The Correlation Theory of Brain Function *

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Abstract

A summary of brain theory is given so far as it is contained within the framework of Localization Theory. Difficulties of this "conventional theory" are traced back to a specific deficiency: there is no way to express relations between active cells (as for instance their representing parts of the same object). A new theory is proposed to cure this deficiency. It introduces a new kind of dynamical control, termed synaptic modulation, according to which synapses switch between a conducting and a non-conducting state. The dynamics of this variable is controlled on a fast time scale by correlations in the temporal fine structure of cellular signals. Furthermore, conventional synaptic plasticity is replaced by a refined version. Synaptic modulation and plasticity form the basis for short-term and long-term memory, respectively. Signal correlations, shaped by the variable network, express structure and relationships within objects. In particular, the figure-ground problem may be solved in this way. Synaptic modulation introduces flexibility into cerebral networks which is necessary to solve the invariance problem. Since momentarily useless connections are deactivated, interference between different memory traces can be reduced, and memory capacity increased, in comparison with conventional associative memory.

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Contents

1 Introduction

The purpose of this paper is to point out a specific deficiency in existing brain theory and to propose a way in which this gap could be filled. Although it leaves open a number of technical questions and presents more a program than an accomplished theory, at least a language is developed to describe processes from the cellular to the cognitive level.

Searching for the function of the brain in all generality may be regarded as a venture bound to fail in view of the diversity of function of even a single brain. It is clear that any answer to the question can only be of a very general kind, much as a "general theory of painting" can only be a description of the process by which pigments are prepared and applied to canvas with a brush, and could say nothing about art, subject, style and interpretation.

There is every reason to believe in the existence of general principles governing the function of the brain. Cerebral anatomy is surprisingly homogeneous in spite of the diversity of functional modalities represented in its different parts. The rapid cultural development of man has created fields of mental activity for which the brain cannot have been prepared by phylogeny in any detailed way. Both arguments seem to force the conclusion that the brain is governed by general principles of organization.

2 Conventional Brain Theory

The literature on brain theory is vast and cannot be summarized here. This chapter concentrates on a set of ideas which is fairly consistent in itself and with experiments. My account passes over most of the rich and sometimes ingeneous detail to which these ideas have been worked out in the literature. However, I try to bring out points where the ideas fail.

2.1 Localization Theory

2.1.1 The Macroscopic Level

Observation of behavioural defects caused by localized lesions of the brain has firmly established that different parts of the brain are preoccupied with different *modalities,* topics of mental activity [Luria, 1973]. Examples are vision, audition, motor control, basic emotions and drives (e.g. aggression, pleasure and hunger), various aspects of speech, and long-term planning of action. The ability to lay down long-term memory can be destroyed by a specific local lesion; however, already existing long-term memory is not affected thereby. Memory traces and the ability to recall seem to be localized together with the modalities to which they refer.

Several kinds of hierarchy can be construed on the basis of the modalities. For instance, sleep-waking regulation, drives, emotions and planning all exert global control over the rest of the mind. However, I will treat all localized topics as on the same level. The term hierarchy will be reserved to forms of cooperation of localized objects.

There are aspects of the mind's function which can not be localized in parts of the brain. Among these are consciousness and reason.

2.1.2 The Microscopic Level

In recent decades localization theory has been refined down to the microscopic level. The information carrying units are thought to be nerve cells. These produce signals in the form of short (1 msec) electro-chemical pulses, which can be recorded with the help of fine electrodes.

How are these signals to be interpreted? For the more central stages of the brain neurophysiology has answered this question in an operational way. In a vast majority of experiments signals are evaluated in terms of a peri-event time histogram. An event is constituted by a stimulus presented to the brain or a response evoked from the brain. The time shortly before and after the event is divided into a raster of small intervals (typically 1 to 10 msec). The event is repeated and the mean number of spikes falling into each interval is recorded. The mean frequency reacts to the event, if at all, by a short increase or decrease. The art of the experimenter consists in finding an event which influences the activity of the cell he is recording from. In this way a topic can be assigned to the cell. A typical topic is "an edge of light with a particular spectral component moving in a particular direction over a point of the retina".

The success of neurophysiology with this type of experiment has been tremendous. It is true, not all of brain has been mapped in this way, and in fact it may not be practical to do so, because some of the events may be difficult to find. Nevertheless, many scientists are ready to extrapolate the microscopic version of localization theory to all of the central nervous system. In this ultimate form localization theory can be summarized as "one cell - one topic": cells are the atoms of meaning.

2.1.3 The Brain as a Projection Screen

The topology of the periphery is preserved in the central representations, e.g. neighboring points of a sensory surface project to neighboring points on a central sheet of cells. To each point of a sense organ (retina, cochlea, skin) there corresponds a small region centrally, often called hypercolumn. Single cells in that region are specialized to particular combinations of quality values describing the point of the sensory surface (e.g. spectral distribution, direction of movement, stereo-depth).

A single peripheral surface is represented by several central fields, which may vary in their emphasis on different qualities, and which usually are connected by topologically ordered fibre projections.

Again, this picture has to be extrapolated to cover all of brain. It presents an experimental challenge to find the precise terms of this extrapolation. Let us suppose it will turn out to be possible. Then the physiological-anatomical picture is that of a screen on which patterns appear corresponding to input, output and internal processing (e.g. emotional, planning), similar to the moving pictures on a colour television screen.

2.2 The Problem of Nervous Integration

2.2.1 The General Question

The picture of the brain as a projection screen is very suggestive, and in its principal traits it is well founded in experimental observation. However, the picture poses a problem, that of nervous integration: in what way do the different patterns of activity interact? To be sure, the machinery for cellular interactions is conspicuously there: each cell is contacted on its dendritic and somatic membrane by many synapses through which its membrane potential can be raised or lowered upon arrival of nerve impulses. The axonal fibres and branches for the transport of impulses from cells to synapses fill the larger part of the brain's volume. The nervous integration question more precisely asks how this machinery is organized. The problem calls for new concepts, and at present it cannot be attacked experimentally.

