The Theory of Embedding Fields with Applications to Psychology and Neurophysiology (I)

by

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PREFACE

The theoretical developments in this monograph were originally motivated by a study of standard experimental effects in human learning situations. These effects have received extensive experimental consideration by many authors over at least a forty year period. Once the original theoretical structure was completed, it imposed natural theoretical extensions that generated familiar experimental effects from a number of other fields of experimental inquiry in psychology, neurophysiology, and neuroanatomy. Each of these further fields is receiving and has received intensive experimental consideration as well.

The enormous literature of the experimental contributions relevant to the theoretical structure of the paper has made the author's task of accurately expressing appreciation for the stimulation of others' contributions a formidable one whose completion, were it possible, would have itself substantially increased the length of the monograph. The task is further complicated by the fact that the work has proceeded by constructing a dynamical system directly from a concentrated body of stable data and then passing in a purely theoretical way to a much larger frame to independently derive results which later were either found to have been conjectured or found experimentally by others, or are new.

Since the purpose of the monograph is to present a unifying conceptual frame which is not tied to the work of any single group of contributors, experimental effects are presented as manifestations of deeper dynamical principles. Since each of these effects, when known, has often been studied by many authors, I have found it necessary to assume that it is well known and mention a particular author's name only when he is inextricably tied to the effect. Extensive bibliographies of references are available in the broad literature of periodical reviews of experimental progress, and will hopefully offset the necessary bibliographical omissions.

My debt to others, whether direct or indirect, is great nonetheless, for a theoretical monograph of this kind would have been quite inconceivable were it not preceded by a long period of vigorous and intelligent experimentation.

Section Titles

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1. Introduction

Much of the mathematical formalism of contemporary psychological theory, for example stimulus sampling theory, consists of a collection of probability formulae which are derived from arguments concerning certain simple operations on abstract point sets. Such a theoretical structure allows one to express several intuitive conceptions of psychological processes in a manner that maintains a close rapport, in principle, with those aspects of collected data that fall within the relatively narrow confines of the theoretical postulates. These uneven virtues have brought gratifying initial success in predicting a few interesting characteristics of data in several simple experimental situations.

At least three related limitations must, however, be observed in the foundations of the theory that transcend any particular predictive criterion. First, the sets over which the theory's formulas lie are merely formal and without any apparent relation to psychologically important structures in the real world. Second, the probability statements that are found in the theory may be formulated in such a way that all of their variables take values exclusively in data sheets giving counts of evoked stimuli and responses. The formal structure of the theory is hereby inextricably tied to simple daily evaluations of the external guises of learning processes and provides little insight into the structure of these processes. Although this simplicity of attack makes possible the proliferation of models to fit a limited number of classes of learning data, it would appear to have also left the impression that when a given model does not serve adequately in a fixed empirical setting, the most profitable goal for

the theorist should be to find another model whose foundations stem quite directly from those of those of the first and whose slightly altered equations and thoughtfully chosen parameters render a closer fit to data more feasible. This impression has persisted in spite of convincing evidence that very slight generalizations of theoretical postulates generate highly nonintuitive formulas of considerable complexity, and that the theory provides no principles by which to extend the simplest formulas to really interesting cases - a third and imposing difficulty.

The present paper entirely abandons this rationale of theory construction and searches for new theoretical foundations in the belief that the extant theories really do not grasp the underlying significance of complicated psychological data in a simple and coherent way. The outcome of this search is a new mathematical structure which overcomes many of these difficulties. Fundamental to the motivation of the new theory is the realization that the dynamics of many psychological problems may be viewed from a unified point of view once the geometrical substructures that characterize each separate problem are elaborated and distinguished. The interrelations between these divers geometrical substructures and their similar dynamical superstructures on the psychological level can be gradually extended and refined until the sense in which they are averages over the fine geometrical and dynamical structure of living brains becomes clear. This passage from psychological to neurological structures is the passage from the macroscopic variables to the microscopic variables needed to understand neural interactions. It is natural to begin our study with macroscopic variables since behavioral interactions proceed on a macroscopic level. Correspondingly, many neurological properties of higher brains are of a beautifully sculpted global character which cannot easily be viewed in their functionally unitary form by contemporary microscopic neurological techniques. The process of passing from macroscopic to microscopic variables shall establish a connection between the psychological and neurological approaches which shall help to illuminate the results of each approach and, hopefully, to unify the course of future studies.

The present paper shall begin by studying in a leisurely and qualitative manner a particular type of psychological data, namely from verbal learning.

Little effort shall be made to give the most concise presentation, since the theoretical ideas are new and one cannot fully understand the value of a new theoretical tool before seeing how smoothly it applies to many interesting examples. On the other hand, since the data in verbal learning is so bountiful and is usually well-replicated, it is impractical to give bibliographies after every theoretical analysis. Rather, it shall be tacitly assumed that every example which we consider represents a central empirical finding unless otherwise stated.

In order to initiate our theoretical study, it shall be necessary to perform an inductive leap to a mathematical system which is, in a natural sense, the simplest system that provides nontrivial insight into the data at large. Such a leap is never particularly easy to motivate at first since it originally arises as a direct perception of the underlying structure of the data. It shall therefore be initially justified by showing that the mathematical object which arises actually gives a qualitative description of a large quantity of data from a small number of principles. After this has been accomplished, some simple intuitive questions and an obvious formal difficulty in the original mathematical system shall impose a natural extension of its structure. Once this extension is before us, a very broad collection of verbal learning data, including several language phenomena of higher type, shall immediately be seen to be within the grasp of the theory. But even more shall be gained in this step, for through it the significance of the dynamical system under study as a representation of global neurological events shall slowly emerge. Indeed, the paper shall proceed from this point to successively more refined representations of neurodynamics and neuroanatomy by extending the original mathematical system in a rational way. The outcome is an explanation of many phenomena in general learning theory, psychophysics, perception, and the more properly neurological sciences. Along the way, some light will be thrown on such topics as the uses and limitations of information theory in psychology, spectral analytic properties of neurological systems, the specific inadequacies of the computer analogy of neural processing, and the weaknesses

of stimulus sampling theory which prevented its generalization to a broad variety of experimental situations. The controversies between Contiguity and Gestalt learning theorists, and between "all-or-none" and "continuous" learning theorists will also be qualitatively resolved, and the work of such people as Hull and Guthrie will be understood from a higher vantage point. Among the neurological facts to be discussed are the EEG rhythms and their significance, thalamo-cortical interactions, cortico-cerebellar interactions, cortico-cortical interactions, lateral inhibitory processes, pre- and post-synaptic habituation, spatial and temporal masking, chemical transmitters and coenzymes, the coupling of membrane polarity to cellular control macromolecules and their repression, neural Use and Disuse, axonal frequency modulation, neural "power laws", the dynamics of neuron pools, plasticity in the growth of neurons, various uses of neural thresholds, temporal and spatial summation effects, cortical conditioning, the cellular distributions in neocortex, cerebellum, and thalamic nuclei and their significance, the interaction of specific and nonspecific regions such as the reticular formation, glial-neuron interactions, the behavior of sequences of specific sensorimotor relay nuclei, and related anatomical and physiological matters. The conclusion of this development is the statement of a general theoretical program carried by a small collection of equations and principles which have a clear neurological interpretation, and which bring together many branches of the psychological and neurological sciences into a harmonious perspective.

2. A Free Embedding Field for Homogeneous Serial Verbal Learning

Thus let us begin with a special empirical situation whose more subtle complexities shall initially be ignored to achieve a tractable though idealized theoretical structure. In particular, suppose that we are given a classical serial rote learning situation with nonsense syllables as the verbal units. For simplicity it is desirable to assume that all units have equivalent theoretical status, so suppose that each syllable consists of a single consonant and that the consonants are matched along a variety of scales, say in associative value, familiarity, and the like. Further assume that the list is n consonants long, and that learning is by the serial anticipation method with visual presentation, followed by an immediate utterance of the stimulus item, and verbal recall. One may imagine that the subject sits before a window in which are sequentially presented the consonants of the list according to a predetermined cyclic plan. It is a matter of daily experience that such verbal units are intuitively perceived as unitary or simple; that is, one never seeks in actual discourse to decompose a given consonant into smaller verbal parts. Thus it is intuitively natural to let each consonant be a fundamental and indivisible mathematical entity. The

interpretation of such a representation shall soon be clearer but at present we merely associate in good faith to each consonant r_i an abstract point p_i, where the subscript indicates the order of presentation in the list. Since the empirical data of this problem in verbal learning is a series of transitions through time from one point (consonant) to another, suppose that the points p_i are the nodes of a completely connected directed graph; that is, from p_i to p_j draw the directed line l_{ij}, and continue this process for all ordered pairs of points (see diagram 1). This simple structure adequately serves to emphasize the principal first-order structural features of the process. It shall be called the structural carrier (of the embedding field) associated with this problem.

The structural carrier represents the geometry of the problem, but it does not in any way encompass the time dependent behavior so familiar in the use of language. In order to discuss this dynamical behavior, it is necessary to define a variety of time-dependent functions that are naturally associated with the points and lines of the structural carrier. Thus, with each p_i associate a positive constant M_i, which is called the total embedding space of p_i. Further associate an (as yet undetermined) function s_i, $0 \leq s_i \leq M_i$, the strength function of p_i. To every line l_{ij}, assign two positive real numbers A_{ij} and p_{ij}, where A_{ij} is the total embedding space of l_{ij} and p_{ij} is the structural connection number of l_{ij}. Similarly denote the as yet undetermined function c_{ij}, $0 \leq c_{ij} \leq A_{ij}$, as the strength function of l_{ij}, and the function $\tilde{c}_{ij} \geq 0$, ≤ 1 as the renormalized strength function of l_{ij}.

It is in terms of these functions that the dynamics of this situation shall first be described. The strength s_i of p_i shall be a quantity fluctuating in time over p_i. At every time t a certain function of $s_i(t)$ will be transmitted through the line l_{ij} and shall be received at the point p_j after a fixed transmission time lag t_{ij}. The effect of this transmitted

quantity from p_i upon the activity of p_j shall in turn depend upon the value of s_j at the time at which the quantity arrives at p_j. Moreover, while this process of strength transmission through the l_{ij} proceeds, it shall influence the growth of the various functions c_{ij}. The values of these functions c_{ij} at future times shall in turn modulate future strength transmissions between the various points, via the $\tilde{c}_{ij}$ functions.

More explicitly, suppose that M_j represents a structural quantity that can be exhausted and that $s_j(t)$ represents the extent to which it is exhausted at time t. The value of s_j at a given time may also be viewed as a kind of activity that pervades a structure whose total extent is M_j. In particular, when $s_j = M_j$, p_j is maximally active. Now assume that if the line l_{jk} were operating at its maximal capacity, a quantity rs_jp_{jk}, $0 < r = \text{constant} < 1$ would be transmitted through it at any time. Thus p_{jk} represents a measure of the transmission capacity of l_{jk} and r is a factor which translates point strength values into transmitted strength values. Assume further that when the transmitted strength reaches the endpoint of the line l_{jk} it crosses a structure which alters its value by the multiplicative factor $\tilde{c}_{jk}$. If the time taken to transmit strength values from p_j to p_k over l_{jk} is t_{jk}, then the strength transmitted from p_j to p_k and received by p_k at time t is $r(s_j(t - t_{jk}))\, p_{jk}\tilde{c}_{jk}$, where the notation $s_j(t - t_{jk})$ merely denotes the evaluation of s_j at time $t - t_{jk}$. In particular, assume that $t_{jj} << \min_{k=j} t_{jk}$, so that as a rough estimate the approximation $t_{jj} = 0$ is made in this case. This is a special assumption that does not hold in more general situations. Let us now further assume that the strength values received from the entire set of points at a fixed p_k combine additively. Then the total transmitted strength received at time t at p_k is $T_k = r \sum_{i=1}^{n} s_i(t - t_{ik})\, p_{ik}\tilde{c}_{ik}$.

We already observed that the effect of strength transmitted to p_k in not independent of the activity at p_k at its time of arrival. In fact,

we suppose that contributions of transmitted strength values will cause a growth in the time derivative of s_k that is proportional to the transmitted strength and the function $M_k - s_k$, which represents the extent to which the quantity M_k has not been exhausted at the time new transmitted strength arrives at p_k. It is for this reason that $E_k = \alpha(M_k - s_k)$, $0 < \alpha$, is called the <u>effective embedding space of</u> p_k. The choice $\alpha(M_k - s_k)$ for E_k is merely the simplest choice of an element in a more general class of effective embedding spaces which we shall encounter. In addition to the growth process hereby determined for s_k, a process of strength decay given by a function D_k is also envisaged to proceed locally at each p_k. The particular process here chosen is a simple exponential one. In mathematical form, the above rules may be written as:

$$\frac{ds_k}{dt} = E_k T_k - D_k, \tag{1}$$

where

$$E_k = \alpha(M_k - s_k),$$

$$T_k = r \sum_{i=1}^{n} (s_i(t - t_{ik}))p_{ik}\tilde{c}_{ik},$$

and

$$D_k = \beta s_k, \qquad \text{for } k = 1, 2, \ldots, n,$$

where α, $\beta > 0$, $0 < r < 1$, and we impose the condition (*):

$$\max_i \frac{\alpha r M_i p_{ii}}{\beta} < 1 \qquad (*).$$

That is, $\frac{ds_k}{dt} = \alpha^+(M_k - s_k)(\sum_{i=1}^{n} s_i(t - t_{ik})\, p_{ik}\tilde{c}_{ik}) - \alpha^- s_k$, where $\alpha^+ = \alpha r$ and $\alpha^- = \beta$ are the growth and decay constants of the equation.

To determine the behavior of the functions c_{ij}, suppose that c_{ij} represents a process that takes place at the end of the line l_{ij}. Call this terminal region the node N_{ij} of l_{ij}. Suppose that the dynamical behavior of N_{ij} is locally determined by the strength quantities which impinge on it from both p_i and p_j. In the present case, the only difference envisaged between p_i and p_j in determining this behavior is that the quantities representing the activity of p_i take longer to reach the vicinity of N_{ij} than those representing the activity of p_j. With this simple distinction between p_i and p_j in mind, the behavior of c_{ij}, too, may be viewed as the net effect of growth and decay processes over a localized geometrical structure. Indeed A_{ij} is, akin to M_k, an exhaustible structural quantity belonging to the node N_{ij}, while c_{ij}, akin to s_k, represents the total extent of an activation process that modulates the strength transmission from p_i to p_j via N_{ij} through time. A_{ij} is simply the maximum value which this process can attain. p_i transmits $r\, s_i(t - t_{ij})\, p_{ij}$ to N_{ij}, and N_{ij} immediately transfers $r\, s_i(t - t_{ij}) \cdot p_{ij} \widetilde{c}_{ij}$ to p_j, where $\widetilde{c}_{ij}$ is a function of $\{c_{ik}\}$.

