Afferent and Intrinsic Organization of Laminated Structures in the Brain

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"A mechanism for producing continuous neural mappings..."
A Mechanism for Producing Continuous Neural Mappings: Ocularity Dominance Stripes and Ordered Retino-Tectal Projections

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1. The Mechanism in General

We propose here a mechanism for the ontogenetic development of continuous projections between two sheets of neurons initially connected together in a disorganised fashion. We here discuss use of this mechanism for two specific purposes, namely establishment of ocularity dominance stripes and of ordered retino-tectal projections.

The proposed mechanism consists of two parts. One gives the two sheets intrinsic structure to provide a way of encoding distances between cells in the same sheet. The other reorganises the fibre projection with the help of synaptic plasticity. Before we discuss the applications we have in mind, we would first like to explain, in general terms, how the two parts of our proposed mechanism act together to set up continuous projections.

The internal organization of the neural sheets allows them to display only certain patterns of neural activity. In order to be used in a distance code the set of possible patterns must overlap. Their precise nature is decisive for the form of the final projection; in the simplest case they are local clusters of activity.

Plasticity is assumed to exist in the synapses connecting the two sheets together, and is here in the form of the correlation synapse, as proposed by many other authors. Each synapse is characterised by its strength, defined as the magnitude of the postsynaptic spike. A synapse is strengthened if there is a coincidence of activity in its pre- and post-synaptic cells, by an amount proportional to the product of the corresponding frequencies. Strengthening of synapses in this way results in a stronger correlation of pre- and postsynaptic strengths. To avoid a runaway situation we have to provide a stabilising mechanism. We therefore assume that the total strengths of synapses converging on one target cell is held constant. This leads to positive or
negative interference between fibres connected to the same target cell. If
such fibres are correlated in their activity (by being identified with the
same pattern of activity), their training effects are mutually reinforcing.
On the other hand, synapses made on the same target cell by fibres which are
anticorrelated never get reinforced.

After some time the modifiable synapses will have been rearranged so that
each possible pattern of activity in the pre-synaptic sheet will evoke one
of the possible patterns of activity in the post-synaptic sheet, thereby
establishing associations between pairs of patterns. For discovering which
patterns become linked together we use the crucial fact that each cell in a
sheet belongs to several overlapping patterns. Under this condition the final
projection has the property that overlapping stimuli will evoke overlapping
responses and the responses to non-overlapping stimuli will not overlap. The
geometrical interpretation of the resulting projections depends on the struc-
ture of the patterns of activity allowed.

At this stage it is worth giving a more biological interpretation of the
procedures involved in modifying the synaptic connections.

Initially, presynaptic fibres, on arriving at their target structure,
divide, forming terminal arborisations which make diffuse contact on the post-
synaptic cells. For our purposes it is irrelevant how many contacts each
fibre makes; all that matters is that no preference is displayed by a pre-
synaptic cell for any particular target cell.

The synapses so formed are strengthened during development according to the
Correlation Rule. However, owing to the operation of the Sum Rule, which could
be implemented by limiting the subsynaptic membrane area or by some sort of
habituation-like sensitivity control, strengthening of some synapses leads to
weakening of others. Thus, as time goes by some synapses reach zero strength,
that is, they disappear. However, the correlation procedure provides a way of
counterbalancing this gradual depletion of synapses. The best way of visualis-
ing this is to suppose that fibres which have already established synaptic
contacts are continually putting out new branchlets to make contacts with neigh-
bouring target cells. In this way the overlapping arborisations of two fibres
will attract or repel each other depending on whether the fibres are correlat-
ed or anticorrelated.

We have considered three different applications of our mechanism. In all
three cases the pre-synaptic structure is identified with the retina, and
we assume that the possible patterns within the target sheet are localised
roundish clusters of activity. The pattern of interconnectivity producing
this is excitation at short range and inhibition at long range.
In the first application the active patterns in the retina are line segments of different orientations, produced by light stimulation. The second sheet is identified with the visual cortex (we have left the lateral geniculate nucleus out of consideration). The retino-cortical projection becomes organised so that the cortical cells have elongated receptive fields (that is, are orientation specific) and that neighbouring cortical cells belong to neighbouring stimulus orientations, as has been found experimentally in the visual cortex of cat (Albus 1975) and monkey (Hubel and Wiesel 1975). This application has been extensively discussed elsewhere (Malsburg 1973).