One can train or ask a subject to react to the presentation of an apple by pressing a button: on command the subject can organize its brain so that upon the appearance of a certain class of patterns in the visual modality another pattern of a certain structure is created in the motor modality. This simple everyday example alone combines in it several complex organization processes which will now be named.

2.2.2 Representation of Structured Objects

In our cultural world we form symbols of a higher order by the juxtaposition (in time or space) of symbols of a lower order, e.g. words out of letters or phonemes. According to localization theory neurons are the basic symbols in the brain. Their position is fixed and cannot be used to form groupings. Another code is required to represent associtation of cells into patterns forming symbols on a higher level.

When we analyse a complex visual scene it is important to break it down into patterns which are simple enough so that we can hope to recognize them, e.g. identify them with objects we saw before. A single pattern in turn has to be broken down into subpatterns, possibly through several stages, e.g. man - arm - hand - finger - joint (cf. [Sutherland, 1968]). It should be possible to group neurons into such a hierarchy of patterns in a flexible way, without the introduction of new hardware for new patterns.

2.2.3 Invariance

It is an everyday experience that there are objects of a relatively fixed structure, which affect our senses in an enormously variable way. E.g. the picture of an apple can vary in perspective and in position and size on the retina, depending on the relative coordinates of eye and apple. It is important to reduce this variability to an invariant representation of the intrinsic structure of the object, in order to be able to generalize, i.e. draw the same conclusions from the perception of an object, irrespective of variations in its appearance [cf. Sutherland, 1968]. An analogous discussion applies to motor patterns.

2.2.4 Memory

There must be a physical basis for the gradual acquisition of information. We usually discuss it under two aspects. According to one the brain changes to acquire new abilities. This will be the subject of the subsequent paragraph. The other is the ability of the brain to recall complex patterns which were active at an earlier time. With this memory in the narrow sense, recall should be evoked by an activity pattern or an input which are sufficiently close to the original pattern.

2.2.5 Self-Organization

Self-organization refers to the ability of the brain to organize structures and activity patterns. The term "organize" implies that the process is directed toward some useful goal, which still has to be defined. A goal we already mentioned is the retention of earlier states of activity. In this way the brain can become independent of external sources of information and can build models for phenomena. Other goals will be defined in later sections.

The ability to organize itself sets the brain in sharp contrast to the computer, which relies entirely on a human programmer. It also is the basis of the reliability of the brain, being able to "repair" deviations from an optimal configuration. Self-organization puts heavy constraints on possible functional schemes for the brain.

2.2.6 Control of Action

The metaphor of the brain as a projection screen assigns a passive role to it. In reality we know that the brain is spontaneously active: The "projector" is an integral part of it, to stay with the metaphor. Accordingly, a solution to the nervous integration problem has to include a scheme for the control of processes and the global integration of action.

2.3 Proposed Solutions

Localization theory, section 2.1, proposes a basic frame into which any functional scheme for the brain has to be fitted. It poses the nervous integration problem, some aspects of which have been presented in 2.2. This section discusses some potential solutions which have been proposed in the literature, and points out some problems which they do not solve.

2.3.1 Synaptic Plasticity

A synaptic connection can be characterized by the size of the postsynaptic conductance transient (PCT) which is produced in the postsynaptic cell upon arrival of a nerve impulse in the presynaptic fibre. PCT size may slowly change under the control of the neural signals on the presynaptic and the postsynaptic side. This leads to a feedback situation: PCT size (together with the presynaptic signal) has influence on the postsynaptic signal, which in turn controls the change of the PCT. If this feedback is positive and if the changes impressed on the PCT are permanent (non-decaying) one speaks of synaptic plasticity. The formation of new synapses may be included in the definition of synaptic plasticity [Ariëns Kappers et al., 1936]. In the case of an excitatory synapse the EPCT (excitatory PCT) is increased (or a synapse established) after coincidence of neural activity on the presynaptic and postsynaptic side. In the framework of localization theory this is a straight-forward implementation of the idea of an association and of Pavlows conditioned reflex. It is usually assumed that plastic synaptic changes need seconds to become established and hours to consolidate (show full effect and stabilize). Synaptic plasticity has been shown experimentally to exist [Bliss and Gardner-Medwin, 1973, Baranyi and Feher, 1981], although it is, in the presence of controlling signals, intrinsically difficult to demonstrate its non-decaying nature.

The instability which is caused by positive feedback has to be controlled somehow. Several schemes have been proposed: an upper limit to the synaptic weight (PCT size for a single pulse); limitation of the sum of all synaptic weights converging on a cell or diverging from a cell; and stabilization of the mean level of activity in the postsynaptic cell. The latter says that if the time average (over, say, several hours) of cell activity exceeds a certain value either all excitatory synapses converging onto the cell are reduced in weight by a certain factor (and if this average is too low the synapses are increased in weight), or the inhibitory synapses are increased in weight.

Synaptic plasticity is thought to be the basis of memory. The positive feedback involved in it leads to the kind of instability that is required for pattern generation and self-organization. In this sense synaptic plasticity is analogous to self-reproduction in biological evolution.

2.3.2 Feature Detectors

In the context of theoretical discussions within the frame of localization theory a cell in the sensory part of the brain is termed a *feature detector, feature* being the term for the event by which the cell is triggered [see e.g. Sutherland, 1968]. Feature detectors may differ in level. On the lowest level they respond to the signal of a peripheral sensory cell. On the highest conceivable level feature detectors respond to the appearance of entire objects [Barlow, 1972]. They are then referred to as cardinal cells. Feature detectors of intermediate level are found experimentally (a typical feature has been described in 2.1.2).

Fairly specific feature detectors are proposed in many models of perception as a basis for the discrimination between complex patterns. The postulated level of feature detectors is regulated by a trade-off. The higher the level (the more specific the cells) the smaller the overlap of the sets of cells responding to different patterns and the easier the task of discriminating between them. High-level features, on the other hand, mean large numbers of cells, less flexibility (because specific trigger features must be adapted to particular pattern universes) and low duty cycle for each particular cell.