To determine c_{ij}, suppose at any time t that the time derivative of c_{ij} is proportional to the products of the point strength values transmitted from p_i and p_j and reaching N_{ij} at time t. By strength "transmitted" from p_j to N_{ij} we mean the contiguous relation of N_{ij} to p_j, which permits the assumption that s_j acts directly upon N_{ij}, up to a multiplicative factor. Notice that since the strength transmitted from p_i to N_{ij} is computed before it crosses N_{ij}, the factor $\widetilde{c}_{ij}$ does not appear. Indeed, once N_{ij} is crossed, the transmitted value loses its identity as a product of the strength activity at p_i and becomes expressible totally in terms of s_j. The growth of the line connection function c_{ij} is hereby determined by the simultaneous activity of p_i and p_j, where simultaneity is measured relative to the time of arrival of transmitted values at N_{ij}, and the activity of the relevant points is reflected entirely by the strength values that they transmit to N_{ij}. Moreover, again in analogy with s_k, the growth rate of the line connection c_{ij} is influenced by its

present state. In fact, define the quantity $E_{ij} = u(A_{ij} - c_{ij})$, $0<u$, in close analogy with E_k, and call E_{ij} the effective embedding space of N_{ij} (or, of l_{ij}). The growth process for N_{ij} is completely determined by requiring that the rate of growth of c_{ij} at time t be proportional to the product of the transmitted strength values received from p_i and p_j at time t, multiplied by the effective embedding space of N_{ij} at time t. To determine the decay process for lines, suppose that c_{ij}, which measures the activated portion of A_{ij}, decays at a rate D_{ij} proportional to the product of its value at time t with the values of the effective embedding spaces (the inactive regions) of p_i and p_j that impinge on N_{ij} at time t. Notice that the effective embedding space of p_i that impinges upon N_{ij} at time t is not proportional to $M_i - s_i$, but is rather proportional to $M_i - s_i(t - t_{ij})$; that is, one can think of a parallel transplantation of the inactive region of p_i at time s along the line l_{ij} until it reaches N_{ij} at time $s+t_{ij}$. This decay law can be heuristically summarized by saying that the inactive region of the effectively connected line structure of l_{ij} decays exponentially into its disconnected state. Or, still heuristically, lines which are not activated fall gradually into desuetude. These laws for the line structure have the following mathematical formulation:

$$\frac{dc_{ij}}{dt} = E_{ij}\,\hat{T}_{ij}\,\hat{\hat{T}}_{ij} - D_{ij}\,\hat{E}_{ij}\,\hat{\hat{E}}_{ij} \tag{2}$$

where

$$E_{ij} = u(A_{ij} - c_{ij}),$$

$$\hat{T}_{ij} = r\,s_i(t - t_{ij})\,p_{ij}, \quad \text{so that} \quad T_{ij} = \hat{T}_{ij}\,\tilde{c}_{ij},$$

$$\hat{\hat{T}}_{ij} = \hat{r}\,s_j,$$

$$D_{ij} = \epsilon c_{ij},$$

$$\hat{E}_{ij} = \rho(M_i - s_i(t - t_{ij}))\,p_{ij},$$

$$\hat{\hat{E}}_{ij} = \hat{\rho}(M_j - s_j),$$

and $u,\ r,\ r,\ \epsilon,\ \rho,\ \hat{\rho} > 0$. That is,

$$\frac{dc_{ij}}{dt} = \gamma^{+}_{ij}(A_{ij}-c_{ij})s_i(t-t_{ij})s_j - \gamma^{-}_{ij}c_{ij}(M_i-s_i(t-t_{ij}))(M_j-s_j)$$

where $\gamma^{+}_{ij} = u\,r\,\hat{r}\,p_{ij}$ and $\gamma^{-}_{ij} = \epsilon\rho\hat{\rho}\,p_{ij}$ are the growth and decay constants of the equation. We also assume that the growth equation for the line functions is more slowly varying than that for the strength functions.

3. Experimental Inputs.

The collection of equations (1) and (2) together constitute the free embedding field equations for this problem (diagram 2). A free embedding field is thus a pair consisting of a structural carrier and a set of functions of the above kind. Such a field is unperturbed by external influences and, as one sees from condition (*), increasingly small strength quantities are transmitted from point to point while the c_{ij} functions gradually decay. In fact, $\lim_{t\to\infty} s_i(t) = \lim_{t\to\infty} c_{jk}(t) = 0$ for all i, j, and k. The free case is hardly ever realized during an active learning session, but it illustrates a pervasive tendency in embedding fields to achieve an undifferentiated and quiescent state. In realistic cases, two general categories of external disturbances influence the field behavior. The first of these is the category of experimental inputs $I^{(e)}_i = I^{(e)}_i(t)$, $i = 1, 2, \ldots n$, which correspond, for example to the presentation to an attentive learning subject of a stimulus item r_i that has a point representation p_i in the embedding field. The second category is that of the internal inputs $I^{(in)}_i = I^{(in)}_i(t,\ s_1, \ldots,\ s_n,\ c_{11},\ c_{12}, \ldots,\ c_{km}, \ldots)$, $i = 1, 2, \ldots n$, which correspond to dynamical interactions between the given embedding field and other field structures which together represent the total on-going psychological behavior of the individual. The internal inputs are sometimes called feedback inputs for this reason. We assume that these two kinds of input influence their respective points in a manner formally identical to that of

the transmitted strength factors; namely,

$$\frac{ds_i}{dt} = E_i (T_i + I_i^{(e)} + I_i^{(in)}) - D_i .$$

In this sense, the quantities T_i, $I_i^{(e)}$, and $I_i^{(in)}$ all behave as external influences on local point activity. In order to geometrically represent these additional features of the field, the structural carrier is enlarged by the addition to each point of a pair of directed lines which terminate at that point, but whose initial points are as yet undetermined (diagram 3).

The functions $I_i^{(in)}$ shall temporarily be ignored. They are quite complicated. The functions $I_i^{(e)}$ are, however, amenable to an elementary treatment, which will be gradually extended as we proceed. Thus suppose that an experimental determination of the times at which verbal units will be presented to a subject is made. Let s_{ij} be the time at which the j^{th} presentation of the i^{th} item is made. Further let

$$J_i^{(e)}(t) = \begin{cases} 0, & t < 0 \\ a_i^{(e)} t \cdot \exp(-b_i^{(e)} t), & 0 < t \leq T^{(e)} \\ 0, & T^{(e)} < t \end{cases}$$

where $a_i^{(e)}$, $b_i^{(e)} > 0$ are fixed constants, and require that

$$I_i^{(e)}(t) = \Sigma_j J_i^{(e)}(t - s_{ij}).$$

Each $J_i^{(e)}(t - s_{ij})$ is called an experimental input with onset time s_{ij}. Every experimental presentation of verbal unit r_i to which the subject attends is conceptualized as an input to p_i of a fixed functional form whose onset time is the time of presentation, and successive inputs to p_i

combine additively. Actually, the onset time of experimental inputs to the embedding field lags behind the experimental stimulus onset by a very brief time interval. We assume this interval to be the same for all points and to be invariant under successive experimental manipulations, in keeping with the simplifying law that individual experimental inputs combine additively. The entire onset time sequence is thus translated by an inessential small constant. In fact, the functions representing successive experimental inputs to a fixed point are not strictly equal to the translates in time of a single function $J_i^{(e)}$; nor do the various experimental inputs strictly add. We can, however, safely ignore these matters in our present elementary discussion since the distribution of onset times is highly regular in the classical experimental paradigms and is large relative to a simple reaction time.

4. $\tilde{c}_{ki} = ?$

A determination of $\tilde{c}_{ki}$ is now possible. The simplest such choice is obviously $\tilde{c}_{ki} = c_{ki}$, and corresponds to the supposition that the effects of the transmission process through any fixed node are independent of the comparable process at any other node. Such a supposition is not entirely correct, however. In particular, for this choice of $\tilde{c}_{ik}$ no nontrivial list could ever be learned perfectly. Since a considerable amount of discussion must be given before the actual situation can be adequately revealed, we shall for the present make a somewhat artificial, but nonetheless highly illuminating, choice of $\tilde{c}_{ki}$ in a special case. Thus, let $M_i = 1$, and $A_{ii} = B$ for all i, while $A_{ij} = A$, $i \neq j$; that is, $A_{ij} = B(1 - \delta_{ij}) + A\delta_{ij}$, where δ_{ij} is the Kronecker delta. Further let $t_{ij} = v(1 - \delta_{ij})$, $0 < v$, and let $p_{ij} = \lambda(1 - \delta_{ij}) + \mu\delta_{ij}$, $0 < \mu, \lambda \leq 1$. For this situation, the choice $\tilde{c}_{ki} = c^*_{ki}$, where $c^*_{ki} = c_{ki} / \Sigma_r c_{kr}$, is in fact a closer approximation to actual events than c_{ki}. The significance of the choice c^*_{ki} is, for example when $\lambda = \mu = 1$, that the total strength transmitted from p_k is given by $\Gamma_k = r\Sigma_i s_k p_{ki} \tilde{c}_{ki} = rs_k$. The line structure determines a

continually varying partitioning of the transmittable strength at a point to the other points in the embedding field whenever the c values leading from a given point are normalized. Let this choice of $\tilde{c}_{ki}$ be assumed unless the contrary is stated in the following qualitative discussion of several of the psychological underpinnings of the embedding field equations.

5. An Example of Directionality of Associations

Suppose for simplicity that the embedding field contains only four points: $p_1, \ldots, p_4$, and that the above special uniformity assumptions are in force; that is, $M_i = 1$, $A_{ij} = B(1-\delta_{ij}) + A\delta_{ij}$, and so on. Assume further that $\mu = 0$ and $\lambda = 1$, or $p_{ij} = 1-\delta_{ij}$. Choose all initial values for the c_{ij}, $i \neq j$, to be equal and small, and suppose that $c_{ii}(0) = 0$ for all i. Also let all $s_i(0) = 0$. We are hereby given a highly homogeneous and quiescent four point field, free from self-excitations. Now deliver an external input to point p_1. p_1 then transmits equal measures of strength to p_2, p_3, and p_4, whence the rate of growth of each of the c_{1j}, $j = 2, 3, 4$, will be the same, by symmetry, and the path strengths c^*_{1j}, $j = 2, 3, 4$, will remain identically constant. Moreover, by $rp_{1j} < 1$, s_1 will be larger than $s_2 = s_3 = s_4$ during a time interval following input onset to p_1 after which all s_i are small. Deliver an input to p_2 during this interval. Following this delivery, the path strength c^*_{12} will grow to the equal detriment of c^*_{13} and c^*_{14}, since $s_3 = s_4 < s_2$. Similarly, the path strength c^*_{21} will grow significantly more than c^*_{23} and c^*_{24} if the s_2 values, now augmented by an input, are transmitted to the p_i, $i = 1, 3, 4$, before s_1 decays to the $s_3 = s_4$ range of values. This distribution of values in the c^*_{2i} depends critically on the relative sizes of the transmission times $t_{ij} = v$, the interval Δ during which s_2 is large, and the interval Λ_{12} between the onset of the input to p_1 and the input onset to p_2. For example, if $\Delta << v$, then we can choose Λ_{12} in such a way that strength transmitted from p_2 to p_1 after the input onset at p_2 arrives at p_1 when s_1 is very small. Since the strength originally transmitted from p_1 to p_3 and p_4 remained within l_{13} and l_{14} for v time

units, Λ_{12} can also be adjusted so that the p_2 transmissions to p_3 and p_4 arrive before their strength functions have decayed too much. Consequently, c^*_{21} will decay to the equal advantage of c^*_{24} and c^*_{23}, which is the reverse inequality of that obtained above. In fact, however, $\Delta_{12} << v$ never occurs in these fields. If it did, the probability that transmitted excitation from some point arrives at another point while the latter is being excited by an external input would be very small, whence the field would remain practically homogeneous, and uninteresting, for all time. The inequality $c^*_{21} > c^*_{23} = c^*_{24}$ may therefore be safely assumed. A more precise understanding of the relative temporal magnitudes underlying it will come in the following pages.

Now let Q_{ij} be the time between the input onset at p_j and the first time that strength induced by the input to p_i reaches p_j. $Q_{12} = |\Lambda_{12} - v|$ while $Q_{21} = \Lambda_{12} + v$. Since $Q_{12} < Q_{21}$, it is usually true that $c^*_{21} < c^*_{12}$. Once $c^*_{21}(t_o) < c^*_{12}(t_o)$, the inequality will tend to preserve itself for times $t > t_o$, since transmittable strength from p_1 will be partitioned in greater quantities to p_2 than will transmittable strength from p_2 be partitioned towards p_1.

We therefore see that if a pair of inputs is delivered successively to p_1, then to p_2, in a homogeneous and quiescent four point field whose constants are adjusted to avoid trivialities, c^*_{12} and c^*_{21} will grow to the detriment of the line values of the other points in the field, with the advantage going to c^*_{12} (diagram 4). Suppose that two four point fields $\mathcal{F}_1$ and $\mathcal{F}_2$ are now given which are identical replicas of one another and have received the successive inputs to p_1 and p_2. Let t_o be the onset time of the input to p_2 in both fields. If at time $t_1 > t_o$, r_1 is presented to $\mathcal{F}_1$ again and c^*_{12} is still relatively large, an external input to p_1 will be delivered and the strength transmitted from p_1 will pass in greatest quantity to p_2, whence s_2 will grow larger than s_3 or s_4. Similarly, if at t_1 an input is delivered to the p_2 of $\mathcal{F}_2$ rather than to its p_1, s_1 will grow larger than s_3 or s_4 due to the favorable normalization of c^*_{21}. Nonetheless, the inequality $c^*_{12} > c^*_{21}$ implies that the maximum value of s_2 achieved in $\mathcal{F}_1$ due to transmission induced by the second input to p_1 will exceed the maximum of s_1 in $\mathcal{F}_2$ due to transmission induced by the second input to p_2. We now suppose that

increasing the value of s_k at any time enhances the probability that the subject will offer r_k as a response at that time. The previous argument then shows that in a homogeneous four point field, the presentation of r_1, then r_2, followed after a pause by r_1 will lead to the evokation of r_2 with high probability. Similarly, r_1, then r_2, followed after a pause by r_2, will yield r_1 with fairly high probability. But whenever the two pauses are of equal length, the probability of evoking r_2 in response to r_1 might well exceed the probability of evoking r_1 in response to r_2.

We now assume that the c^*_{ij} function plays the role usually ascribed to the ill-defined concept of the "strength of association" from r_i to r_j through time. We also subscribe to the following standard terminology: the temporally "forward" direction in a list is the direction in which new inputs arrive. In particular, p_{i+1} is more forward than p_i in this sense. The temporally "backward" direction is the reverse of the forward direction. Using this familiar terminology, it may be said that contiguous inputs to two points in a symmetrical four point field cause associations to be formed in both the temporally backward and temporally forward direction. The forward direction is however, usually preferred.

6. An Example of Symmetry of Associations.

Notice that although we have imagined a four point field, external inputs were only delivered to the two points p_1 and p_2. What happens if we strip away the points p_3 and p_4 to which inputs were never delivered? Had we originally been given a two point field with points $\{p_1, p_2\}$ and otherwise identical initial conditions, $c^*_{12} = c^*_{21} \equiv 1$ for all time. We can interpret this fact to mean that the evokation of r_2 follows the presentation of r_1 with certainty, and vice versa, which shows that the two point field maximizes the symmetry of forward and backward structuring possibilities among all possible embedding fields. To an observer who can detect only the presentation of external inputs and the evokation of overt responses, this comparison of two and four point fields must be a cause of considerable chagrin. For it shows that the presentation of an identical array of external inputs to two

different embedding fields can yield strikingly different results in the overt response paradigm. Since our imaginary observer cannot see the interpolated fields, he must woefully assume that equal stimulus conditions do not generate equal response distributions. Such an observer is in a hopeless situation, for he does not yet have a complete set of variables at his disposal with which to discriminate the two situations. He must come to realize that the manner in which a set of points $\mathcal{P}$ is embedded in a larger point field $\hat{\mathcal{P}}$ can strongly alter the dynamical behavior associated with $\mathcal{P}$ even when the additional points $\hat{\mathcal{P}} \sim \mathcal{P}$ are excited only by indirect transmitted excitations from $\mathcal{P}$ and are entirely unperturbed by external inputs.