2. Ontogenesis of Ocularity Dominance Stripes (Ch.v.d.M.)

Bands of cells in area 17 of monkey visual cortex are dominated in alternation by input from the left and from the right eye (LeVay et al. 1975). To explain this phenomenon we first take as given the retinotopical projection of the two eyes, via the lateral geniculate nucleus, onto the cortex. Let us now consider two small regions in the two retinae connected to the same cortical region. It is assumed that in the unorganised state of the system the retino-cortical projection can on the small scale be considered random, in the sense that the values of the appropriate synaptic strengths can be regarded as a set of random numbers. Thus ocular dominance is initially distributed randomly over the cortex; for all cortical cells the ratio between the combined synaptic strengths from one eye to those from the other eye is near to unity.

To cause the retinocortical connections to reorganise to give the cortical cells a strong ocular dominance we suppose that the retinal spontaneous activity is correlated for cells in just one eye and is anticorrelated for cells in both eyes. (This anticorrelation could be produced by inhibition within the LGN). No further condition is required except that each retinal region must have several degrees of freedom, that is, it should feature in several different (and overlapping) patterns. The reason for this will become clear later.

As a result of the anticorrelation mechanism, as organisation proceeds cortical cells will tend to disconnect from one eye and thereby establish exclusive contacts with the other. Furthermore, since overlapping patterns of activity in one eye will come to evoke overlapping patterns in the cortex, whole regions of cortex will tend to connect to the same eye. In other words, neighbouring cortical cells will avoid developing different ocularity. Therefore, in the final structure reached after development the length of the line dividing regions of different ocular dominance will be at a minimum.
In the computer simulations done so far the model used had a structure very similar to that used previously (Malsburg 1973). Two sets of five cells, representing the two retinas, were connected in an initially random fashion to a cortical sheet in the form of a hexagonal array of 169 excitatory ("E")- and 169 inhibitory ("I")- cells. Each E-cell excited its six closest E- and seven closest I- neighbours. Each I-cells inhibited a surround of 61 E-cells and 60 I-cells lying within the hexagonal surround of "radius" 4. The theoretical model employed for the cortical cells was similar to that used by Malsburg (1973). (A difference was that EPSP's and IPSP's were multiplied by factors proportional to the difference between the actual membrane potential and the excitatory and inhibitory equilibrium potentials, respectively. This is more realistic than having a linear compound PSP, and it also makes the differential equations describing the interacting cortical cells inherently stable).

The retinal patterns consisted of activity in the five pairs of cells [1,2] [2,3] [3,4] [4,5] and [5,1] in the left and in the right eye, and at each time instant one of these 10 patterns was active.

Figure 1 shows the time course of reorganisation of the cortical E-cells. The two ocularities are denoted by + and -. The first state corresponds to a random distribution of ocularities. In subsequent stages the line drawn between regions of different ocular dominance gets shorter and shorter until in the last stage shown there are ocular dominance stripes corresponding to a minimal configuration.
The spacing between the stripes is determined by the range of the inhibition. This leads to the experimental prediction that during the ontogenetic phase in which ocularity dominance is developed there is a locally organised system of intracortical inhibition with a range equal to the spacing of the developing ocular dominance stripes.

It is now clear why the two retinal regions must have several degrees of freedom: to each stimulation the cortex responds by activity in the form of a distribution of isolated round clusters of activity. The clusters activated by one eye must be able to cover the whole surface of the stripes of the appropriate ocularity, different clusters being activated at different times. The question whether these additional stimulus parameters have some functional meaning will not be discussed here.

A more detailed account of the work described in this section will be published elsewhere (Malsburg 1976).

3. The Establishment of Topographically Ordered Projections

(D.J.W. and Ch. v.d.M.)

We now consider the problem of the establishment of topographic mappings themselves, such as the orderly retinotectal projections found in lower vertebrates. Although we here refer to our pre- and post-synaptic sheets as "retina" and "tectum" the mechanism discussed could well apply to other systems.