Many models employ cardinal cells because they seem to solve the problem indicated in 2.2.2, representation of complex objects. In reality that problem is not solved by cardinal cells. Either a cardinal cell is able to represent a whole class of objects. Then the individual object cannot be represented in detail, because the signal of a single cardinal cell is too crude. Or there has to be a cardinal cell for each pattern (a person with a new facial expression constituting a new pattern!). The number of cardinal cells required would then be forbidding (even if the invariance problem 2.2.3 had been solved somehow), and it would be impossible to recognize new patterns which differed from familiar ones merely in detail. In addition, a cardinal cell would have to be silent (possibly for decades) until its pattern appeared again, but there is every reason to believe that a cell which is forced to stay silent for a day (e.g. by deafferentation) will change its synaptic make-up to become active again.

From this discussion it follows that high-level feature detectors do not solve any of the nervous integration problems. Low-level feature detectors, on the other hand, are an experimental fact and have to be the building blocks of any theory under the roof of localization theory.

2.3.3 Cell Assemblies

Stimulation of some part of the brain will switch on many cells simultaneously. It therefore appears natural in the context of localization theory to regard sets of simultaneously activated nerve cells as the basic internal objects. The nervous integration problem requires that such sets should not just be passively activated by stimuli, that they should rather be dynamical units, integrated by interactions. The *cell assembly* [Hebb, 1949] is a model idea describing a certain system of such interactions.

A cell assembly is a set of neurons cross-connected such that the whole set is brought to become simultaneously active upon activation of appropriate subsets, which have to be sufficiently similar to the assembly to single it out from overlapping others. In view of the fluctuating nature of cellular signals activation of cells in an assembly is simultaneous only on a coarse time scale, longer than, say, 50 msec.

Assembly reconstitution, or its completion from parts, has been proposed as the fundamental process of brain function. Important special cases would be the attachment of abstract symbolic representations to sensory patterns (recognition), the reactivation of supplementary information stored by past experience, and the generation of a response pattern which has previously been associated with a stimulus. According to this view, the action of the brain is controlled by a succession of such completion processes, alternating with the (partial) decay of assemblies (due to some delayed disfacilitation) leaving residues which, together with new stimuli, form the germs for other assemblies.

Analysis of long periods of brain activity would reveal a hierarchy of subpatterns which appear as part of many assemblies. The dynamics of assembly completion possibly could be interpreted as interaction among subassemblies, analogous to excitation and inhibition exchanged between single cells. Subassembly interactions would have to be realized with the help of the synaptic interactions of the constituent cells.

It is an unsolved question whether assembly interactions with these specifications are possible [see Legendy, 1967]. However, the assembly concept has a more fundamental flaw. When a particular assembly is active, there is no basis on which it could be analysed into subassemblies: it just consists of a mass of simultaneously active cells. (The above analysis into subassemblies was only possible in a Gedankenexperiment.) This must lead to serious ambiguities. For instance, when we see a visual pattern, it is not only necessary to know which collection of features apply to it, but also in which way they are grouped. Even if the feature set is so complete that it can only be combined in one way into an image it is important to know this combination. (When we see two people in the street we usually don't confuse which jacket is worn together with which trouser by one of them.) In particular, it must be possible to represent the result of a successful figure-ground discrimination.

2.3.4 Associative Memory

Assemblies are supposed to be formed by synaptic plasticity. A pair of simultaneously stimulated cells establishes or strengthens its synaptic connection (in case there is a fiber bridging their distance). If this happens to many pairs of cells in a repeatedly activated pattern an assembly can be formed. Several detailed schemes for this process have been proposed and analyzed under the name of associative memory. Analysis has been made possible by simplifying assumptions (e.g. linearity, only one exchange of interactions, small overlap). It has been shown that many overlapping assemblies can be stored and retrieved in the same network without too much interference between them.

The lack of internal structure in assemblies leads to a serious difficulty of associative memories: each memory trace recalls a fixed subset of cells without possible variation apart from noise. However, cognitive psychology makes it obvious that realistic memory traces often correspond to a network of subpatterns connected in a flexible way to fit a particular situation.

2.3.5 Visual Perception, Perceptrons

Visual perception presents two outstanding problems, figure-ground discrimination and invariants extraction. The perceptron approach [Rosenblatt, 1961] to perception, which makes use of most of the ideas reviewed so far, demonstrates quite explicitely the inadequacies of those ideas to solve the two problems mentioned.

Perceptrons are meant to be models for sensory subsystems of the brain. A typical perceptron consists of threshold units (neurons) of three kinds, S, A and R, i.e. sensory, association and response units. These are arranged in layers which are sequentially connected: $S \to A \to R$. Cross-connections within a layer, or even backward connections may also exist. The A-units play the role of feature detectors. The $A \to R$ connections are modifiable by synaptic plasticity.

The prominent feature of a perceptron is its ability to reorganize itself in response to the repeated activation of a certain subset s of S-units such that subsequently a specific R-unit fires precisely when s is presented.

The invariance problem calls for a single R-unit to respond to the presentation of a pattern p in any position in S. Rosenblatt proposed to solve the problem by the introduction of a second layer A' of feature detectors, sandwiched between A and R: $S \to A \to A' \to R$. A unit a' in A' responds to the presentation of a certain feature in arbitrary position within S. Unit a' is enabled to do so by the presence of units a_i in A, $i = 1, \ldots, N_{a'}$, each of which responds to the particular feature in a position x_i in S. All units a_i have a connection to a', which fires if at least one cell of the set $\{a_i\}$ is activated. Many different feature detectors analogous to a' are present in A'. The pattern p will activate the same subset of A', independent of its position in S. A specific R-unit can now be trained to react to this subset. Activity in a unit of A' makes a statement about the presence of a particular subpattern (or feature) of p. In order to generalize over position in S information about the position of the subpattern is discarded. If the features are sufficiently complex it may be possible in principle to recover the relationships of overlap and reconstruct the full pattern p, in a way analogous to solving a jigsaw puzzle. This reconstruction, however, is nowhere done in a perceptron, and the recognition of p has to be done on the basis of the uncorrelated feature set represented by the active units in A'. This is only possible if the features represented in A' are of sufficiently high level, which means that they are very numerous, or specialized to a particular universe of patterns in S, or both. The machinery needed, particularly in A, is gigantic (as is demonstrated by a recent simulated version of a perceptron [Fukushima, 1980]). It is evident that an enormous improvement over the perceptron could be made with the help of a scheme by which the overlap conditions of subpatterns would be expressed.