7. The Immersion of Subfields, the Stability of Local Dynamics, and Effective Dynamical Contractions.

In the laboratory, one is usually confronted with subjects who already possess a working command of an entire language, not merely of two or four points. The comparison between two and four point fields shows that we cannot hope to ultimately ignore the many thousands of points representing the language units in the subject's command without seriously altering our theoretical conclusions. The desire to conceptually decompose a given field into arbitrary subfields of lesser complexity, to study these subfields thoroughly, and to return to the total field by simply pasting the pieces together is seen to be impossible from the outset. We have no linearized approximation at our disposal. We must, rather, always expect in the following pages that as increasingly refined considerations come before us, our old conclusions will show themselves as merely first approximations. The alternative is to present a very complicated nonlinear system at the beginning, without giving the slightest hint of how one can naturally come to understand it.

To avoid the question of how point sets $\mathcal{P}$ come to be embedded in larger sets $\hat{\mathcal{P}}$ as a subject's learning experience grows, we suppose that the field which we are considering at any moment represents an idealization

of the entire point field that interacts with a given class of external inputs. The spontaneous growth of new points is a hard question to which we shall return in gradual steps. The comparison of two and four point fields raises another question to which we can turn immediately with profit. This question can be phrased in several equivalent ways: Under what external input conditions will the dynamics of a large point field approximate the highly stable and symmetric dynamics of a two point field? Under what conditions will the embedding of a given point set into a larger set cause little change in the local dynamics of the original set? If new points are constantly being added to a subject's total point field as his experience grows through time, how does it happen that the subfields representing the learning of old material are not entirely disrupted by the addition of new points and their interactions?

Imagine an embedding field with $n >> 2$ points and practically the same homogeneous initial conditions as before. Alter the condition on p_{ij} by requiring that

$$p_{ij} = \begin{cases} 1 & j = i+1 \pmod n \\ 0 & \text{otherwise} \end{cases}$$

where the symbol $k \pmod n$ denotes the counting of integers modulo n. This condition may also be written $p_{ij} = \delta_{j,\, i+1 \pmod n}$ (diagram 5). (A comparable analysis to the following can be achieved when $j = i+1 \pmod n$ is replaced by $j \in U_i$ = any small ($<<n$) subset of indices.) In this situation, $p_{i(\mathrm{mod}\ n)}$ transmits strength only to $p_{i+1(\mathrm{mod}\ n)}$. Deliver an input to a fixed point, say p_1. Observe that the maximum strength transmitted from p_1 to p_2 at any time is less than or equal to $r < 1$. The strength transmitted to p_3 via p_2 as a result of the input to p_1 is of the order r^2 (<1) at most. Similarly, the transmitted strength to p_k derived from the input to p_1 over the transmission chain $p_1 \rightarrow p_2 \rightarrow \cdots \rightarrow p_k$ is of the order r^{k-1} at most, whenever $k < n$. Contributions that arise from higher order transmissions of the form $p_1 \rightarrow p_2 \rightarrow \cdots \rightarrow p_n \rightarrow p_1 \rightarrow p_2 \rightarrow \cdots \rightarrow p_k$ may be ignored since n is large. It is general rule that higher order transmissions through a cyclic structure

are killed off with successive transmission steps at a rate that is at least multiplicative.

Notice that a comparable sequence of transmissions could have been achieved by setting $p_{ij} = 1 - \delta_{ij}$ and $c^*_{i,\,i+1\,(\mathrm{mod}\ n)}(0) \approx 1$, for strength transmission is determined by the product $p_{ij}c^*_{ij}$. In either case, since the strength transmitted to p_n after an input to p_1 is dominated by $r^{n-1} \ll 1$, an observer at p_n could easily be deceived into believing that no external input had been delivered to the field, even though p_n is strongly connected to p_1 by the line l_{n1}. The structural connections p_{ij} and the line strength functions c^*_{ij} exercise an extraordinary control in their interaction with fixed inputs on the distribution of strength throughout the point field.

Suppose that we are given a field in which these line functions are initially so determined that all inputs to the field arrive at a point set $\mathcal{P}$ which transmits practically no strength to the point set Q, where $\mathcal{P} \cap Q = \emptyset$, and the point strength values of Q are very low at the outset. In the previous example, we can choose $\mathcal{P} = \{p_1, p_2, \ldots, p_{n-1}\}$ and $Q = \{p_n\}$, and can let the collection of external inputs be the input to p_1. Then Q may safely be ignored when studying the dynamics induced by the external inputs to $\mathcal{P}$. Or what is the same, $\mathcal{P}$ can be embedded in $\mathcal{P} \cup Q$ without seriously altering the local dynamics of $\mathcal{P}$. The line functions p and c^* have determined an <u>effective dynamical contraction</u> of $\mathcal{P} \cup Q$ to $\mathcal{P}$ relative to the given external input paradigm to $\mathcal{P}$. Notice that the choice of input paradigm is critical in determining an effective dynamical contraction. In the case $\mathcal{P} = \{p_1, p_2, \ldots, p_{n-1}\}$ and $Q = \{p_n\}$, for example, if inputs are delivered to p_{n-1}, then p_n may no longer be excluded from the set of points which nontrivially influence the dynamics of $\mathcal{P}$.

The problem of determining when an embedding $\mathcal{P} \subset \hat{\mathcal{P}}$ will not disturb the dynamics of $\mathcal{P}$ is the problem of studying how the products $p_{ij}c^*_{ij}$ restrict, or effectively contract, the distribution of strength to proper subsets of $\hat{\mathcal{P}}$ under specific input paradigms to $\mathcal{P}$ at any chosen time. Under complicated input paradigms, the set $\mathcal{P}$ which we are naturally let to consider might itself

change through time, so that the problem becomes one of studying embeddings $\mathcal{P}(t) \subset \hat{\mathcal{P}}$. For example, consider any set $\hat{\mathcal{P}} = \{p_1, p_2, \ldots, p_n\}$ of points such that every p_i is line-connected to at least one other point of $\hat{\mathcal{P}}$. Subject $\hat{\mathcal{P}}$ to a sequence of external inputs that runs cyclically through all of $\hat{\mathcal{P}}$ in the indexed order. In this situation, we cannot hope to isolate a fixed Q which is never dynamically important. Rather, we must examine subsets $\mathcal{P}(t)$ which surround that point to which the last input was delivered. In fact, the closer the line structure approximates $p_{ij} c^*_{ij} = 0$ whenever $j \neq i+1 (\text{mod } n)$, the greater the dynamical contraction will be in this $\hat{\mathcal{P}}$, which is, of course, the $\hat{\mathcal{P}}$ of serial verbal learning. In order to emphasize the importance of a given external input paradigm $\mathcal{J}$ on a field $\mathcal{F}(\hat{\mathcal{P}})$ with point set $\mathcal{P}$, we shall often denote the field so determined by the pair $(\mathcal{F}(\hat{\mathcal{P}}), \mathcal{J})$.

Either as a result of structural isolation of a point by the proper choice of small p_{ij} values or by the approximation of the line structure to the configuration $c^*_{ij} \approx 0$ whenever $j \notin U_i$, where U_i is a small set of indices including $i+1 (\text{mod } n)$, many of the stability properties of a small field can be expected even in a large field under a global cyclical input schema. We infer that dramatic restrictions of p_{ij} values hardly ever occur in simple verbal learning by the fact that all transitions between matched verbal units are possible to learn. The search for the most effective dynamical contractions in serial verbal learning is thus reduced to an examination of those cyclic input sequences under which the c^*_{ij} functions contract most rapidly to some analog of the asymptote $c^*_{ij} = \delta_{j, i+1}, i \leq n-1$.

8. A Rule for Responding.

We earlier postulated that an external input to a p_i with $c^*_{ij} \approx 1$ will generate the response r_j with high probability. The intuitive reason for this was that practically all of the transmitted strength at p_i will be funnelled to p_j over l_{ij}, whence $s_k << s_j$, $k \neq i$, and the various p_k, $k \neq i$, will exert only trivial dynamical effects on the field. We can extend this postulate to say that whenever an input generates strength transmission to a

small set of points, responses from among these points will be more probable than responses from the points which have received only meager transmissions. The intuitive rule becomes: the most effective dynamical contractions generate the most stable responding. Such a rule is completely in keeping with our original reason for studying dynamical contractions. We saw that under an embedding of $\mathcal{P}$ into $\hat{\mathcal{P}}$ we could preserve the dynamics of $\mathcal{P}$ only by choosing the $p_{ij}c^*_{ij}$ functions restrictively. The present rule extends this observation to say that those points whose interactions are the most highly contracted will simultaneously be the points whose dynamics are most resistant to extensions of $\mathcal{P}$ to $\hat{\mathcal{P}}$ and the points most likely to generate responses upon being excited. The points whose excitation by an external input generates the most uniform strength distributions across the field are simultaneously the points which are the least likely to remain dynamically unchanged under extensions of $\mathcal{P}$ to $\hat{\mathcal{P}}$ and the points which have the least behavioral relevance to the subject. The field parsimoniously tends to preserve only those interactions which have arisen through experience. In the case of serial verbal learning, for example, realizing the asymptote $c^*_{ij} = \delta_{j,i+1}$, $j \leq n-1$, means both that the presentation of an input to p_i will always generate the correct response r_{i+1} and that the dynamics of $\mathcal{P} = \{p_1, p_2, \ldots, p_n\}$ will remain maximally stable under the addition to the field of new points and their interactions.

9. Towards a Resolution of Contiguity and Gestalt Theories: Local Strength Fields Do Not Suffice

Achieving a line asymptote like $c^*_{ij} = \delta_{j,i+1}$ is not sufficient to insure stable responding under all cyclic input sequences to p_1, then to p_2, and so on. The length w of the time interval between successive inputs must also be considered. As w decreases to small values, the number of points which simultaneously have high strength values increases, the dynamical contraction becomes less effective, and responding becomes less stable. The ordering of the inputs and all of the initial values of field quantities are the same for all values of w, yet the response distribution varies with w.

By preserving the ordering of inputs as w varies, we have preserved all "contiguity" relations between the points. By preserving all initial values, we have insured that we always begin with the same field, whatever the w. Any classical contiguity theory must predict that all choices of w generate the same response distribution. This does not occur either in the laboratory or in an embedding field. No classical contiguity theory can be adequate. This criticism is not restricted to contiguity theories, but extends to any psychological theory which does not display a running time variable in a prominent place.

It is nonetheless true that one can speak meaningfully in terms from the vocabulary of a contiguity theorist in discussing some of the simplest properties of the c_{ij} and c_{ij}^* functions. But contiguity considerations break down even if we merely alter the temporal relations between the inputs and leave the initial field invariant, or just alter the initial field and leave the input paradigm invariant. Such variations always allow us to find strength distributions which involve interactions over broad portions of the field. When this occurs, we can picturesquely say that the field behaves in a peculiarly Gestaltist way. Part of the tenacity of some of the most perplexing controversies between Gestalt field theorists and peripheralists in the past is doubtless due to the fact that the structures which they studied actually are capable of exhibiting effects which are sometimes compatible with the one theoretical perspective, sometimes with the other. The centrality of this issue and the impossibility of deciding it in favor of any one theoretical group are mirrored by the fact that even our simplest embedding field can exhibit both types of behavior under very natural variations in field conditions. Recognizing that the difficulty lies more in a poverty of theoretical conceptions than in the need for a real choice, let us turn to a finer study of the interplay of these two types of effects.

10. Concentration Sets and Virtual Points

Again imagine a serial nonsense syllable situation with points $p_1, p_2, \ldots, p_n$ labelled in the order of input presentation. Assume the usual

symmetries in initial values, with $p_{ij} = 1 - \delta_{ij}$, and suppose that the time between successive inputs (the intratrial interval) is the same for all items of the list but (possibly) the last and the first item which usually enjoy a longer interval between onset times (the intertrial interval). Define the function $R_{ij} = \min(|i-j|, |n+i-j|, |n+j-i|)$ as a measure of the temporal remoteness of pairs of points relative to the cyclic input sequence, denote the point to which the most recent input has been delivered up to time t by $P = P(t)$, and let $R_{Pk} = R_{k}$. Suppose that the intratrial interval w is fixed relative to the theoretical constants of the embedding field at a presentation rate corresponding to, say, a two second empirical rate; that is, adjust w until it is larger than the largest intratrial intervals for which the c^*_{ij} structure shows only very small fluctuations under cyclic repetitions of the input sequence. Also suppose that the intertrial interval is at least $2w$ in length. We observed in briefly comparing contiguity and Gestaltist perspectives that varying w causes nontrivial effects on the field. We study this and related phenomena now, by setting w at steadily increasing values and considering the strength distribution over all points in the field during a complete single cyclic run through the list at each fixed value of w. A rigorous way to do this is to suppose that a copy $\mathcal{F}^w(\mathcal{P})$ of the field $\mathcal{F}(\mathcal{P})$ is given for every w. Present one cycle of the input sequence to $\mathcal{F}^w(\mathcal{P})$ with an intratrial interval of w. Let s_k^w be the resulting strength function of the p_k point in $\mathcal{F}^w(\mathcal{P})$. As w increases, an increasing proportion of $\Sigma_k s_k^w$ will be "concentrated about P", in a sense which we now explicate:

For any point p_i, let $\bar{p}_i = i$ and choose $\epsilon > 0$. Let $\mathcal{G}_\epsilon(P, t)$ be the maximal set of the form $(p_{\bar{P}-i}, p_{\bar{P}-i+1}, \ldots, p_{\bar{P}}, p_{\bar{P}+1}, \ldots, p_{\bar{P}+j})$ of points p_k for which $s_k(t) \geq \epsilon s_{\bar{P}}(t)$, and let $|\mathcal{G}_\epsilon(P, t)|$ be the number of points in $\mathcal{G}_\epsilon(P, t)$. $\mathcal{G}_\epsilon(P, t)$ is called the ϵ-<u>concentration set about</u> P <u>at time</u> t. As a function of P, $|\mathcal{G}_\epsilon(\cdot, \cdot)|$ increases as P moves from the beginning to the middle of a single run of the input sequence for each w and all ϵ. This happens because the strength residues and the induced transmissions of the first few inputs accumulate as more inputs occur. After P moves from the

middle towards the end of the input cycle, $|G_\epsilon|$ will begin to decrease. This decrease is due to the fact that by choosing the intertrial interval sufficiently long, no new inputs are delivered to the field while the strength values for points near the beginning, middle and, later, at the end of the list have a chance to decay. When considering G_ϵ for P near the end of the list, it is useful to imagine that after $P = p_n$, external inputs continue to be delivered, perhaps several times in succession, to an additional "virtual" point p^* at the w rate. p^* does not at all interact with the p_i and has no dynamical significance apart from the convenience of stating: it is while $P = p^*$ that the $|G_\epsilon|$ shrinkage at the end of the list occurs, and all remarks concerning special effects at the "end" of a single cyclic input sequence refer to times when $P = p^*$. This fact might be found slightly subtle because of its complete triviality: if P did not become trapped in p^* after $P = p_n$, then p_n could not be the end of the list, under our assumption that the intertrial interval is strictly greater than the intratrial interval. While if the intertrial interval is not strictly greater than the intratrial interval, the shrinking effect at the list's end, if it occurs at all, will occur for entirely different reasons, to which we shall presently come. The $|G_\epsilon|$ shrinking effect at the end of a single input cycle is simply due to the additional opportunity for strength decay near the list's beginning, middle, and finally its end, that a longer rest period between inputs at the list's end provides.

For increasingly large settings of w, $|G_\epsilon|$ as a function of P will approach a minimal constant which depends on the constants of the field equations and inputs, and on ϵ. It is in this sense that $\Sigma_k s^w_k$ concentrates about P as w increases. For intermediate values of w, the growth and decay of $|G_\epsilon|$ as a function of increasing P, for variable choices of ϵ, will often be accompanied by the general tendency for $c^*_{\overline{P}, \overline{P}+1}$ to decrease as P proceeds from the beginning to the middle of the list and then to increase as the end of the list is reached. For as increasing ϵ's are chosen, $G_\epsilon(P, t)$, with P and t fixed, shrinks to a set which continues to contain $p_{\overline{P}+1}$ even as other points are eliminated, but the rate with which

other points are eliminated is usually smaller near the middle of the list than at its ends. The strength distribution for P near the ends of the list has less mass and is more peaked than for P just beyond the list's middle (diagram 6).