The special assumptions we make here are: the stimuli to direct the system's development are provided by spontaneous activity in the retinal cells. Secondly, as a result of the lateral connections within a sheet, its typical pattern of activity at any instant of time is strong activity in a small localised area and little or no activity elsewhere. Under these conditions, in the final configuration each small area in the retina is associated with a small area on the tectal surface; overlapping retinal areas connect to overlapping tectal areas, or in other words, there is a continuous mapping of retina onto tectum.

We also have to supply information to specify the orientation of the map of retina onto tectum. An economical way of doing this is to set up a few pairs of pointers, that is, to choose a few retinal cells to initially make preferential contact to a few chosen tectal cells. Any cell pairs, as long as their relative orientation is correct, can act as pointers; we are not obliged to choose those pairs which are to match together in the final mapping and as few as two pairs can suffice. Establishment of the mapping then proceeds in a step-by-step fashion. The cells adjacent to the pointers are the next to establish definite contacts, and so order spreads out over the whole of both surfaces.
We have performed extensive computer simulations on various systems containing 30 - 50 retinal cells connecting with 30 - 60 tectal cells, to show that projections set up in this manner display "systems matching" behaviour (Gaze and Keating 1972). Whatever the relative size of retina and tectum, the mapping which develops is an ordered projection of the whole of the retina over the whole of the tectum. Subsequent alteration in the size of one or both sheets brings about a rearrangement of connections so that the new retina becomes mapped across the whole of the new tectum. The projections are always ordered and undistorted. If the pointers chosen did not come from corresponding sites in the two sheets then, as the mapping develops, these cells gradually change their partners so as to avoid distortions in the final pattern of projection.

A full treatment of this subject, including details of our computer simulation experiments, will be found in another publication (Willshaw and Malsburg 1976). For a recent review of much of the experimental work on the retina-tectal projection in amphibia and fishes the reader is referred to the paper by Prestige and Willshaw [1975].

It should be added that for the applications discussed in section 2 and 3 an interpretation in terms of spike activity in nerve cells is not the only possibility. One could as well think of a system in which cells are labelled by a particular selection of molecules taken from a larger set of possible ones. The expression "this set of cells is active at one time" would then be translated into "this set of cells all contain molecules of the same sort" and "activity in the pre- and post-synaptic elements of a synapse is correlated" would become "the collection of molecules in the pre- and post-synaptic elements has a similar composition". Our mechanisms in this form may be able to explain ontogenetic problems in early embryonic stages when there is not yet any nervous activity. We are at present exploring this possibility.

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Spatial Filtering in the Visual Pathway

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Introduction

In recent years two different concepts have developed of the functional organization of the visual cortex. One, derived mainly from physiology and functional anatomy (see B. Brooks and R. Jung [1973] and R. Jung [1973] for references) takes visual cortex to be organized, at least in its earlier stages, for the detection of standing edges and bars, appropriately presented, with a systematic local variation in cortical receptive field size, giving rise to optimally sensitive detection at differing locations. The other conception, derived from psychophysics (see J.G. Robson [1976]), holds that the visual cortex is a kind of spatial Fourier analyzer, with each location in the cortex sensitive to a number of spatial frequencies, and each spatial frequency being optimally detected in many locations. In this paper I outline a theory that seeks to reconcile psychophysical and physiological findings, with known details of the properties and distribution of retinal ganglion as well as cortical cells, and with the retino-geniculo-cortical map (see J.D. Cowan and H.R. Wilson [1976] for an extensive review).

How to Model Large Scale Nervous Activity

Any theory that seeks to integrate anatomical, physiological, and psychophysical data must contain a model of the perceptual process. In what follows it will be assumed that perception is a dynamical process. That is, the visual cortex is taken to be a net of excitatory and inhibitory cells, a time-varying proportion of which are activated in any given instant, by geniculate or other excitation, or by intrinsic local currents. As such, the cortex is an excitable dynamical system possessing stationary states in which a constant proportion of cells are activated (see H.R. Wilson and J.D. Cowan [1972, 1973] and J.L. Feldman and J.D. Cowan [1975a,b], J.D. Cowan [1974]). In particular there is a stationary state corresponding only to random low