An R-unit can supress all a' units not belonging to its own trigger set if appropriate inhibitory back-couplings $R \to A'$ are present. Rosenblatt proposed to solve the selective-attention problem in this way. He recognized, however, that this is no solution to the general figure-ground problem, since learning and recognition of a figure have to precede the suppression of its background. He admitted that new concepts were needed for the problem of figural unity [Rosenblatt, 1961, p.555]. Again, this calls for a scheme by which cells in A' could express their relatedness in terms of separate figures.

3 The Correlation Theory of Brain Function

3.1 Modifications to Conventional Theory

This section introduces i) a scheme for the interpretation of cellular signals which is a refinement of the one given in 2.1.2, and ii) a short-term analogue of synaptic plasticity.

3.1.1 Correlations between Cellular Signals

In paragraph 2.1.2 I discussed the experimental procedure by which the correlation of a cellular signal to an event is detected. The averaging in the peri-event-histogram method is important to get rid of an apparently random time structure within the cellular response. This time structure will now become important.

Consider the spike trains emitted by two cells in the central nervous system. These signals may be evaluated in terms of a *correlation* [1] . It is supposed to measure the similarity between the two signals and should at least discriminate between synchrony and asynchrony in their temporal fine structure (with a resolution of 2 or 5 msec). It has to be assumed that the trivial state in which all cells are globally correlated with each other is suppressed by a system of inhibitory connections which permits only a small fraction of all cells to be active at any one time.

[1]The term 'correlation' is not meant to imply a specific mathematical formulation.

3.1.2 Synaptic Modulation

The synaptic connection between brain cells i and j is characterized by a strength w_{ij}. It is a measure for the size of the PCT evoked in cell i upon arrival of a spike from cell j. I here postulate that the weight w_{ij} of an excitatory synapse depends on two variables with different time-scale of behaviour, a_{ij} and s_{ij}. The set $\{s_{ij}\}$ constitutes the permanent network structure. Its modification (synaptic plasticity) is slow and is the basis for long-term memory. The new dynamic variable a_{ij}, termed *state of activation* of synapse ij, changes on a fast time-scale (fractions of a second) in response to the correlation between the signals of cells i and j. With no signals in i and j, a_{ij} decays towards a resting state a_0, within times typical for short-term memory. With strong correlation between the signals the value a_{ij} changes such that w_{ij} increases *(activation).* With uncorrelated signals a_{ij} changes such that w_{ij} decreases to zero *(inactivation).* This behaviour of the variable a_{ij} will be referred to here as *synaptic modulation.* It can change the value of a_{ij} significantly within a fraction of a second. Not all synapses from a given cell to other cells can grow at the same time, since the inhibitory system referred to in 3.1.1 prevents those target cells from all firing simultaneously; also the synapses received by a cell compete with each other, for the same reason. The physical basis for synaptic modulation is not clear; it might correspond to the accumulation or depletion of some chemical substance at a strategic location in or near the synapse. The relevant postsynaptic signal is here taken to be the cell's output spike train, but it may also be a more local dendritic signal. As a simple example one could assume $w_{ij} = a_{ij}s_{ij}$ with $0 \leq a_{ij} \leq 1$ and a resting state a_0 within the interval $(0, 1)$.

3.1.3 Refined Plasticity

The variables $\{s_{ij}\}$ are controlled by what I shall call *refined synaptic plasticity:* strong correlation between the temporal fine structure in the signals of cells i and j causes s_{ij} to grow; this growth may be thought to be limited in the usual way (e.g. by sum-rules). Absence of correlation does not directly reduce s_{ij}.

The analogy between synaptic modulation and refined plasticity is apparent. Both are controlled by correlations in the signals of cell pairs in a positive feedback fashion. They differ in time-scale of decay (seconds for a_{ij}, decades to permanent for s_{ij}), and of build-up; and they differ in the way they are controlled. The a_{ij} react only to the two locally available signals and are both increased and decreased by correlations and their absence. The s_{ij} are only increased by local signals and are decreased in response to the growth of other synapses.

3.2 Elementary Discussion

3.2.1 Sources of Correlations

Correlations between the signals of cells can be caused by time structure in sensory signals exciting the cells. However, there is a more important source of correlations. Time structure in cellular signals can be created spontaneously, e.g. by a tendency of cells to form bursts of spikes. Correlations arise if this time structure is transmitted to other cells by excitation or inhibition.

3.2.2 Effects of Correlations

One effect of the correlation between signals in cells i and j was already mentioned: activation of the synaptic weight w_{ij}. Specific connection patterns (e.g.reflex arcs) can be created in this way, and a pluripotent network can be turned temporarily into a specialized machine.

Secondly, a correlation between the signals of cells i and j enables them to positively interact to excite a third cell k (if $w_{ki}, w_{kj} \neq 0$): the individual signals may not be able to transcend the threshold of cell k, whereas simultaneously arriving signals may. Two subnetworks with uncorrelated activity patterns may coexist without interfering.

Thirdly, correlations control (refined) synaptic plasticity. The absence of correlations between two activity objects, even if they sometimes coexist within the same span of a second, keeps them from developing mutual synapses.

3.2.3 Correlation Dynamics

The dynamical system introduced so far for the cellular signals and temporary synaptic strengths forms a basis for organization. The correlation between the signals in cells i and j and the states of activation a_{ij} and a_{ji} of their common synapses form a positive feedback loop, driving a_{ij} and a_{ji} away from the resting state a_0, and the signal pair away from a structureless uncorrelated state. In this way correlations can stabilize their own existence and cease to be transitory and shaky statistical epiphenomena. Different synapses on one cell compete with each other, as was pointed out above, and certain sets of synapses cooperate with each other. For instance, the direct pathway from cell i to cell j cooperates with the indirect pathway from i to a cell k to cell j. These dynamical interactions between synapses and corresponding signals tend to stabilize certain optimal connectivity patterns (together with their corresponding signal patterns). These can be characterized locally as having sparse connections (to avoid competition), which are arranged so as to have optimal local cooperation between them.