A concentration set of lines $L_\epsilon(P, t)$ is defined in a similar way relative to the set $\{c^*_{\overline{P}_j}\}$ by $L_\epsilon(P, t) = \{l_{\overline{P}_j}: c^*_{\overline{P}_j}(t) \geq \epsilon\}$. If $|L_\epsilon(P, t)|$ denotes the number of its elements, then as a function of increasing P, for small ϵ, $|L_\epsilon|$ will first increase and then decrease whenever the input paradigm is a single run of a cyclic input sequence with relatively long intertrial interval and intermediate values of w. The dilation, then contraction, of $|L_\epsilon|$ with increasing P is the exact counterpart of the decrease, then increase, of $c^*_{\overline{P}, \overline{P}+1}$ with increasing P. This behavior of $|L_\epsilon|$ for intermediate values of w is an example of an effective dynamical contraction in the normalized line structure near the ends of the list relative to the list's middle. We will see that this contraction in the lines in turn generates a similar contraction in the strength distribution about P induced by repeated input cycles. The relative contraction of the strength field produces many of the known bowing phenomena in serial learning.

Before we proceed to establish this connection, notice that all discussions of the "middle" of the list must be understood relative to the size of w and the size n of the field. For sufficiently large w and sufficiently small n, with no change in the growth and decay constants of the field, the dilation of $|L_\epsilon|$ in the list's "middle" is minimal, and "one-trial learning" of the list occurs. For the ranges of w and n customarily used used in serial experiments, however, w is so small and n so large that significant relative contractions are in fact produced.

11. Bowing Phenomena in Serial Learning

After the fairly long intertrial interval, the nearly exponentially decaying point strength values arising from the first input cycle will have subsided so completely that the line structuring induced by a second input

cycle presentation will be relatively unconfounded by strength residues from the first presentation. The general form of the concentration functions will therefore be preserved on the second trial. The various relative dilations and contractions in the concentration functions might well be accented even further on the second trial by the nonuniform residue of line structure remaining from the first input cycle. For when an input is delivered during the second cycle, the strength will be transmitted through a more highly concentrated normalized line structure than on the first cycle to a subset of points whose point strength values will grow to considerably higher maxima than those of the points not in the concentration sets. As a result, the line functions in the concentrated set will grow correspondingly faster and will usually induce a parallel growth in the normalized lines. By continuing this argument over successive repetitions of the cyclic input sequence, we see that a progressive effective dynamical contraction in the lines occurs until $c^*_{i,\,i+1}$ far exceeds $c^*_{i,\,j}$, $j \neq i+1$, for all $i \neq n$. Nonetheless, letting $\Delta(i,\epsilon)$ be the first time at which $c^*_{i,\,i+1} \geq 1-\epsilon$, $0 \leq \epsilon \leq 1$, and letting $|\Delta(i,\epsilon)|$ be the number of inputs delivered to p_i at times $t \leq \Delta(i,\epsilon)$, $|\Delta(i,\epsilon)|$ as a function of i will often take its maximum near the middle of the list and its minimum near the beginning of the list, with intermediate values for the end. In terms of the number of trials necessary to reach a criterion of $1-\epsilon$ in the normalized lines, we are inclined to say that the middle of the list will be the hardest part to learn.

12. Remote Associations and Asymptotic Chaining in Serial Learning

The gradual contraction of the line concentration set to the linear asymptote $c^*_{i,\,i+1} = 1$, for $i \neq n$, helps to explain standard facts about the distribution of remote associations over a given trial and for increasing numbers of trials. The "strength of associations" from p_i to p_j is again measured in this elementary discussion by c^*_{ij}. The remoteness of p_j from p_i is roughly given by R_{ij}. R_{ij} is a convenient but crude measure of remoteness since it accounts only for ordering relations among the p_i's and omits reference to the difference between intertrial and intratrial intervals.

To offset this difficulty, include p^* in the list as $\{p^*_{n+1}, p^*_{n+2}, \ldots, p^*_{n+k}\}$, where k is the greatest number of integral multiples of the intratrial interval into which the intertrial interval can be decomposed. Measure R_{ij} with respect to the augmented list $\{p_1, p_2, \ldots, p_n, p^*_{n+1}, p^*_{n+2}, \ldots, p^*_{n+k}\}$ in order to include temporal effects. No lines join the p_i with the various virtual points p^*_{n+j}, which we include merely to conveniently compare effects produced at some p_m on one trial with effects produced at another p_m on some other trial.

Fix attention on some p_i. As the linear asymptote is progressively reached with successive input cycle presentations, the bulk of the strength transmitted from p_i to points p_k for which R_{ik} is large will increasingly have to be transmitted to p_k in several steps by way of less remote points. For on any one trial, the c^*_{ik} values generally decrease with increasing R_{ik} in an initially homogeneous field. With the passage of successive trials, the c^*_{ik} for which R_{ik} is largest decrease the fastest, the c^*_{ik} with R_{ik} in an intermediate range decrease less quickly, and so on. As the linear asymptote is approached and the more remote points become only weakly associated with p_i, the strength transmissions flow very much as they did when we chose $p_{ij} = \delta_{j, i+1}$, for $j \leq n-1$. Asymptotically, a chain of successive transmissions becomes necessary to excite a remote point. We have already noted that k-step strength transmission in such a chain is killed off at a rate faster than r^k. As learning proceeds, the frequency of remote associations will thus approach a negatively accelerated function of R_{ik}. After many trials, only a few p_j near to p_i will receive nontrivial strength transmissions when an input is delivered to p_i. The set of remote associations gradually shrinks to sets like $\{p_{i-1}, p_{i+1}, p_{i+2}\}$, and finally to $\{p_{i+1}\}$ alone, at the same time that the negatively accelerated distribution of remote associations is approached. The exponential local decay of point strength at a uniform rate β across points will also contribute to the successive approximation through time of a negatively accelerated strength distribution process to increasingly small sets of points.

Implicit in these remarks is the fact that a similar process holds throughout for the forward and the backward line structures. Usually $c^*_{i, i+1} > c^*_{i, i-1}$, $i \neq 1, n$, by an obvious extension of the argument used to show that

$c_{12}^{*} > c_{21}$ in the four point field. The backward effects will usually be smaller than the forward effects, but similarly distributed. In particular, for a p_i chosen near the middle of a sufficiently long list, remote associations in the forward direction will often include more points and be more frequent than remote associations in the backward direction. A convenient way to discuss such effects is to let the set of points $\{ p_1, p_2, \ldots, p_{\bar{P}(t)} \}$ be called the past field of the input sequence on a given trial, and to call $\{ p_{\bar{P}(t)+1}, p_{\bar{P}(t)+2}, \ldots, p_n \}$ the future field. When $P(t)$ is near the middle of the list, we can usually pair off lines to the future field with lines to the past field so that the line strength of the future field line exceeds that of the past field line.

Our discussion up to now has been concerned with simple thought experiments that illustrate important features of the embedding equations which are compatible with experience. These examples do not nearly offer a complete protocol of the numerous effects that can arise under an actual input sequence, but as more of them are considered, the reader's intuition into the sources of these effects and their interrelationships should correspondingly increase.

13. Feedback and Random Inputs and the Sharing of Stimulus and Response Properties

Some slightly more subtle dynamical effects will now be considered. We require that whenever the subject utters a consonant r_i, whether as a guess of a forthcoming list item or merely to say the stimulus item aloud, an input is delivered to p_i. The onset time of this input will be shifted slightly in time from the onset of the evoked response, but we will ignore this shift in our qualitative discussion. Those inputs which represent the saying of a consonant immediately after it is presented by the experimenter shall again be called experimental inputs. All other inputs are called feedback inputs. We assume for simplicity that the functional form of the two types of inputs corresponding to a subject's utterances is the same, and put the discussion

of this assumption aside until we are prepared for it. For the present, simply let $J_i^{(e)}$ be determined as before, and write $a_i^{(e)}$ and $b_i^{(e)}$ to distinguish these constants of $J_i^{(e)}$ from $a_i^{(f)}$ and $b_i^{(f)}$, which occur in $J_i^{(f)}$, the feedback input. (f) will always be used to mark a feedback quantity. The total input to p_i now takes the form

$$\Sigma_j J_i^{(e)} (t-s_{ij}^{(e)}) + \Sigma_k J_i^{(f)} (t-s_{ik}^{(f)}),$$

where $s_{ik}^{(f)}$ is the time at which the k^{th} feedback input is delivered to p_i. In an initially homogeneous field under a serial input paradigm, we can let $a_i^{(e)} = a_j^{(e)}$, $a_i^{(f)} = a_j^{(f)}$, and so on.

This assumption about feedback inputs implies that responses enjoy many stimulus properties. In the serial verbal learning situation, this is particularly true. It has sometimes been argued to the contrary that guessing responses, in particular incorrect guesses, should not be viewed as self-induced stimuli since they do not always seem to influence the response record. We shall show that the fact that feedback inputs are delivered does not always entail a noticeable change in the response record, and shall delineate those situations for which this is true. The argument that feedback inputs do not exist because the response record does not always change will hereby be shown to be a nonsequitor. In fact, this particular nonsequitor is closely bound to an enduring controversy between "all-or-none" theorists and "continuity" theorists of learning which is fed by a misunderstanding of the way in which inputs interact with field structure in general. The combatants in this controversy, as in the Contiguity vs. Gestaltist controversy, have armed themselves with an incomplete set of theoretical variables. We shall return to this matter after making some preliminary observations based on the existence of feedback inputs.

Since we have assumed that a subject's guesses directly perturb the field, it is important to state more clearly the rules determining when a subject will guess. This we shall do very roughly now to permit a discussion

which will lead naturally to a more precise determination of these rules later on. Suppose that c^*_{ij} increases on successive trials. After successive experimental inputs to p_i, s_j will usually grow to ever higher asymptotes. If s_j exceeds a certain prescribed size τ at a time when it majorizes all other s_k, $k \neq i$, we assume that the subject will utter the consonant r_j. This utterance corresponds to the delivery of a feedback input to the point p_j. Assume for simplicity that no more than one response can occur between any pair of successive experimental inputs.

We now add another member to the family of field inputs: a random input $J^{(r)}_i$ which represents a small fluctuating input such that $\Sigma_i J^{(r)}_i$ is equally distributed in the mean among all p_i in a homogeneous field. $J^{(r)}_i$ is a type of internal input caused by events in regions of the subject's brain which are not represented by $\mathcal{F}\{p_1, \ldots, p_n\}$ and which must consequently be considered as random fluctuations until we are ready to extend $\mathcal{F}\{p_1, \ldots, p_n\}$. The $J^{(r)}_i$ are introduced at this time as a natural complement of the functions $J^{(f)}_i$, which represent the "external" perturbations which $\mathcal{F}\{p_1, \ldots, p_n\}$ sends to itself.

14. Error Distributions

Using these extended postulates, we can interpret our previous remarks on c^*_{ij} functions in terms of response distributions. As usual, once $c^*_{i,i+1} \approx 1$ for all $i \neq n$, the presentation of an isolated input to a p_j, $j \neq n$, will induce one-step transmission almost exclusively to p_{j+1}, and this transmission process will tend both to preserve the normalized line values and to lead to the evokation of the correct response r_{j+1}. For a point p_k near the middle of a long list, the remarks on remote associations become: Anticipatory errors after an external input to p_k will be more frequent than backward errors as a result of the forward bias in the c^*_{kj} structure. Before the c^*_{kj} functions contract from their originally homogeneous state, however, errors might well be scattered broadly about the list. This does not mean that large quantities of errors will surely occur, but only that the distribution of errors will tend to be quite uniform if errors do occur. As the line

concentration functions contract through time, the distribution of errors will contract as well, and will often approximate a distribution that decreases almost multiplicatively with remoteness due to the successive approximation of the normalized line structure to a cyclic chain. One effect of this contraction is the enhancement of the $c^{*}_{\overline{P}j}$ values for small R_j, since the maxima of these s_j, given an input to P, will be larger than the maxima attained within a field of the same size with uniform line structure. The persistent evokation of a nearby anticipatory error is the frequent behavioral correlate of this phenomenon; for example, r_{i+2} is given instead of r_{i+1} in response to an experimental input to p_i.

These remarks have a counterpart for positions near the beginning and the end of the list. For example, the backward line structure is often relatively stronger than the forward line structure for a point p_i at the end of a list than for a point p_k at the list's middle. The response distribution will pattern itself in close correspondence with these relative differences in distribution. Such inequalities as $c_{i,i-1}/c_{i,i+1} > c_{k,k-1}/c_{k,k+1}$ do depend delicately on the ratio of the intertrial and intratrial intervals, but a quantitative study of this dependence is beyond the scope of this introductory discussion.

15. Mixing Distributed and Massed Practice

Distributed practice will be relatively more advantageous than massed practice at the beginning of learning than at its end. For we have already seen that, given a homogeneous field, increasing w causes a decrease in $|L_\epsilon|$ as a function of P. Distributing practice (choosing w large) at the beginning of learning therefore contracts the instantaneous strength distribution generated about P by external inputs, whence the linear line asymptote is more rapidly approximated, and more rapid learning is achieved. Once the normalized line structure is partially contracted, it can better channel the incoming experimental inputs than can a homogeneous field, and thereby reduces the homogenizing effect that widespread simultaneous strength activity has on the normalized line structure.

If the experimenter switches from distributed practice to successively more massed practice after the normalized path structure becomes partially contracted, this channeling, or funneling, capacity of the lines is preserved, thereby enhancing the growth of the $c^*_{i,i+1}$, $i \neq n$, functions in the usual way. At the same time, the new greater contiguity of successive inputs under the massed paradigm even further strengthens the correct paths without the danger of broad strength scatter. The reversed order of massed-then-distributed practice is relatively inferior since massed practice at the beginning of learning spreads the instantaneous strength field over large numbers of points when the lines are still homogeneously distributed. The rate of line contraction to the linear asymptote is hereby slowed, and distributed practice does not begin until the period of its greatest usefulness in creating a highly contracted strength field has passed. The relative magnitudes of these reversal effects depend sensitively on the massed and distributed input rates and on the number of trials devoted to each rate—a nondenumerable infinity of situations in all. Our comparison merely illustrates in a qualitative way some of the features common to all of these situations which can be used to guide the understanding of any one of them. Experimental papers in the literature unfortunately do not always explicitly state all of the variables necessary to achieve a quantitative understanding of reversal situations. In particular, merely saying that subjects were run on massed practice until one-half of a certain learning criterion is fulfilled and then on distributed practice until the criterion is perfectly fulfilled gives little insight into the relative usefulness of the two types of practice and on the total effort required to reach the one-half way mark. With this omission, the inclusion of data on such matters as the total number of errors generated under massed and distributed practice becomes theoretically useless, since it is impossible to judge the rates of error emission.

The reversal situation provides our first example of an input regime which is not merely a regular repetition of a given input cycle. Much more general input paradigms can easily be imagined. Every such paradigm can be delivered to an embedding field and will generate a well-determined change

in field structure. With such a program in mind, it is encouraging that our first deviation from a strictly repetitive input paradigm has generated familiar serial learning effects.

16. Reversing Input Rates on Presentation and Recall Trials

An analog of the reversal situation is the following: A list of moderate length is presented for just one trial at a fixed rate. The presentation trial is followed by a single recall trial at another fixed rate. We expect that a slow presentation rate and a fast recall rate will be better than a fast presentation rate and a slow recall rate at enhancing the recall of the beginning of the list relative to its end. $c^*_{i,\,i+1}$ growth for points at the beginning of the list is encouraged more by slow delivery of inputs on the presentation trial than by fast delivery, since $|C_\epsilon|$ is smaller in the slow case. As usual, the concentration function will increase as the middle of the list is reached and will decrease as the list's end is approached. Forcing fast recall of items, however, increases the rate of input to the system without changing the local rate of strength decay. $|C_\epsilon|$ will therefore not shrink to small values as P approaches the end of the list because point strength across the field has not had an opportunity to decay. Correct responding from points near the end of the list will be correspondingly reduced, even though on the presentation trial their line structure better approximated the linear asymptote than did the lines of points near the middle of the list.

