts in Fig. 2a and b ignore some recent reports of afferent fibers supplying input to cortical loci several hundred micrometers apart (Gilbert and Wiesel, 1979). Much as one would like a precise point-to-point projection in the input to the visual cortex, with a grain at least as fine as that of retinal ganglion cells (and of the resolution of the visual field in psychophysics), some smearing does apparently occur. It is possible, however, that the branching geniculocortical input fibers are a residue of an early developmental stage in which the projection had not yet been refined by a learning process, such as is known to occur in the setting up of binocular vision (Hubel and Wiesel, 1965; Wiesel and Hubel, 1965).

The plasticity of intracortical connections as a basis of learning is an issue which I have avoided, although it is implied in the formation of cell assemblies among neurons with a common orientation. This is a natural assumption, in line with the general interpretation of the function of the cerebral cortex in associative memory (Braitenberg, 1978a,b; Palm, 1982).

I have also remained silent about various problems connected with the coordinate transformations in the projection of the visual field onto the cortex. In particular, the magnification factor varying as a function of eccentricity has led Fischer (1973), Schwartz (1976, 1977a,b), and others (Weimann and Chaikin, 1979; Braccini *et al.*, 1982) to a mathematical description with many interesting consequences. Partial experimental verifiction was provided by Tootell *et al.* (1982). These ideas are very relevant for any model of macroscopic analysis of the visual input, but are beyond the scope of the present chapter, which does not go beyond the mechanisms of local feature extraction. Schwartz (1977a,b) produced a model of orientation columns which competes with the model proposed here, and shares with it the advantage of not having to postulate unlikely

schemes of detailed neuronal wiring. But it precedes the discovery of "blobs," i.e., of a two-dimensional periodicity of the visual cortex which provides the basis for a much more elementary and therefore, perhaps, more appealing model of orientation hypercolumns.

Finally, I should mention other explanations of the puzzling phenomena connected with orientation sensitivity of visual cortical neurons. A model which has many features in common with ours is that described by Dow and Bauer (1983, 1984). This is also based on the idea of centrally organized hypercolumns proposed by von Seelen (1970) and Braitenberg and Braitenberg (1979). However, this model requires a more elaborate neuronal wiring than ours, and embodies the curious assumption that the cytochrome oxidase blobs represent the grain of the retinotopic map. This assumption is connected with the very high magnification factor measured by the same authors (see Table 1) but is quite astonishing in the face of the small total number of blobs, which does not exceed 9000 on each side. The Dow and Bauer model assumes special connections along two orthogonal axes of the cortical plane: psychophysics and electrophysiology provide arguments both in favor and against this idea.

The idea of small cell-assemblies uniting cortical neurons with similar response properties, one of the corner stones of the present model, also occurs in some recent reports by Shaw and Co-workers (Shaw and Pearson, 1983; Pearson *et al.*, 1983). However, a mechanism generating the elongated fields of individual neurons is not part of their theory, nor is the centric arrangement of orientation columns.

Not too many theoreticians (or practicing neurophysiologists, for that matter) have given serious attention to the problem of how the synaptic network is organized between the geniculocortical input and the neurons of the visual cortex to give them the well-known feature-abstracting properties. Rose's (1979) paper is remarkable in that it attempts to reduce the whole variety of simple, complex, and hypercomplex cells to different geometrical relationships between geniculate input fibers and cortical neurons, with intracortical inhibition shaping the receptive fields especially in the case of hypercomplex cells. The layout of orientation columns, however, is not explained by by this model.

Another scheme which relies on the geometry of the projection of input fibers on the cortical plane in combination with intracortical inhibition is that proposed by Dobson (1980). Here, parallel rows of elements in the cortex forming a hypercolumn correspond to fanning rows of elements in the retina, and inhibition between rows in the cortex therefore corresponds to inhibition between different orientations in the visual fields. This model explains many details of single-neuron physiology. It proceeds, however, from a premise which is contrary to ours, assuming as it does linear arrays of orientations in the cortex whereas we assume an arrangement around centers.

The model by Swindale (1982) is more concerned with the developmental forces which may produce an array of orientations starting from an initially homogeneous condition, rather than with the neuronal mechanism responsible for the physiological effects. The model is perhaps more complicated than it ought to be if the requirement for the genesis of orientation columns is nothing but the spacing of inhibitory centers at regular distances in the cortex, as we have proposed.

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