The slow component s_{ij} of synaptic strength is plastically modified only by strong correlations, i.e. mainly when connectivity forms an optimal pattern. Therefore the structure of the permanent network tends to be a superposition of optimal connectivity patterns. When input to the network activates certain cells (possibly in a correlated fashion), a dynamical process of organization sets in, as a result of which synapses forming an optimal network are fully activated and all other synapses are deactivated.

3.3 Network Structures

It is not clear how optimal connectivity patterns can be characterized globally. This chapter proceeds on the basis of the conjecture that they have a topological structure to them, the neighbourhoods of which may correspond to overlapping sets of co-active cells. (Mathematical rigour is not attempted in the notation.)

3.3.1 The Topological Network

Let S be a set of n cells, E an appropriate space of low dimensionality, m a map assigning to each cell of S a point in E, and p a natural number, $p \ll n$. The topologically structured network $\tilde{S} = (S, m, E, p)$ is constructed by connecting each cell of S with its p nearest neighbors in E by excitatory synapses. I will refer to $\tilde{S}$ simply as a *topological network.*

A topological network embedded in ordinary space is a very common idea (cf. e.g. [Beurle, 1955]). The point made here is that there is no need for a network to stay with this natural embedding. This has the important consequence that there is a huge number of topological networks on the same set S, even if E, p and the set of assigned points in E are kept fixed. Namely, instead of m one can consider Pm, with any permutation P of the assigned points in E. See fig. 1 for an example.

3.3.2 The Correlate of a Topological Network

Before one can deduce the dynamical behaviour of the network activity much more detail has to be specified. However, we are here only interested in certain aspects of the dynamics and a few assumptions suffice.

Let synaptic weights be constant for the moment. Since an activated excitatory synapse between two cells creates correlations in their signals, "neighbors" in a topological network are correlated. The topological aspect is important since on the one hand the topological network is cooperative: each synapse is helped by others in establishing a correlation (e.g. 1-3 and 1-2-3 in fig. 1a cooperate); on the other hand, the network can still be decomposed into many weakly coupled subnetworks, in contrast to globally cross-connected networks.

Two kinds of signal pattern can exist in a topological network. In one there are waves running through the network (see e.g. [Beurle, 1955], for an example see fig. 1). The diffuse network of inhibition keeps the cells from firing in global simultaneity. The other kind of pattern stresses the analogy to the spin-lattice of a ferromagnet [Little, 1974]: a cell randomly switches between an active and a silent state. In doing so it is influenced by its "neighbors". If a majority of neighbors is active, the cell will also be more likely to fire, if a majority of neighbors is silent, the cell will more likely be silent. The strength of coupling between the behaviour of the cell and its environment can be characterized by a parameter T, analogous to the temperature of the spin lattice. For $T = 0$ the coupling is rigid: all cells in the network switch up and down simultaneously. For infinite T there is no coupling and cells are independent. We are interested here in intermediate values of T, for which a cell is correlated with its neighbors and this correlation decreases over a characteristic "distance" which is a fraction of the "diameter" of the network. With either kind of cellular activity the structure of the network is expressed in the signals by correlations. Such a signal structure will be called a *correlate.*

Now allow the synaptic activities $\{a_{ij}\}$ to vary. Consider a set C of cells which are excited by input activity. Suppose C is part of several sets of cells $S', S'', \ldots$ which are internally connected by topological networks $\tilde{S}', \tilde{S}'', \ldots$. If these topologies are independent and all synapses are in the resting state, C is globally coupled in a non-topological fashion. The connectivity in C is then probably unstable. A stable state can be reached after one of the topological networks, say $\tilde{S}'$, has been activated and the others have been inactivated. In order for this to happen, the complement of C in S' has to be invaded, to fill the holes left by C in the topology of $\tilde{S}'$. After the network with the topology of $\tilde{S}'$ has been activated, activity can no longer invade the rest of the other sets $S'', \ldots$, because the p-environments of their cells, even if they are active in S', never fire synchronously.

Correlate reconstruction is the fundamental process of correlation theory. It must take place on the fast time scale of thought processes. Its synergetics is a complicated matter and needs further detailed work. An important special case is discussed in the next paragraph.

3.3.3 Projection between Topological Networks

Consider two structurally identical networks $\tilde{S}_1$ and $\tilde{S}_2$ on disjoint sets S_1 and S_2 of n cells each. The two sets are connected to each other by a one-to-one projection R of activated synapses connecting cells in corresponding position, so that R corresponds to an isomorphism. This defines on $S_1 \cup S_2$ again a topological structure which can carry a correlate, with correlations at short distance in $\tilde{S}_1$ and $\tilde{S}_2$, and between cells in S_1 and S_2 which correspond to each other according to R. This special kind of topological correlate can be approached from different starting configurations, as will be discussed now.

3.3.3.1 Consider first the case with R in the resting state and correlates corresponding to $\tilde{S}_1$ and $\tilde{S}_2$ active in S_1 and S_2 but not mutually correlated. R will have a weak synchronizing influence on pairs of corresponding cells in $\tilde{S}_1$ and $\tilde{S}_2$. The so induced correlations will activate the synapses of R, and strengthen the $S_1 - S_2$ correlations, until the stationary state is reached with fully activated R and the activity strongly correlated between S_1 and S_2. On the other hand, if on S_2 a network with a considerably different topological structure were activated, R would be deactivated.

The case is very reminiscent of the basic two-cells-one-synapse situation: correlation in (internal structure of) the correlates on $S1$ and S_2 leads to R-activation, lack of correlation to deactivation. In this sense S_1 and S_2 can be regarded as composite analoga to single cells.

3.3.3.2 Let $\bar{R}$ be a system of synapses connecting each cell of S_1 with each cell of S_2. Let $\tilde{S}_1$ and $\tilde{S}_2$ be isomorphic topological networks on S_1 and S_2. The synapses of $\bar{R}$ initially are in their resting state. A very similar system, referring to an ontogenetic problem, was simulated in [Willshaw and von der Malsburg, 1976] with two-dimensional E, and was treated analytically for one-dimensional E in [Häussler and v.d. Malsburg, 1983]. There, it was shown that $\bar{R}$ can dynamically reduce to a one-to-one projection between the isomorphically corresponding cells in $\tilde{S}_1$ and $\tilde{S}_2$. The system is able to spontaneously break the symmetry between several possible projections.