A fast presentation rate of inputs will cause the concentration function to rise very quickly as P moves away from the beginning of the list. Large values of this function will be maintained throughout the middle of the list as well. The correct line structure will therefore be relatively weak at the beginning and middle positions of the list. The end of the list will have its usual line structuring advantage, since no further inputs becloud the strength distribution once $P = p^*$. The contraction of the instantaneous strength distribution that is encouraged by the slow recall trial cannot help

substantially to induce correct responses in the beginning and middle of the list in this situation, for the correct normalized line structure had never formed during the presentation trial. On the other hand, the slow presentation rate will insure relatively small values of the concentration function near the end of the list so that the extant correct line structure here will not be obscured by global strength scatter in its interaction with inputs to produce correct responses.

This example once again shows that the interaction of the line functions with the instantaneous distribution of strength values under a specific input paradigm through time accounts qualitatively in a simple way for important phenomena that must be considered mysterious if even one of these concepts, or an analog thereof, is removed from the analysis.

17. Reminiscence, Sustaining Inputs, and Dual Fields

A similar discussion can be applied to the phenomenon of reminiscence. Consider a field that closely approximates the linear asymptote. After the last input arrives and the strength field decays, but before the normalized line structure decays substantially, one can easily envisage a period during which a new experimental input will produce a strength distribution that is more contracted and thus more favorable to correct responding than a strength distribution produced by an input delivered right after the last learning trial. A quantitative understanding of reminiscence presupposes an intensive study of the distribution of internal inputs that perturbs the embedding field after experimental inputs cease. Can we by a priori arguments qualitatively understand what such a distribution must look like to produce phenomena like reminiscence and the extraordinary stability of memory in general?

Recall that the functions of a free embedding field converge to zero as time passes. A field cannot be freed from inputs for such a long time after the experimental inputs cease that this homogeneous limit is approached, for we should then have no memories. Internal inputs must therefore perturb the field constantly whenever experimental inputs are not being delivered. On

the other hand, when experimental inputs are being delivered, it is important that internal inputs be small--unless the subject is guessing--for otherwise line residues could never properly form in the first place, and whatever memories we should have would be confused ones. We will therefore look for a mechanism to spontaneously produce internal inputs when experimental inputs are absent and which suppresses internal inputs when experimental inputs appear.

How shall the point to which an internal input is delivered be determined? The only way to distinguish the points of a field with equal M_i values is to examine the inhomogeneities in the lines connecting them. The only way that an internal input-generating mechanism has at its disposal to examine the inhomogeneities in these lines is to deliver an input and to measure the inhomogeneities in the distribution of strength that is thereby generated. These inhomogeneities will only be felt at the input-generating mechanism if the induced strength is transmitted to the mechanism over some sort of line structure. We must therefore search for an extension of the original field that interacts with it in a reciprocal way. If all of the internal inputs are delivered randomly, the line asymptote generated by the experimental inputs will quickly be homogenized. One internal input must therefore help to determine the next.

In particular, if one internal input arrives at p_i, then the line residue will certainly be best preserved if the next input arrives at p_{i+1}. The input generating mechanism fortunately has precisely one way of knowing which point p_{i+1} is: p_{i+1} is that point which transmits the greatest amount of strength to the input mechanism after an internal input is delivered to p_i. We must conclude that the input mechanism's behavior is determined by a weighting process whereby all of the various strength values emitted by the field at any time are compared, and the largest internal inputs are sent to the points which have transmitted the most strength. In this way, the input generating mechanism can imitate step-by-step the ordering of experimental inputs and thereby preserve the line structure induced by these inputs.

It is too much to expect that the input generating mechanism will

always go through the entire experimental cycle over and over again. All that is required is that if an internal input is delivered to p_i and if some other internal input is to be delivered shortly thereafter, it should be delivered to p_{i+1}. The internal inputs must imitate the experimental paradigm locally. If just one internal input is delivered, say to p_i, the loss is not great, since the line residue will transmit most of the strength to p_{i+1}, which is the transmission most likely to preserve a high $c^*_{i,i+1}$ value. Because the input generating mechanism has this property of generating short strings of inputs in the proper order, we say that it generates a local walk on the embedding field.

The input generating mechanism must itself be a type of embedding field. Since it is so closely bound to the originally embedding field, we will call it the dual field of this embedding field. We must search for a dual field which can generate local walks. Posed in this way, the problem is really almost qualitatively solved, but we will approach it later from a still simpler point of view and only then will we begin to write down equations for dual fields. It is a most striking fact nonetheless that purely macroscopic psychological data, plus the observation that we remember some things very well, has generated the notion of a field extension which is not macroscopically visible, and has even given us the basic property of this field.

Notice in passing that a dual field must also be closely related to general guessing and free association phenomena. For these phenomena always begin with a "random" input to some field which generates another input on its heels, and then the process repeats inself. Dual fields will give insights into many problems all at once.

18. Clustering in Serial Response Distributions

Several somewhat less standard effects will now be mentioned. Suppose that $c^*_{i+1,i+2}$ is given to us relatively large. Then if experimental inputs are successively delivered to p_1 and to p_{i+1}, much of the strength generated at p_{i+1} will be transmitted expeditiously to p_{i+2} instead of

being scattered over the point field. The growth of $c^*_{i,i+1}$ will thereby be augmented even though $c^*_{i,i+2}$ will be strengthened over the lines l_{ik}, $k \neq i, i+1, i+2$. Now suppose that the usual input cycle is presented repetitively, again given an initially high $c^*_{i+1,i+2}$. Once more $c^*_{i,i+1}$ will achieve a relative advantage. As trials continue, the advantage to $c^*_{i,i+1}$ will be passed on to $c^*_{i-1,i}$, then to $c^*_{i-2,i-1}$, and so on. Viewed from a fixed point p_i, it makes little difference if two or many successive $c^*_{i+k,i+k+1}$, $k \geq 1$, values are large when producing a high $c^*_{i,i+1}$ value. The relevant factor is the rapid channeling of transmitted strength from p_{i+1} to a small set of chained points during the time interval immediately following the delivery of experimental inputs to p_i and p_{i+1}. The s_i induced by an experimental input to p_i will usually decay to insignificant values before it can be transmitted over a long chain of points. In other words, the effective length of a string of $c^*_{i+1,i+2}, c^*_{i+2,i+3}, \ldots$ of high line values, relative to the process of channeling strength from p_{i+1}, stabilizes at a small integral constant value.

Consequently, once the length of a string of high c^* values exceeds this small constant, if other field effects remain fairly uniform, the rate at which new c^* values is added to the string of large c^*'s will be approximately linear. One measure of this rate is $\Delta_{\epsilon}(i) = \Delta(i,\epsilon) - \Delta(i+1,\epsilon)$ for suitably chosen ϵ. Deviations from linearity will be caused by the fact that other high c^* values are being produced on every successive trial, so that the distribution of strength is not controlled to the same degree on sucessive trials by the growing chain. This loss of control by the growing chain is compensated by the fact that successive trials tend to augment precisely those line values which would enter the chain at a later stage. The lines $l_{i-k,i-k+1}$ for k the largest are precisely those which are the least benefited by the chain induction and those which have the highest values when the chain finally reaches them. The two growth effects are nicely blended. The channeling capacity is not transferred from point to point with complete efficiency even on a single trial. The tendency for the efficiency of channeling transfer to

decrease between a given p_{i-k} and its immediate successor, as k increases, is partially offset by the very increase in length of the chain of successors which is the hallmark of decreasing efficiency. For as the efficiency of transfer over a single $c^*_{i-k,\,i-k+1}$ decreases, a greater total c^* weight is donated to $l_{i-k,\,i-k+2}$ and $l_{i-k,\,i-k+3}$, which still is compatible with the necessary transmission of s_{i-k} to a small set of points in the future field.

Similar remarks hold for the augmentation of $c^*_{i+1,\,i+2}$ growth when $c^*_{i,\,i+1}$ is large. Here, however, the line channeling keeps the irrelevant past strength field contracted while it simultaneously transmits a high strength value from p_i to p_{i+1}. Again the rate of chain growth might well approach linearity. The two chaining processes can proceed simultaneously to yield the effect of a growing chain of high c^* values in both the temporally forward and backward directions about the p_i, p_{i+1} locus. A corresponding effect on the distribution of correct responses through time will be observed. The differentially large initial $c^*_{i,\,i+1}$ hereby induces a clustering effect in the response paradigm about p_i and p_{i+1} in both the forward and backward directions which often propagates at approximately a linear rate over trials.

19. Towards a Resolution of All-or-None and Continuous Theories of Learning: Errors for Which We Do Pay and Errors for Which We Don't Pay

Suppose now that the line structure and the random internal input function are such that r_k has been uttered in response to an experimental input to p_i, where $k \neq i+1$; an overt error has been committed. The evokation of this error generates the delivery of feedback input to the point p_k at the onset time of its evokation and c_{ik} growth will be facilitated accordingly. If the evokation of an error r_k causes c_{ik} to grow even further, how can errors ever be corrected? This is clearly a subtle matter and a full explanation requires extensive considerations. We shall merely sketch some of the most important factors at this time.

Consider two fields $\mathcal{F}_1$ and $\mathcal{F}_2$ for which identical symmetrical initial conditions are assumed. Let an interval of time $[a, a+z]$ of the duration of several successive experimental inputs be given during which the functions s_i, s_{i+1}, and s_k, i and k fixed, $k \neq i, i+1$, are the same in both $\mathcal{F}_1$ and $\mathcal{F}_2$, and assume that all dynamical events occurring before t = a have had only a trivial effect on c_{ik}, $c_{i,i+1}$, and $c_{i+1,k}$. c_{ik}, $c_{i,i+1}$ and $c_{i+1,k}$ are thus the same in both fields during $[a, a+z]$. Suppose that s_i, s_{i+1}, and s_k during $[a, a+z]$ have in both fields been induced by an experimental input to p_i followed successively by a feedback input to p_k and an experimental input to p_{i+1}: in response to the presentation of p_i, the error r_k is evoked and is corrected by the presentation of r_{i+1}. All of these conditions hold in both $\mathcal{F}_1$ and $\mathcal{F}_2$. To distinguish $\mathcal{F}_1$ and $\mathcal{F}_2$, suppose that the s_u, $u \neq i, i+1, k$, are all very small in $\mathcal{F}_1$, while in $\mathcal{F}_2$ there exist s_u's which are quite large during $[a, a+z]$.

In $\mathcal{F}_1$, the growth of c^*_{ik} will closely parallel that of c_{ik} in the period before the second experimental input, and although the growth of $c_{i,i+1}$ after this event will deter the c^*_{ik} growth, a positive net effect will usually be expected, so that the error really does acquire an advantage in the residual normalized line structure. In $\mathcal{F}_2$ on the other hand, the large s_u values will induce large values for the corresponding dc_{qu}/dt, $q = i, i+1, k$. Consequently, not only will $|dc^*_{ik}/dt|$ usually be small, but also dc^*_{ik}/dt might well be negative.

This difference between c^*_{ik} growth in $\mathcal{F}_1$ and $\mathcal{F}_2$ has important implications. For example, consider the first few trials of a typical serial learning situation, when very often the line structure is quite uniform. The small degree of channeling in strength transmission that one finds here generates a broad distribution of large strength values that resembles the strength field of $\mathcal{F}_2$. The discussion of $\mathcal{F}_2$ shows that an error r_k evoked when r_i is presented need not cause c^*_{ik} to grow. On future trials, therefore, no record of the evokation of the error exists in the normalized line structure, and it is the interaction of the normalized line structure with the instantaneous strength distribution that causes responses. The

evokation of an error early in learning therefore need not in the least cause difficulties in learning the correct response on later trials. In fact, whenever a field like $\mathcal{F}_2$ is produced, this conclusion holds. Such effects have indeed often been reported, but have been to the present encased in the terminology of "all-or-none" learning, which all too frequently provides little more than a rubric for them.

On the other hand, if the error r_k is evoked after an experimental input to p_i in a more highly line structured situation, a smaller concentration set will be found. If this set concentrates almost exclusively at the points corresponding to successive experimental inputs and an interpolated error, we shall be in the case of $\mathcal{F}_1$. The growth of c^*_{ik} in $\mathcal{F}_1$ is matched by a greater difficulty in overcoming the error r_k and learning the correct response on later trials. Such interference effects are commonplace. Numerous experiments have studied them by training subjects to some nontrivial partial criterion on a given list, rearranging the list items, and retraining the subjects on the new list. The time which these subjects need to reach a certain criterion on the second list is compared with the time required by fresh subjects to reach the same criterion on the second list alone. It is the classical transfer paradigm and the results are well-known. Retroactive inhibition may be similarly understood. All of these classical serial transfer situations leave one with the impression that the learning of items, including the correction of errors, proceeds in a gradual or "continuous" way.

The situation before us now is, in spirit, similar to that which kept alive the controversy between Gestaltists and Contiguity theorists. We saw earlier that this controversy was sustained by the existence of empirical situations which provided data that was well-taylored to the needs of each party. We also showed that an embedding field also exhibits each kind of behavior under well-specified conditions. We have in the comparison of $\mathcal{F}_1$ and $\mathcal{F}_2$ now shown that a single embedding field can also exhibit behavior of both the "all-or-none" and of the "continuous" variety, the one or the other depending on the input sequence and the initial field structure. Once again it seems that a controversy cannot be resolved

by a victory. The worst that should be sought is a better catalogue of the initial and boundary conditions compatible with each type of behavior.

It is useful to observe here that evidence supporting the "all-or-none" theorists is often of two types. On the one hand, the evokation of errors in fields like $\mathcal{F}_2$ is shown not to interfere with later learning. On the other hand, certain paired associate situations are studied in order to demonstrate that once an association is mastered, it is evoked correctly thereafter with very high probability. It is a striking fact, which we shall later study in greater detail, that the paired-associate situation represents, even more than $\mathcal{F}_1$, a highly contracted form of embedding field, in fact a close analog of the two point field, for which responding is very stable. Thus, "all-or-none" theorists find their greatest support in either homogeneous fields under input sequences which initially generate a broadly scattered strength distribution, or under input sequences which from the outset generate highly contracted strength fields. It is in the intermediate range of fluctuating strength field contractions that the "continuous" theorists find their bulwark. Since most standard learning paradigms involve a successive approximation from rather homogeneous initial field structures to terminal field structures exhibiting highly contracted concentration sets, the theoretical victory banner must pass hands twice during a single experiment.

20. One Way to Correct Errors

In our discussion of errors to now, the remoteness of errors has not occupied a central conceptual position, but only arises as a special type of interaction between line and strength functions. Were this not the case, we would be in serious difficulty. For before learning begins under homogeneous initial conditions, no natural temporal order whatsoever exists in the field's dynamics which can distinguish an ordering in the field's points. Questions of ordering pairs of points by their remoteness are initially quite meaningless, and our prescient labelling of the points in the order of

experimental inputs is merely formal handicraft. The homogeneous field is designed to be able to incorporate into its own natural dynamical evolution any temporal ordering of events imposed upon it by the envioronment. The elimination of errors is a part of this replication process which is definable only relative to a fixed temporal ordering. Errors are not intrinsically classified within the field in terms of remoteness until they are all eliminated, for it is only when learning of some list is perfect that the field's dynamics exhibit a true temporal order. Many classical psychological concepts are of this type: they become accurate reflections of field activity only asymptotically.

A closer study of error correction requires the introduction of some convenient notation. Let $\pi_u^{(e)}(t)$ $(\pi_u^{(f)}(t))$ denote the event of delivering an experimental (feedback) input to p_u at onset time t. Suppose that the intratrial interval w is longer than v, and let the sequence $\pi_i^{(e)}(t)$, $\pi_k^{(f)}(t+h_1)$, $\pi_{i+1}^{(e)}(t+w)$, $0<h_1<w$, occur in a field like $\mathcal{F}_1$. Also suppose that the sequence repeats itself on several successive trials with h_q replacing h_1 on the q^{th} trial: $\pi_i^{(e)}(t)$, $\pi_k^{(f)}(t+h_q)$, $\pi_{i+1}^{(e)}(t+w)$, $0<h_q<w$. As these events proceed, c_{ik}^* and $c_{k,i+1}^*$ will both at first grow. As $c_{ik}^* c_{k,i+1}^*$ increases, p_{i+1} will receive more second-order transmitted strength from p_i via p_k, in addition to its experimental input. Moreover, the strength transmitted over the chain $p_i \rightarrow p_k \rightarrow p_{i+1}$ will be received during a period overlapping $\pi_{i+1}^{(e)}$'s greatest effect on s_{i+1}. Consequently, the growth of $c_{i,i+1}$ will be augmented, and during periods which overlap the periods of maximal growth for c_{ik}. c_{ik}^* will therefore gradually decrease and $c_{i,i+1}^*$ will increase on successive trials until their values are so close that either no response will occur after $\pi_i^{(e)}$ or the responses r_k and r_{i+1} will begin to oscillate. Since on every successive trial $\pi_{i+1}^{(e)}$ is sure to follow $\pi_i^{(e)}$, $c_{i,i+1}^*$ eventually becomes very large, and the correct response is learned.

Of course, this is not the only way in which errors are corrected. Another way is available if the intratrial interval is not too short. Under

these conditions, a subject can rapidly deliver in a subvocal fashion a series of alternating pairs of the form $(\pi_i^{(f)}, \pi_{i+1}^{(f)})$ between successive experimental inputs. If $\pi_i^{(f)}$ and $\pi_{i+1}^{(f)}$ are subvocally alternated at a fairly uniform rate and sufficiently often, then a significant increase in the symmetry, as well as the absolute magnitude, of the pair $(c^*_{i,i+1}, c^*_{i+1,i})$ might be induced. The entire pattern of inputs and initial values must be analysed to deduce such a symmetrizing effect with certainty. In particular, the dependence of $c^*_{i+1,i}$ on the behavior of the subject in the next time interval can upset this effect in a serial paradigm. If the serial input sequence is abruptly terminated to test the effect immediately after such a repetitive cyclic alternation is somehow guessed to have occurred, then the input paradigm locally approximates that of a cyclic two point field. For the case that is directly testable, therefore, the symmetrizing effect becomes theoretically trivial.

The fact that subvocal practice occurs and is not directly measurable is in itself not a theoretically disabling fact. Difficulties do arise, however, from the fact that the subvocal practicing techniques employed will vary between individuals to a much higher degree than does the common dynamical process necessary for serial learning to occur. In consonant fields, this process differentiates individuals wholly by the proper setting of constants and initial values. No difficulty occurs in doing this, since it is clear what the fundamental units of such fields are: they are simply the consonants themselves. When we construct higher-order embedding fields to represent the accumulated experience of a given individual, the fundamental units are not nearly so easy to guess, and they vary considerably between individuals. The consonant and higher-order fields of a given individual are, moreover, dynamically dependent. The variability which we find in these higher-order fields is therefore capable of inducing a greater variability in the dynamics of the consonant fields of different individuals than we should expect from a cursory inspection of the simple structural carriers of consonant fields. This variability is introduced by the delivery of internal feedback inputs to the consonant fields from the higher-order fields, and no contemporary

psychological technique can accurately measure these internal inputs. Comparatively fast presentation rates have been useful at an early stage of research because they reduce the variability of practice strategies by driving the simple consonant fields and thereby partially suppressing the influence of more complex fields.

Nonetheless, whenever the task of conceptualizing even the rudiments of the entire process of verbal learning arises, the specification of such higher-order phenomena as subject strategies cannot be completely avoided. Indeed, it is easy to conceive of a subject for whom all of the correct responses are available but who perversely refuses to say them aloud. Or a subject might simply have a ferocious cough. We escape dilemmas of this kind by calling them pathologies of the process and by noting that one must distinguish between the concepts of response availability and response evokation. The former concept is the more pertinent one in a preliminary study. We will now give an approximate solution to the problem of specifying response availability. The interpretation of this solution will be interesting in its own right when we study how it can be dynamically realized in extended embedding fields which take higher-order phenomena into account in an intrinsic way.

21. Response Availability and the Information Functional

The embedding field that we have been using represents the averages of certain activities that proceed through time over extensive portions of the cerebral cortex and related cell structures. The consonant field cannot be expected to be in a simple correspondence with a particular localized structure in a brain. At the same time, it does aim at representing that portion of the learning process which corresponds to actual variations in the brain's memory structure. To study how these variations become behaviorally available, it is necessary to examine the manner in which the memory structure acts upon other regions of the brain to induce conscious verbal forms and motor acts. The first step in

this examination amounts mathematically to the specification of certain functions of the point strength functions and of appropriate criteria whose fulfillment by these composed functions corresponds to the availability of a particular response. Perhaps the simplest useful choice of functions is the following one:

Let

$$s_i^* = \frac{s_i}{\Sigma_j s_j} ,$$

$$H_i = H_i(s_1, s_2, \ldots, s_n) = - s_i^* \log_2 s_i^* ,$$

and

$$H = \Sigma_i H_i$$

H is the familiar information functional of the normalized s_i functions. Now let

$$\overline{H} = 1 - \frac{H}{\log_2 n}$$

and

$$A_i = s_i \overline{H} .$$

A_i is called the availability function of p_i. These functions have the following properties: (1) $0 \leq H \leq \log_2 n$, so that $0 \leq \overline{H} \leq 1$. (2) $\overline{H} = 1$ only when $s_i^* = 1$ for some i; that is, when $s_j = 0$, $j \neq i$, and $s_i > 0$. (3) $\overline{H} = 0$ only when $s_i^* = 1/n$ for all $i = 1, 2, \ldots, n$; that is, when $s_i = s_j > 0$ for all i and j. $\overline{H}$ is thus a measure of the distinctiveness of the distribution of the s_i. A_i on the other hand is a composite of the absolute magnitude of s_i alone and the distinctiveness of the instantaneous global strength distribution. Even if $\overline{H}(s) = \max_w \overline{H}(w) = 1$, $A_i = \min_w A_i(w) = 0$ unless $s_i^* = 1$.

Now suppose that a response criterion constant Υ, $0 < \Upsilon < \min_i M_i$,

is given and the following response availability rule: Let $\pi^{(e)}(t_0)$ occur, and suppose that the following conditions are first satisfied after t_0 at $t = t_1$, where $t_0 < t_1 <$ the onset time of the next experimental input: (1) $\Upsilon \leq A_i(t)$, (2) $A_j(t) \leq \Upsilon - Y$, for all $j \neq i$, where Y is a fixed constant in $(0, \Upsilon)$. Under these conditions, the response r_i becomes available at time $t_1 + \lambda$, where $0 < \lambda << v$. λ is often a small fluctuating quantity, but since we shall shortly study some sources of these fluctuations, fixing λ at a constant value now causes no difficulty. Add the following behavioral strategy: The first available response following an experimental input will be evoked publicly when it becomes available. Whether or not a second available response will be evoked as readily in a given intratrial interval is a matter of much greater individual variability. We fix ideas by supposing that no more than one feedback input will be delivered in a given intratrial interval.

In keeping with the principle that every dynamical quantity should be associated with an appropriate portion of the structural carrier, an emitter line $l_{i,(r)}$ is drawn from each p_i to a common response point $p_{(r)}$ (diagram 7). A_i must be viewed as the result of the excitation of $p_{(r)}$ by strength transmitted from $\{p_i\}$ to $p_{(r)}$. The fact that the result of this excitation is A_i, and not simply $s_{(r)}$, means that $p_{(r)}$ carries a dynamical process that must be viewed over a larger field than a single point $p_{(r)}$ if we want to understand the interactions whereby $A_i(s)$ is produced from $s = (s_1, s_2, \ldots s_n)$. Lack of a c function in the definition of A_i means that transmission from $\{p_i\}$ to $p_{(r)}$ does not induce residual biases in $\{c_{i,(r)}\}$ that were not already included in $\{c_{i,(r)}(0)\}$. The absence of nontrivial p_{ik} functions is a homogeneity assumption that does not hold when the consonant field is not itself homogeneous. In inhomogeneous cases, A_i and H_i are replaced by

$$A_i(p_{(r)}, t_{(r)}; s_1, \ldots, s_n) = \tilde{s}_i \cdot \left[1 - \frac{\tilde{H}}{\log_2 n} \right]$$

where

$$p_{(r)} = (p_{1,(r)}, p_{2,(r)}, \ldots, p_{n,(r)}),$$

$$t_{(r)} = (t_{1,(r)}, t_{2,(r)}, \ldots, t_{n,(r)}),$$

$$\tilde{H} = \Sigma_j \tilde{H}_j,$$

$$\tilde{H}_j = -\tilde{s}_j \log_2 \tilde{s}_j,$$

and

$$\tilde{s}_j = \frac{p_{j,(r)}\, s_j(t - t_{j,(r)})}{\Sigma_k\, p_{k,(r)} s_k(t - t_{k,(r)})}.$$

Here $p_{j,(r)}$ is a nonnegative constant which plays the same role for $l_{j,(r)}$ as p_{ik} does for l_{ik}, $t_{j,(r)}$ is a transmission time lag. The homogeneous $A_i(s)$ is $A_i(1, 0; s)$. In fact, we are free in the homogeneous case to set $t_{k,(r)} = \epsilon$, for all k, and to require that responses become available $\lambda - \epsilon$ time units after (1) and (2) are satisfied, just so long as ϵ is any number in $[0, \lambda]$. No variation of this kind changes the behavioral record.

22. Response Availability and Response Interference

The specification of availability functions A_i and of a response criterion allows us to survey previous remarks about responding more rigorously. These remarks usually took the form: an isolated input to a point p_i with $c^*_{i,i+1} \approx 1$ generates a strength distribution that concentrates at p_{i+1} so intensely that r_{i+1} occurs with high probability. We also observed that the repetition of experimental input sequences generates precisely those asymptotic line distributions, like $c^*_{i,i+1} = 1$, $i \leq n - 1$,

which best facilitate those response processes that are controlled by intensely concentrated strength distributions, and that these asymptotic line distributions ensure that the responses produced after long practice are the ones we set out to learn.

$\overline{H}$ provides an excellent measure of the degree to which the normalized strength distribution $(s_1^*, s_2^*, \ldots, s_n^*)$ is concentrated over the field at any time. $A_i = s_i\overline{H}$ measures jointly the normalized global strength distribution and the intensity of this distribution at p_i at any time. Measuring all magnitudes relative to τ, we can say that whenever $A_i \geq \tau$ and s_i is itself not very large, the distribution of $\{s_i\}$ clusters strongly about p_i. On the other hand, whenever $A_i \geq \tau$ and s_i is very large, the activity at p_i overshadows even a somewhat more broadly scattered strength field. The conditions (1) and (2) together assure that r_i responding occurs only if one of these alternatives holds at p_i at the same time that neither of them holds at any other point. The strength field must concentrate intensively about precisely one point before that point can generate a behavioral act in a consonant field. The fact that two conditions, (1) and (2), are needed to specify this shows that the process of selecting a response involves a more subtle temporal interaction than can be displayed as a process over the single point $p_{(r)}$.

Since A_i provides a sensitive measure of the intensity of activity and its concentration at p_i, all of our previous qualitative remarks about responding hold for responding that is controlled by A_i and τ. The advantage is that now all of our remarks become quantitative. In particular, suppose that $s_j(t_0) = s(t_0)$, for all $i \neq j$, and that $s(t_0) < s_j(t_0)$ in the two fields $\mathcal{F}_1$ and $\mathcal{F}_2$. Let $\mathcal{F}_1$ possess n points and $\mathcal{F}_2$ possess m points, where $m < n$. The definition of A_i shows that $s_j(t_0)$ must be larger in $\mathcal{F}_1$ than in $\mathcal{F}_2$ for r_j to occur at time $t_0 + \lambda$, and we can measure how much larger it must be. The response disadvantage of $\mathcal{F}_1$ relative to $\mathcal{F}_2$ is a simple case of response interference within a

nontrivial class of response alternatives. Using only the ideas presented to now, we could catalogue many subtle response interference effects, and in fact, all of the remarks about concentration sets are remarks about when "responsesare interfering" and when they are not.

It is more important to realize that until the response criterion is met and a feedback input is delivered, no field point behaves any more like a response than any other. Even when a feedback input is delivered, it is not so very different from an experimental input delivery that we can feel comfortable in distinguishing stimuli and responses by observing the inputs to which they correspond. Yet apart from differences in input,we cannot intrinsically recognize a stimulus or a response at all by just looking at the dynamical behavior of a field. The vocabulary of "stimuli" and "responses" is a production of Man that is useful when it is applied as a convenient rubric. When it is used to imply drastic general dynamical differences between "stimuli" and "responses", it violates an incomparably more subtle production of Nature.

23. Sets of Response Alternatives and Psychological Sets

No small part of the difficulty which has been found in distinguishing stimuli and responses is due to a misunderstanding of how responses are generated. A prerequisite to knowing how a response is generated is knowing what a "set of response alternatives" is, and one cannot discuss this latter concept without knowing under what circumstances a response becomes available. In particular, an enormous point field can be given, but if only a very small number of points at any time have nontrivial strength values, then the definition of A_i shows that only this small set of points effectively serves as a set of response alternatives at that time. If a temporally isolated input is delivered to

only one point, then the set of response alternatives in the time period immediately following the input is wholly determined by transmission from that point to other points in the field. This transmission might reach only one point, or possibly hundreds of points, depending on the line structure at that time. The set of response alternatives varies accordingly. Moreover, even if not a single additional input is delivered thereafter, five minutes later the set of response alternatives will be entirely different, since strength transmission and decay continue even while the experimenter rests.

The field of response alternatives is coextensive with the activated strength field at any time. Both flow from point set to point set, guided by the total arrangement of inputs and the equations of embedding dynamics. To discuss whether a point serves as a response is to discuss when it becomes available. And so on. It becomes abundantly clear that the hope to understand learning by doing no more than assiduously counting stimuli and responses is bound to be frustrating, for it is based on an incorrect view of how learning proceeds.

The difficulties in defining a "set of response alternatives" are identical with the difficulties which arise in defining a "psychological set." Both concepts vaguely suggest that certain clusters of units are more important than others in different circumstances. Notions of "response alternative" of the "response interference" type refer to these clusters with the emphasis on a preference for certain units, which is usually guided by a consideration of the expected response asymptote. "Psychological set" emphasizes the mere "relevance" of one unit to another. In embedding dynamics, "relevance" is determined largely by the line structure. If p_j is "relevant" to p_i, then $p_{ij}c^*_{ij}$ is large, so that an input to p_i will excite p_j by transmission, and an excited p_k has more behavioral significance than an unexcited p_k.

The concepts of "sets of response alternatives," "response interference," and "psychological set" reveal a general situation. Each

of them intuitively suggests psychological phenomena that overlap one with the other, and none of them provides a clear insight into any one psychological fact. This is true of many, if not most, of the large collection of psychological concepts to which we had access to now. The natural application of such an unwieldy collection of ambiguous concepts is a confusing effort to find which combination of concepts does the least violence to a given empirical setting. Our prospects now are much better. For we can see precisely where the multitude of old concepts fails, and we have a new, simple, and small vocabulary with which to discuss our empirical ideas in a quantitative way.