3.3.3.3 Several topological networks $\tilde{S}_2$, $\tilde{S}_2'$, $\tilde{S}_2''$, ... may exist in S_2 (in addition to $\tilde{S}_1$ and $\bar{R}$). Before a topological correlate can be established on $S_1 \cup S_2$, several decisions have to be made: between $\tilde{S}_2$, $\tilde{S}_2'$, $\tilde{S}_2''$, ... and between possible one-to-one mappings corresponding to one of the structures on S_2. These decisions have to be made simultaneously. This is likely to cause chaos instead of a specific correlate. However, if symmetries between the various structures are slightly broken already in the inital state, an ordered stationary state may be reached securely, as is made likely by extrapolation from experience with a case similar to 3.3.3.2.

3.3.4 Composite Elements

In section 3.1 I have introduced the basic machinery of correlation theory in terms of cells, correlations of their signals, synapses and their modulation. The discussion of 3.3.3 has prepared the way to the use of a very similar language on a higher level. The idea consists in considering sets of topologically connected cells instead of single cells as network elements. The sets may then be termed *composite elements.* Likewise the ensemble of cellular signals of a set may be regarded as a *composite signal*, and the ensemble of fibres connecting two composite elements as a *composite connection.* The correlation between two cellular signals was defined in terms of synchrony and asynchrony between spike trains. Correlation between the signals of two composite elements has to be defined as a structural correspondence between the composite signals in terms of the composite connection between the elements. Each single synapse between two composite elements should be modulated by a globally evaluated correlation between the composite signals. This is made possible by the fact that a temporal correlation in the signals locally available to the synapse can only be established in the context of a global correlation between the elements, as was discussed in 3.3.3.1.

Composite elements can again form networks: S_i, $S_j, \ldots$, with composite connections R_{ij}. For a correlate between the composite elements to form it is necessary that the different composite connections be locally consistent with each other. Introduce an arbitrary but fixed numbering of cells in each element. A one-to-one projection R_{ij} is then equivalent to a permutation matrix P_{ij} in which each non-zero element corresponds to a synapse. In a triplett of elements S_i, S_j, S_k the permutation P_{ik} must be the same as $P_{ij}P_{jk}$ in order to be consistent. Stated differently, the composite permutation matrix corresponding to a closed chain of connections must be unity: $P_{ij}P_{jk}P_{ki} = 1$. (The condition can be relaxed for chains of elements which are longer than the correlation length. This opens the door to the whole complexity and richness of non-trivial fiber bundle or gauge field structures.) Also on this new level the dynamical interactions between signals and synapses stabilize certain preferred connectivity patterns and correlations, and again it may be conjectured that they have a topological structure.

In applications it may be necessary to introduce super-composite elements. Paragraph 3.6.4 will give an example. The elaboration of particular structures is, however, a complex dynamical and mathematical problem.

3.3.5 The Synergetic Control of Action

How can the dynamical behaviour of the brain's network structure be characterized globally? Suppose the state of the brain at time t could be understood as a superposition of structures, termed modes, with the following properties: A mode is a subnetwork of active cells and synapses which, if left to itself, would reproduce its form, possibly change its amplitude. (Decomposition into modes has been rigorously carried out in a neuronal system in [Häussler and v.d. Malsburg, 1983].) To predict the state of the brain at time $t + \Delta t$, decompose its state at t into modes, let each of them grow or decay for the interval Δt, and superpose the results again. With the help of a global control parameter it often can be achieved that only one or a few modes grow and all others decay. It is conceivable that such global control exists in the brain. If only one mode grows it soon dominates the state of the system. If several modes are related by a symmetry they grow or decay with the same speed. This is the reason why symmetry breaking, i.e. the selection of one of the related modes, is difficult to achieve.

The distinguishing feature which allows a mode to grow fast is maximal local self-amplification and optimal cooperation of locally converging dynamical influences, e.g. correlation between signals converging on one cell.

If growth of a mode is sufficiently slow there is time for the exchange of signals between all parts of the network. All locally available information is then integrated into the one global decision - growth or decay. After a mode has grown and established itself for some time, conditions may cease to be favourable for it, either because the mode has prepared the way for a successor mode which starts to compete successfully, or because the environment has changed, or simply because of some kind of fatigue. Thus the brain is ruled by a succession of modes. This view emphasizes the analogy to many other self-organizing systems [Haken, 1978], and would put the brain into sharp contrast to the computer and other man-made systems with detailed central control.

Memory may be thought of as the ability of the brain to change its network so as to improve the success of modes which were once active. In the extreme case an entire global mode which once dominated the brain's state for a short moment can be reestablished. A physical basis for this ability is synaptic plasticity, which reinforces those networks which are strongly activated.

3.4 Applications of Correlation Theory

3.4.1 Visual Elements

Light stimulation of one retinal point can directly affect several thousand neurons in visual cortex. Together they form a composite element of lowest level, a *primary visual element*. Each neuron is specifically sensitive to a particular combination of quality values characterizing the stimulus: level of brightness or darkness, spectral distribution, spatial distribution of light within a receptive field, stereo depth, direction and speed of movement. Visual cortex contains multiple representations of the retina. These are interconnected by diffusely retinotopic fiber projections. Primary visual elements may be composed of cells in several visual areae and even in thalamus. The part of the brain formed by primary visual elements will here be termed V.

Consider a particular visual element while the eyes are slowly scanning over a scene. When a light edge crosses the receptive field of the element, a subset of cells is activated simultaneously. The subset describes the quality of the edge of light. This simultaneous excitation triggers activation of synapses and formation of a correlate within the active subset of the element under consideration. A subnetwork results which now represents a composite feature detector. Its signal expresses a composite quality which can be recognized even from mixtures of signals from different visual elements. Confusion is excluded by signal correlations within a set of fibres coming from one primary visual element.