24. Where is the Middle of a List? The Immediate Memory Span

The specification of a quantitative response availability rule completes the task of constructing a simple consonant field. This field shall be extended and refined in several directions in the following pages, but we can introduce certain quantitative concepts now which are pertinent to a consonant field in all of its forms. In our qualitative discussion, frequent reference was made to a list's "middle". The "middle" of a list may easily be heuristically distinguished from its two ends. Yet the "middles" of lists of varying length which are constructed from different verbal units and presented at different rates all behave in different ways. The simplest example of this variability is found in the two point field with points p_1 and p_2, which seems to have two ends and no middle. Under an input sequence $\{\ldots, \pi_2^{(e)}(t-2h), \pi_1^{(e)}(t-h), \pi_2^{(e)}(t), \pi_1^{(e)}(t+h), \ldots\}$, however, the field of $\{p_1, p_2\}$ behaves as if it were all "middle" with no ends. A definition of "middle" is needed which distinguished a list's middle by its dynamical behavior. Such a definition is usually given in terms of the idea of an immediate memory span, which we now make precise.

Suppose that n disjoint lists L_i, $i=1, 2, \ldots, n$, of matched

consonants, a consonant field $\mathcal{F}$ containing all of these consonants, two intratrial intervals w_1 and w_2, and an intertrial interval T are given. L_i is assumed to contain i consonants. Let L_n be presented once with intratrial interval w_1. Require the subject to say the consonants aloud as they appear. Pause for T time units. Present L_n for a second time with intratrial interval w_2 by the anticipation method. If L_n is not anticipated perfectly on the second trial, after a short rest period, present the truncated list L_{n-1} in the same way. If L_{n-1} is not anticipated perfectly, present L_{n-2}, and continue this procedure until some L_k is anticipated perfectly. Whenever L_k is anticipated perfectly, so are $L_1, L_2, \ldots, L_{k-1}$. k is the immediate memory span for this situation. We denote k by $k = \Lambda(\mathcal{F}; w_1, w_2, T; L_1, L_2, \ldots, L_n)$ and say that k is the immediate memory span of $\mathcal{F}$ for L_k with respect to (w_1, w_2, T). k provides a measure of the field's capacity for learning lists of the simplest possible kind.

The determination of $\Lambda(\mathcal{F}; w_1, w_2, T; L_1, L_2, \ldots, L_n)$ for suitable $(w_1, w_2, T; L_1, L_2, \ldots, L_n)$ is a matter of practical significance in trying to assess the initial field structure of a given subject. It is, in particular, intuitively clear that knowing Λ for a sufficiently large set of vectors $(w_1, w_2, T; \{L_i\})$ will enable one to determine the field constants to an arbitrarily high degree of accuracy. In order to achieve a prescribed level of accuracy in these estimates, the number of vectors which we study becomes larger as the field's initial structure is taken to be increasingly inhomogeneous. A practical difficulty in using Λ is that the trials required to find Λ deform the initial field structure. If the initial field is not to be too strongly deformed by the Λ testing procedure, then the various lists, their lengths, presentation rates, etc. must be arranged so that the density of inputs to a given field point through time at a given presentation rate and for a given list position is as uniform as possible. $\Lambda = \Lambda(\mathcal{F}; w_1, w_2, T; \{L_i\})$ is a strongly varying function in each of its variables.

The study of serial learning becomes nontrivial only when lists L of the same type as the testing L_i are used whose length $\Lambda(L)$ is strictly greater than $\Lambda(\mathcal{F};w_1, w_1, T; \{L_i\})$. If $\Lambda(L) = \Lambda(\mathcal{F};w_1, w_1, T; \{L_i\}) + 1$, then the "middle" of a list will be very near its last point. If, on the other hand, $\Lambda(L) >> \Lambda$, then the list will often exhibit a broad middle, dynamically speaking, since the advantageous decay processes that occur when $P=p^*$ often do not propagate to the points of the middle until these points have already undergone their most significant strength interactions. If $\Lambda(L) >> \Lambda(\mathcal{F};w_1, w_1, w_1; \{L_i\})$ and the subject is required to repeat aloud all $\pi_i^{(e)}$ when they occur, it is not always true that learning shall occur. Broad strength scatter occurs practically everywhere over $\mathcal{F}$ and the entire list can soon behave like an indefinitely long middle. Even the initial advantage to the beginning of the list can easily be lost on successive trials since the absence of virtual points p^* prevents the repetition of this advantage on later trials. The initial advantage itself introduces some line channeling on successive trials, but as w_1 is taken increasingly small, even this advantage decreases on the first trial, and as $\Lambda(L)$ is taken ever larger, the line residue of the i^{th} trial will become increasingly homogenized by trial i+1.

25. Multiple Crossings and Field Extensions

This situation is in marked contrast with the two point field $\{p_1, p_2\}$ under $\{\ldots, \pi_1^{(e)}(t-h), \pi_2^{(e)}(t), \pi_1^{(e)}(t+h), \ldots\}$ for which, again, the field behaves like an indefinitely long middle, but whose list is simple to learn. Part of the reason for this simplicity lies in the renormalization of the dynamics of a large field to just two points, and the fact that p_1 (p_2) excitation is sure to excite p_2 (p_1). This is not the only reason. For consider the list 11010001101010000110. This list is also normalized to just two points: p_0 and p_1, but no one will claim that it is as easy to learn as 1010101010101010. Lists whose

items repeat themselves in nontrivial patterns introduce new considerations.

The reason for this is easily seen by comparing the diagrams

(α)

1010101010101010

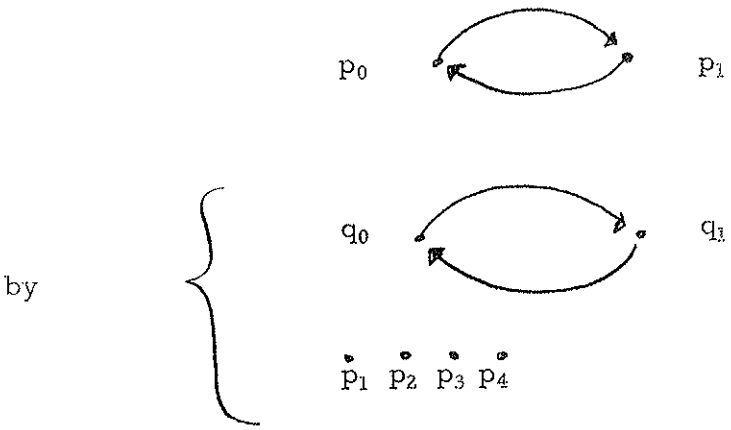
(β)

11010001101010000110

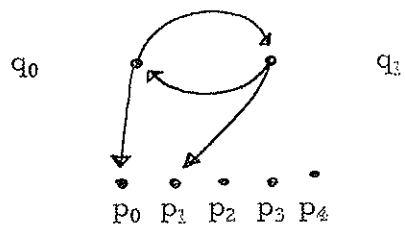
In these diagrams, $p_i \rightarrow p_j$ means that p_i precedes p_j somewhere in the list. The simplicity of 1010101010... is revealed clearly in (α), for p_1 always leads to p_0, and p_0 always leads to p_1, so that a cyclic line asymptote is possible. On the other hand, in (β), all possible orderings of p_0 and p_1 occur. In fact, the list 101100 has the same diagram as (β), but it is far easier to learn than the list of (β). (β) does not uniquely determine its list. We call a diagram like (β) a diagram with multiple crossings.

(β) can be more readily learned by learning the list (γ): 1211321111421, which is thirteen letters long instead of twenty letters long. The first 1 in (γ) means that the (β) list begins with 1. The first 2 of (γ) means that two 1's follow each other in succession in (β). The 1 after the first 2 in (γ) means that a single 0 follows the two 1's in (β). The 1 after 121 in (γ) means that a single 1 follows the single 0 following the first two 1's in (β). And so on. This shorter list (γ) uniquely determines the list of (β), but its point field is no longer $\{p_0, p_1\}$. It is at least as large as $\{p_1, p_2, p_3, p_4\}$.

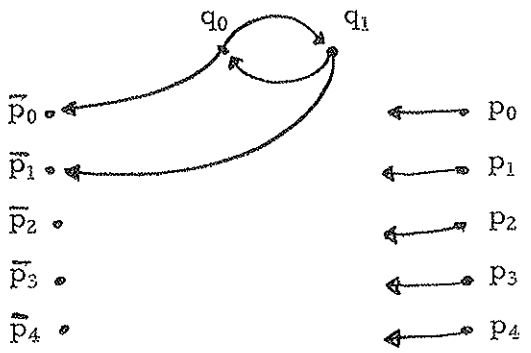
No experimental input ever arrives at $\{p_2, p_3, p_4\}$, however, so that this transformation from (β) to (γ) occurs entirely by the intervention of internal feedback inputs from higher-order fields. In fact, $\{p_1, p_2, p_3, p_4\}$ does not itself uniquely determine the (β) list. We need the additional rule that, after the first 1, the next letter in (γ) gives the number of 1's in a row in (β), the letter after that the number of 0's in a row, and so on. We therefore need an additional field $\{q_0, q_1\}$, disjoint from $\{p_i\}$, where excitation of q_0 (q_1) at a time when p_i is excited means that i repetitions of 0 (1) should be evoked. Our point field is therefore the cross-product of $\{p_1, p_2, p_3, p_4\}$ with $\{q_0, q_1\}$: $\{p_1, p_2, p_3, p_4\} \times \{q_0, q_1\}$. Only the joint excitation of some (p_i, q_j), along with the rules that $q_1 \Rightarrow q_0 \Rightarrow q_1$ and that we begin by exciting q_1 before q_0 completely specifies the list of (β). Thus we replace



when the p_i are all mutually connected. The excitation of q_0 (q_1) signifies that 0 (1) has been activated. If we draw



to represent this fact, then p_0 and p_1 receive excitations from q_i points and from p_j points, and the diagram becomes a degenerate representation of (β) once again. We therefore recall that $\{p_i\}$ refers only to the memory structure, while $p_{(r)}$ refers to the response structure. $p_{(r)}$ must be expanded into $\{\bar{p}_0, \bar{p}_1, \ldots, \bar{p}_4\}$, an extended set of response points, so that we can draw



Now when q_i is excited, it directly facilitates the response point $\bar{p}_i$ without interfering with the dynamics of $\{p_i\}$. Still further structure is required. For if (q_i, p_j) is excited, a mechanism must exist such that the first r_i excitation is accompanied by an internal feedback input to p_1, the second r_i is accompanied by an internal input to p_2, and so on until the i^{th} r_i is accompanied by an internal input to p_i. After this last internal input is delivered, the other q_j point must become excited and p_j excitation must be replaced by excitation of p_j's successor in list (γ).

It is apparent from these remarks that the smallest field which is capable of learning a list with multiple crossings is larger and more complicated than a simple consonant field, and requires control mechanisms that are not critical in simple consonant fields. The only way to avoid a language with multiple crossings is to use a very large vocabulary of disjoint points which initially possess a simple structural carrier that asymptotically decomposes into a

collection of linear or cyclic line structures. Such a field does not exhibit very subtle behavior, however. The most fundamental liability is exemplified by the fact that one could never be certain of saying even A-B-C-D-E and B-A-D at one's discretion, since in a consonant field if $c^*_{BC} \approx 1$, then $c^*_{BA} \approx 0$. Thus if one were sure of his capacity to say A-B-C-D-E, then he must replace B-A-D by B-C-D. An extension of a consonant field is therefore surely required, and this extension must, at all costs, feel comfortable in the midst of multiple crossings. Some of the fundamental principles needed for the construction of extensions of this kind will be given in the following pages.

26. Multiple Lines

To the present, the line structure that we have used has been of a very simple kind: given any ordered pair (p_i, p_j) of points, a directed line l_{ij} and terminal node N_{ij} have been drawn which carry a certain dynamical process. More complicated line structures are commonplace, and their effects are manifest in a variety of verbal learning situations. An important example of such structures is the following. Given any set $P = (p_{i_1}, \ldots, p_{i_k})$ of points and a point p_m, draw emerging from each point of P a directed line which joins with all directed lines so drawn in a common line that terminates in p_m. That is, we draw a fan of lines from P leading to p_m (diagram 8). Call this line l_{Im}, where $I = (i_1, \ldots, i_k)$. l_{Im} is an example of a multiple directed line. From this point of view, each l_{ij} is a simple directed line. Let l_{Im} terminate on the multiple node N_{Im}. To each l_{Im}, associate constants A_{Im} and p_{Im}, and a function c_{Im} very much as in the simple case. Require that the strength transmitted through a multiple line l_{Im} and received at p_m at time t be proportional to

$$\left(\prod_{k\in I} s_k(t - t_{km})\right) p_{Im} \; \tilde{c}_{Im} \;,$$

where $\tilde{c}_{Im}$ is as before some function of the line structure that is yet to be determined. Let the proportionality constant be $r_I = r(|I|)$, where $|I|$ is the number of elements in I, and let $r_{(p_i)} = r(1) = r$. This rule means that the strength transmitted through a multiple line from the various points interacts within the multiple node in a multiplicative way before transmission to the endpoint is completed. When $I = (p_i)$, the transmitted strength is computed according to the old rules for simple directed lines. For a multiple line structure,

$$T_i = \sum_I \left[r_I \left(\prod_{k\in I} s_k(t - t_{ki}) \right) p_{Ii} \tilde{c}_{Ii} \right] \;,$$

$$\hat{T}_{Ij} = r_I \left(\prod_{k\in I} s_k(t - t_{kj}) \right) p_{Ij},$$

$$D_{Ij} = \epsilon c_{Ij} \;,$$

$$\hat{E}_{Ij} = \rho_I \cdot \prod_{k\in I} \left(M_k - s_k(t - t_{kj})\right) p_{Ij}$$

$$E_{Ik} = \mu (A_{Ik} - c_{Ik}) \;, \quad \text{and so on,}$$

so that the embedding field equations for a multiple line structure are

$$\frac{ds_i}{dt} = E_i(T_i + I_i^{(e)} + I_i^{(f)} + I_i^{(r)}) - D_i, \quad i = 1, 2, \ldots, n,$$

and

$$\frac{dc_{Ik}}{dt} = E_{Ik}\hat{T}_{Ik}\hat{T}_{kk} - D_{Ik}\hat{E}_{Ik}\hat{E}_{kk}, \text{ all } I \text{ and } k .$$

Now choose $r_I = r^{|I|}$ for specificity, so that the various branches of the multiple line l_{Ij} combine independently, and let $A_{Ij} \leq A_{kj}$ whenever $k \in I$.