Visual elements have been introduced here as those collections of cells which are affected from one retinal point. One could possibly also consider somewhat larger patches of cortex (and thalamus) as elements. Those larger elements would then be capable of forming correlates corresponding to patches of visual texture. There is no need for the brain's "hardware" to contain complex feature detector cells. Only cells responding to rather simple stimuli are required, from which complex composite feature detectors can be "manufactured on the spot" by activation of synaptic networks.

3.4.2 Figure-Ground Discrimination

Suppose all visual elements in the primary region V are integrated by a fiber system which connects feature sensitive cells in one element with cells specific for the same local quality in many other elements. Suppose two elements so interconnected are stimulated by a similar composite quality and correlates have formed in both of them, so that the situation described in 3.3.3.1 is given. In due course the connection between the elements will be activated and the composite signals of the elements will correlate with each other. On the other hand, if the two elements were stimulated by radically different composite qualities, mutual synapses would be deactivated and the signals would decouple.

Suppose a visual scene contains a region F characterized by local qualities which change continuously from point to point inside F and which change discontinuously across the boundary of F. (A prominent role among these qualities will be played by differential velocity caused by perspective movement or object displacement.) The mechanism just described will set up a network of activated synapses and a correlate which fills the region of primary visual cortex excited by the image of F. All elements taking part in it will signal this fact by mutual local correlations. There will be no correlations across the boundary of the network.

In this way the scene is decomposed into a patchwork of figures. Moreover, a figure is decomposed into a hierarchy of parts, the strongest correlations signalling affiliation to one part of the figure, weaker ones affiliation to adjacent parts, and so on. This decomposition of the visual scene into a hierarchy of correlates starts already prior to recognition of patterns, a stage of the process which was termed "preattentive vision" by B. Julesz [1981].

3.4.3 Invariant Image Representation

The correlation structure described in the last paragraph has to be built up anew for each image fixation. Another part of the visual system, to which I will refer as I, can accumulate visual information over longer periods of time and independently of retinal image location. A physical prerequisite for this is a fiber system which connects each element of V with each element of I. (This strong assumption can later be relaxed considerably.) If all these fibers were activated at the same time a great jumble of signals would converge on the elements in I. It is, however, possible to deactivate most connections and activate only topologically ordered one-to-one projections between V and I.

I assume that the elements in V and in I are tied together by topological networks N_V and N_I, respectively. (This is a statement about permanent weights.) The topology is the natural one of two-dimensional visual space. Consider for simplicity a situation in V with just two active correlates F and G. F refers to figure and G to ground. Correlations in both F and G are topologically structured by activated subnetworks of N_V. The components of N_V connecting F

with G are deactivated. Initially there may only be spontaneous activity in I, the correlations of which are topologically structured by N_I. Connections from F and G which converge on one element of I carry noncorrelated signals and cannot cooperate to cause excitation, correlation or synaptic activation. If one considers I and just the F-part of V as two super-elements, the situation is that of 3.3.3.2. As was pointed out there, a stationary state will be reached in which a one-to-one projection is activated which connects neighboring elements in F to neighboring elements in I. If symmetries are not broken by other influences, the scale and orientation of the $F \to I$ projection will be such that the image of F fits best into I. At the same time the correlate structure of the intra- and inter-element networks in F is transferred to the corresponding elements in I. (This is analogous to the transfer of retinal markers to the tectum in [v.d. Malsburg and Willshaw, 1977].)

The simulations of [Willshaw and von der Malsburg, 1979] have shown that the simultaneous presence of two independent correlates, like F and G, can lead to a final state with two independent mappings of the kind described, one for F and one for G. The network I can then tune its correlate to F or G.

New mappings between V and I have to be set up for each new image fixation. This is enormously facilitated by relevant structure in I built up during previous fixations. Relative image distortions between fixations are digested by distortions in the projections which are established. Over the course of many fixations more and more information about a figure, although arriving through different parts of the retinae, can be gradually accumulated in I.

After a mapping between V and I has been activated, information can be transferred from I back to V. The afferent information can thus be scrutinized by the retrograde activation of composite feature correlates.

In distinction to the perceptron approach to the invariance problem, the geometrical structure of the figure is explicitly represented in I. There is no need to recover it from the distribution of active feature detectors (cf. 2.3.5).

3.4.4 Interpretation of an Image

Before an image can be recognized it must be brought into contact with ideas of shapes and objects stored in memory. Let us invoke a part M of the brain. To a neurophysiologist M would appear similar to I. However, it would be dominated by specific connection patterns which have been layed down previously by synaptic plasticity and which correspond to abstract schemata of objects. These can be revived by resonance with structures in I to carry correlates. Recognition between structures in I and in M is possible on the basis of a correspondence of detailed network structure, which in turn is expressed in terms of correlations in signals. The situation was discussed in 3.3.3.3. Several relevant memory traces may be activated simultaneously or consecutively.

The representation of an object in I has to be fairly insensitive to image size, position, orientation and (slight) distortion. It therefore lacks information about these parameters and it is necessary for structures such as M to have access to the primary image in V. This is possible with the help of full direct fiber systems connecting M with V. These can be easily structured during a fixation because all elements of an image in V are functionally labelled by correlations with the corresponding elements in I. The original image can be scrutinized by the selective set-up of part-networks referring to parts of it. A full interpretation of an image is constituted by a correlate in a super-network composed of many super-elements such as V, I and M, partly belonging to other modalities of the brain.

Memory in its direct form precisely reestablishes correlates which were previously active. The observed great flexibility of memory traces could be explained if memory in this extreme form were restricted to certain substructures of the brain, like the M mentioned above. For instance, we know that the memory trace corresponding to a human face leaves unspecified all accidental aspects, e.g. perspective, illumination and expression. The trace has to be complemented by particular correlates in other areae, like V and I, before it can be compared with a real image. This flexibility cannot be accounted for with cell assemblies which cannot be analyzed into parts.

4 Discussion

4.1 The Text Analogy

The relationship between correlation theory and conventional brain theory may be clarified with the analogy to the way our cultural world makes use of symbols. When we write text we employ a set of basic symbols, letters or ideograms. Out of these we form higher symbols, words, sentences, paragraphs. We do so by producing many copies of the basic symbols and arranging these in spatial patterns. According to localization theory there is a fixed set of basic symbols with fixed locations in the brain. According to conventional theory higher level symbols are formed by the simultaneous activity of many of these. This is analogous to summarizing a paragraph with the help of letter statistics. According to conventional synaptic plasticity experience is accumulated in a way analogous to a measurement of the probability with which letters appear simultaneously in large pieces of text (large enough to represent the amount of information which we hold momentarily in our mind). Conventional theory tries to mend the obvious deficiency by introducing more and more letters and ideograms. This, however, creates more problems than it solves.

In correlation theory higher symbols are constructed out of lower ones with the help of correlations and modulating synapses. The full hierarchy of patterns is thereby represented. The refined plasticity of correlation theory is analogous to measurement of the probabilities of letters or words to be adjacent. Dynamical selection of synergetic networks is analogous to a grammar which allows only strings of letters with a certain structure to be formed. Correlation theory thus allows for much more complete information to be stored, and used for the reconstruction of previously active patterns, than does conventional theory.

Another point may be clarified with the text-analogy. Microscopic localization theory (2.1.2) attributes to each cell its own meaning. Also a letter or ideogram has its own meaning. However, if a symbol is placed in a certain context, its meaning is enormously refined or even completely changed. The crude meaning attributed to a cell by the peri-event-histogram of neurophysiology can, according to correlation theory, become refined; e.g. a primary visual cell may, in a particular moment, signify the red component in the image of the planet Mars in the night sky, not as interpreted by the proverbial little green man sitting in our brain, but due to the dynamical integration of the cell into a complex network.

4.2 The Bandwidth Problem

Important elementary thought processes take place within 200 to 400 msec. This sets an upper limit to the interval in which correlations can be measured. A lower limit to the time resolution with which signals can be observed is set to several milliseconds by differences in transmission delays. Since in addition noise must be tolerated, a bandwidth of 100 Hz for the cellular signal seems to be an optimistic estimate. With this bandwidth not too many signals can be distinguished in an observation time shorter than 200 msec. On the other hand, the discussion in section 3.4 has demonstrated how important it is that the activity patterns express highly differentiated structures in terms of signal correlations. It is, however, not necessary that all this structure be expressed in parallel within short time intervals (of, say, 200 msec) by stationary correlations. For instance, two cells may have significantly anticorrelated signals for a short period. All synaptic pathways between them and to common target cells will then be inactivated. If the inactivated state of synapses is rather insensitive to uncorrelated signals, there is then no need for continued expression of the anticorrelation in the signals of the two cells. In short, the effects of earlier correlations are stored in short-term memory. From an undifferentiated initial state in which every part is cross-coupled with every other a highly differentiated state can thus be reached in a sequential manner. In each step some parts of the network are decoupled from each other so that later no confusion between their signals can arise. The price to be paid is the time necessary for this sequential process and the path-dependence of the final state.

Synaptic plasticity can be regarded as a means to store past signal correlations on a more permanent basis, so that the signals are freed from the necessity to make certain distinctions. The time required to reach certain final states is thereby reduced. This increased efficiency is partly paid for by a restriction in generality. Composite elements have more complicated signals than single cells and can express correlation structures much more efficiently. They are enabled to do so by their special connectivity patterns. In an extreme case a nervous system (or subsystem) is completely specialized by its permanent network structure and no further synaptic modulation is necessary for the completion of its tasks. This is the case studied by what was called here conventional brain theory.

4.3 Facing Experiment

There is an enormous body of empirical knowledge about the brain, gathered in such diverse fields as clinical observation, anatomy, neurophysiology, psychophysics and cognitive psychology. Correlation theory must be judged mainly as an attempt at integrating the heterogeneous perspectives offered by the various methods into one coherent picture.

Localization theory, although strongly supported by experiment, created unrest because it posed the nervous integration problem, which has not been solved by conventional theory. In this situation many scientists have resigned from inquiring into the brain's function or have contented themselves with philosophical answers. Correlation theory tries to make sense of localization theory and proposes a solution to the nervous integration problem.

Neurophysiology tells us that cellular signals fluctuate. Since this temporal fine structure is not locked to the stimulus it is often taken as a sign of unreliability. According to correlation theory such rapid modulation of the signal is essential for brain function.

According to cognitive psychology and common observation there is a short-term memory with a life-time of many seconds. Cellular signals have a temporal correlation length considerably shorter than 100 msec. Synaptic plasticity on the other hand is too slow. Synaptic modulation introduces a dynamical variable as a basis for short-term memory which could have the correct characteristic time constant.

The most convincing support for the theory will have to be found on the microscopic level: demonstration of organized correlations, synaptic modulation, and synaptic plasticity controlled by precise correlations.

The theory requires validation of its claims also on another, the functional, level. Elementary processes can be simulated on computers and analyzed mathematically. More complex processes will have to be demonstrated with the help of specialized machines.

4.4 Conclusion

The brain and its function present us with a tangle of interrelated problems none of which can be solved, or even be precisely formulated, on its own. In this situation a concept of global brain organisation is needed which partitions the integral problem into defined technical questions. Correlation theory may be regarded as such a global scheme.

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Figure Caption

Figure 1. Two topological networks on the same set S of 12 cells. E is the two-dimensional plane, $p = 4$, and the mapping m assigns the cells in natural order to equidistant points on a circle. **a)** shows the resulting topological network $\tilde{S}_1 = (S, m, E, p)$. **b)** shows $\tilde{S}_1$, however with a permutation P applied to cell positions. In **c)** the second topological network $\tilde{S}_2 = (S, Pm, E, p)$ is added. **d)** and **e)** show cellular activity (correlates) in the form of travelling waves as shaped by $\tilde{S}_1$ (left side of graphs), or $\tilde{S}_2$ (right side). Line segments symbolize short bursts of activity. In d) the order of cells on the ordinate is as in a), in e) the order is as in b) and c). Each correlate activates its own network and deactivates the other. Simulations of cell activity and synaptic modulations have shown that $\tilde{S}_1$ or $\tilde{S}_2$ can be dynamically recovered from the superposition, c), (v.d. Malsburg and Häussler).