27. Propagating Symmetries and Hierarchies of Nonlinear Averages

Intuition into the behavior of a multiple line is not difficult to acquire. Let a quiescent strength field and a symmetric initial line structure be given: All $s_i(0) \approx 0$; $A_{ij} = A(1 - \delta_{ij}) + B\delta_{ij}$; $0 \leq c_{ij}(0) = c$, $i \neq j$; $0 \leq c_{ii}(0) = c_{jj}(0)$; $0 \leq c_{Ik}(0) = c_{Jr}(0)$ whenever $|I| = |J|$ and $r \in J$ if and only if $k \in I$; etc. Now deliver an input to a point p_j. During an initial period of transmission, the strength transmitted through all non-simple lines will be practically zero, since $s_j \approx 0$, $j \neq i$. Once the s_j begin to grow and the p_j to transmit, all lines will contribute transmitted strength to their recipient points. Due to the symmetry of the structure, however, the dynamical behavior of all s_j, $j \neq i$, and all c_{Ik}, $k \neq i$, $i \notin I$, $|I| = \text{const.}$, will be identical. Similarly for the c_{Ii}, $i \in I$, $|I| = \text{const.}$, the c_{Ii}, $i \notin I$, $|I| = \text{const.}$, and the c_{Ik}, $k \in I$, $k \neq i$, $|I| = \text{const.}$

Given any set F of functions, let F be partitioned into maximal subsets F_i such that every function in F_i has an equal initial value, for all i. Whenever the functions of F_j also have equal values throughout the time interval $[0, T]$, we say that the partition $F = F_1 \oplus F_2 \oplus \ldots \oplus F_n$ propagates in $[0, T]$. If a partition propagates in $[0, \infty)$, we say simply that the partition propagates. In the present situation, the partitions into maximal subsets of the strength and line functions do not propagate, but they do propagate in $[0, T]$, where T is the onset time of the input to p_i. At time T, we refine the partition of strength and line functions into a larger partition based on whether or not the index i

occurs in the functions of a given set of the old partition. This partition then propagates in $[T, T']$, where T' is the onset time of the next input to the system. New inputs thus introduce asymmetries into the propagating sets. Once these asymmetries are taken into account by refining the old partition, the symmetries of the new partition propagate until another asymmetry is introduced into the system by a new input. Such a combining into propagating internally symmetric classes of the various embedding functions is a common phenomenon, which is reminiscent of the probabilistic process of taking conditional expectations. The analogy is so strong that we call this successive refinement of propagating sets the conditioning process.

It suffices to study one function from each propagating set. In the present example, we must consider one s_j, $j \neq i$, s_i, and one c_{Ik} from each of the four classes of c functions for each fixed $|I|$, $|I| = 1, 2, \ldots, n$. We observe the following useful facts about these functions: $c_{Ik} \leq c_{Jk}$ whenever $I \supset J$, just so long as $M_i \leq 1$ for all i. $c_{Ik} \leq c_{Ii}$, since s_i is generally greater than s_k. The symmetry of the simple line growth for the c_{kj}, k, $j \neq i$, and so on, will not be disturbed by multiple activity since the entire line structure is assumed to be initially symmetrical, and $\pi_i^{(e)}$ introduces the only source of asymmetry. Under a general input sequence, however, an asymmetry in a function p_{Ik} or c_{Ik} can offset symmetries in the simple line structure that might have propagated under the input sequence if no non-simple line structure existed. The strength transmitted to p_k through the multiple line l_{Ik} will influence the behavior of s_k and thus of all (say) c_{uk}, $u=1, 2, \ldots, n$, in a manner patterned after the asymmetry. Future isolated inputs to a p_u will be distributed through the simple line structure $\{c_{uk}: k=1, 2, \ldots, n\}$ even if the multiple line structure is never non-trivially activated. An asymmetry in the multiple line structure can hereby be carried to the simple line structure, which is capable of later propagating this asymmetry even in

the absence of the multiple structure. This remark illustrates the general tendency for multiple and simple line structures to induce similar configurations in one another. The range of sensitivity of the two types, or rather the hierarchy, of line structures is not uniform, however.

The c_{Ik} for increasingly large $|I|$ provide residues of increasingly global patternings of past strength distributions. The $c_{Ij}(t)$ are nonlinear averages which measure how the dynamical behavior of the larger I subsets of the point field affect the behavior of the individual points during their excitation periods up to time t. As $|I|$ increases, it is instructive to view the $c_{Ij}(t)$ as the higher-order densities representing these non-linear interactions at time t; indeed, the analogy of these functions to higher-order correlation functions is clear. From this perspective, one notices the possibility of considering the c_{Ij}'s for large $|I|$ as recursively defined nonlinear functionals of the c_{Jk}, $|J| < |I|$. Such a representation is not natural in the present case, however. For all the c_{Ij} functions modulate a single strength field $\mathcal{S} = \{s_i\}$ whose points are of a uniformly simple type and are not themselves hierarchically arranged. Any attempt to define the c_{Jk}'s recursively would necessarily weaken this direct nonlinear bond with the simple strength field, whence the resulting, weakened field equations, whatever their exact structure, could only hold in the distribution sense relative to a fairly small class of test functions. By exhibiting the strength field explicitly one can describe the embedding process not weakly, but in a wholly deterministic manner. By building over a uniform dynamical field a veritable stack of interaction structures for weighing and recording past activities and guiding, according to this record, the evolution of future activity, we provide ourselves with an exquisite example of strong nonlinear binding.

That the strength field is always nonhierarchically organized is not a universal embedding phenomenon. Whenever $\mathcal{S}$ can be decomposed into locally homogeneous levels $\mathcal{S}_1, \mathcal{S}_2, \ldots, \mathcal{S}_n$, however, the interaction structures must be catalogued according to the level of strength

field from which they lead and to which they transmit. Under no circumstance can such strength fields be eliminated and replaced by a recursive definition of the c_{Ij}'s viewed as non-linear functionals operating between points, or states, which serve merely as passive geometrical carriers of interactions with no local dynamical structure of their own.

28. $ABC \rightarrow D \neq C \rightarrow D$

The psychological issue that is raised by the existence of multiple line structures is illustrated in the following question: Why is it that the sequence of inputs ABC almost invariably brings to mind the letter D, while C presented alone, especially under auditory delivery, has comparatively variable effects? An approximate answer to this question is simple from the present viewpoint.

Let p_A, p_B, p_C, and p_D be the points corresponding to A, B, C, and D, respectively. As a result of past practicing on the alphabet, $c_{(ABC)D}$, $c_{(AB)D}$, $c_{(AC)D}$, and $c_{(BC)D}$, in addition to c_{CD}, have achieved high values. Since presenting C corresponds to an input to p_C alone, one-step strength transmission can proceed along little more of the line structure than its c_{CD} component. On the other hand, the delivery of inputs successively to p_A, p_B, and p_C that corresponds to the presentation ABC causes all of the functions s_A, s_B, and s_C to have fairly large values simultaneously during a critical time interval following the presentation. Thus all of the relevant multiple lines, including the simple lines, will transmit an appreciable amount of strength to p_D. s_D will consequently grow to a much higher asymptote than for the C presentation alone. D will be correspondingly more available for response.

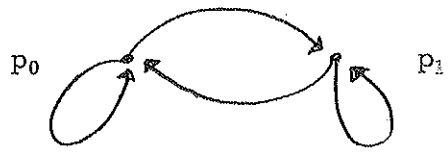
Similarly, it is clear that the sequence ABCDE will be little more effective than CDE in evoking F, since the transmitted

strength values corresponding to p_A and p_B will often have decayed almost completely before the input to p_E can generate an appreciable transmitted quantity, whence $\prod_{k \in (ABCDE)} s_k$ will always be small. Thus the multiple lines $l_{(ABCDE)F'}$ $l_{(B\ldots E)F'}$ $l_{(ACDE)F'}$ etc., shall transmit practically no strength to augment s_F over and above the values transmitted through the $l_{(CDE)F'}$ $l_{(DE)F'}$ etc. The $c_{(ABCDE)F'}$ etc., values shall therefore be small. Note further that this remark is independent of the special choice $r_I = r^{|I|}$. $r_I = r$ would have served just as well.

All of these effects have of course been found in serial learning data. In classical psychological language, one might refer to the above as phenomena induced by "compound stimuli." It should be clear from our discussion, however, that the concept of a "stimulus" can effectively be defined only be a very delicate consideration of temporal factors. Thus, if C is presented alone, C is the stimulus. If BC are presented in quick succession, B, C, and BC are in some sense all stimuli. But if B and C are presented at highly disparate times, C is more of a stimulus than B. And if ABCDE are presented even rather quickly, ABCDE will hardly act as a stimulus at all, even though CDE is a very effective one. It would thus appear that the term becomes increasingly unwieldy, and even misleading, as more information about the process is obtained. It is partly for this reason that the noncommital term "input" has been used from the start in this paper. It should be observed, however, that a similar criticism does not yet apply to the term "evoked response". The difference is simply that external "stimuli" are transformed within the neurological structure in a way that is largely hidden from the measuring devices of contemporary investigators, while at least in simple verbal learning, evoked responses can be recognized and recorded without difficulty.

29. Multiple Lines Distinguish Multiple Crossings

The existence of multiple lines also provides some insight into multiple crossings. In particular, in comparing ABCDE with BAD, we see that multiple lines from A, B, and C generate D in the first case, while lines from B and A generate D in the second case. The critical observation is that new sets of lines are activated in different multiple crossings and the total magnitude of the transmission over these newly incorporated lines depends sensitively on such temporal factors as the ordering of items. Thus, although diagrams like



do not uniquely determine their list in a simple consonant field, in a field with multiple lines, the various list possibilities corresponding to a given diagram are dynamically distinguished one from the other.

A convenient way to express this fact is to consider Euclidean $n(2^n+n+1)$ space, $\mathbb{R}^{n(2^n+n+1)}$. $\Omega(t) = (s_1(t), \ldots, s_n(t), c_{11}(t), \ldots, c_{1n}(t), \ldots, c_{nn}(t), c_{I1}(t), \ldots, c_{Jn}(t))$, for fixed t, is a point in $\mathbb{R}^{n(2^n+n+1)}$, and $\Omega(t)$ traces out a curve in $\mathbb{R}^{n(2^n+n+1)}$ parameterized by t. The curve generated by a list when all $c_{Ii}(0)=0$, $|I| \geq 2$, corresponds to the case of a simple consonant field. One can find several lists with the same diagram which generate curves $\Omega(t)$ that are very close to each other in $\mathbb{R}^{n(2^n+n+1)}$ norm. When $c_{Ii}(0) > 0$ for many $|I| \geq 2$, however, it is much

harder to find several lists with the same Ω curve in $\mathbb{R}^{n(2^n+n+1)}$. Letting $\|(x_1, \ldots, x_n)\| = \Sigma_i |x_i|$ and $|||x(\cdot)|||_t = \int_0^t \|x(w)\| dw, t > 0$, we see that variation of the initial value vector $(c_{I1}(0), \ldots, c_{Jn}(0))$ induces a splitting in $|||\cdot|||_t$-norm of the Ω curves. In fact--and this is a crucial point--variations in $(c_{I1}(0), \ldots, c_{Jn}(0))$ induce a splitting in $|||\cdot|||_t$-norm of the projection of Ω onto its first n coordinates, $\Omega_n = (s_1, \ldots, s_n)$. By seeking ever richer line structure possibilities, we can split the possible Ω so completely that no two clearly distinct input sequences share a common Ω. Multiple crossings become distinguishable in this way.

30. $\tilde{c}_{Ik}$ = ? Renormalizing Fields and the Sharpening of Strength Distributions

A determination of the functions $\tilde{c}_{Ik}$, $|I| \geq 2$, shall now be made. When all $I = (i)$, the choice $\tilde{c}_{ik} = c^*_{ik}$ will be recalled. For situations which possess nontrivial multiple lines, however, even the choice of lines which form a normalizing class comparable to the sums $\Sigma_k c_{rk}$ in $c^*_{ri} = c_{ri}/\Sigma_k c_{rk}$ is ambiguous; the rule "normalize with respect to all lines emerging from the same point" simply has no easily discernible meaning in the multiple case. Indeed, this is not surprising, since the original choice of normalization was merely a convenient artifice by which to deal with certain special empirical situations. The most striking property of this artifice is that the total strength emitted from a given point at any time is distributed to the rest of the point field in such a way that the effects of

the transmission over a given directed line on the recipient point are not independent of the transmission to all points that emerge from a fixed locus. The function c^*_{ik} roughly describes this dependence, and gave valuable qualitative insights into the possibility of achieving such asymptotes as the linear asymptote $c^*_{i,i+1} = 1$, $i \leq n-1$, in the serial case, into the futility of too strongly distinguishing "all-or-none" and "continuous" learning effects, and so on. The fact that the mapping $c_{ik} \rightarrow c^*_{ik}$ transforms the transmitted values in l_{ik} by a normalization before they actually generate strength in the recipient point is somewhat unnatural, for it has no geometrical counterpart within the structural carrier of the field.

The difficulty lies still deeper. For every situation in which the normalization has appeared, the transmission time from any point to its receiving point has always been v, apart from the practically zero time required for self-excitation. Our success in normalizing across points was due to the fact that the dynamical events occurring at all of the nodes N_{ij}, $j \neq i$, at any given time were strongly controlled by a single event occurring v time units earlier at p_i. The success of the normalization was thus based both geometrically and dynamically on homogeneity restrictions on the field. Since we considered only input sequences which were highly regular, these homogeneity assumptions were not brought glaringly into focus, as they would have been under a very inhomogeneous array of inputs.

The choice $\tilde{c}_{ki} = c^*_{ki}$ is thus inadequate in the general case. It is not defined in a natural way for multiple lines and it loses its rationale as a homogeneous approximation for general choices of transmission time lags and of input distribution. The problem therefore poses itself of finding some substitute for the

$\tilde{c}_{ki} = c^*_{ki}$ choice which preserves the most desirable properties of this choice and which simultaneously has an intrinsic meaning in a general embedding field. The first change which is forced upon us is that $\tilde{c}_{Ii}$ must be a function of c_{Ii} alone. This requirement is obviously imposed by the geometry of the lines. In fact, we make the simplest possible choice: $\tilde{c}_{Ii} = c_{Ii}$.

How can the normalization condition be recaptured for this choice? The old condition $\tilde{c}_{ki} = c^*_{ki}$ involved a normalization of the strength transmitted by a given point before the strength reached its recipient points. For the new choice $\tilde{c}_{Ii} = c_{Ii}$, it is obvious that this normalization must be carried out after the transmitted strength reaches its recipient points. We must therefore envisage a new structure, sensitive to the dynamical events at every point, which is capable of carrying out this normalization. The behavior of this structure must be describable in terms of some sort of field equations. In particular, 1) the given embedding field $\mathcal{7}$ must be extended by the addition of new points ω, (2) these ω must possess some analog of a strength function, and (3) the lines connecting $\mathcal{7}$ with ω must allow momentary fluctuations in the strength distribution of $\mathcal{7}$ to alter the strength functions of ω in such a way that the reciprocal influence of the transmitted strength from ω to $\mathcal{7}$ will behave like a normalization of the strength distribution of $\mathcal{7}$. The simplest example of such an extension is the following one.

Augment the structural carrier with a single renormalization point p_W and to each set $\{p_i, p_W\}$ associate the pair l_{iW}, l_{Wi} of directed lines (diagram 9). As before, these lines shall be the carriers of transmitted quantities. Define constants M_W, p_{iW}, and p_{Wi} with the usual significance and let the renormalizing strength functions w_i be determined by:

$$dw_i/dt = \lambda (M_W - \Sigma_k w_k) (\theta p_{iW} s_i(t-t_{iW})) - \delta w_i ,$$

where $0 < \theta \leq 1$, and $0 < t_{iW} << \min_{k \neq i} t_{ik}$. Let the internal input from p_W to p_i be given by

$$I_i^{(in)} = \hat{\theta}\, w_i(t - t_{Wi})\, p_{Wi},$$

where $\theta \approx \hat{\theta}$, $p_{Wi} \approx p_{iW}$, and $t_{Wi} \approx t_{iW}$. This definition of $I_i^{(in)}$ is our first example of an internal input function for the original field $\mathcal{F}$ whose source lies in another embedding structure.

The total embedding space M_W of p_W can come into the service of any p_i. All of the p_i compete for M_W, as we see from the definition of the effective embedding space of p_W, $E_W = \lambda(M_W - \Sigma_k w_k)$. s_i is transmitted from p_i to p_W as $\theta\, p_{iW} s_i(t - t_{iW})$. w_i is carved out of the effective space E_W and is transmitted to p_i as $\hat{\theta}\, w_i(t - t_{Wi})\, p_{Wi}$. The interaction of $\mathcal{F}$ with p_W is thus of a reverberatory character. The effectiveness of a given $s_i(t)$ at generating a large $I_i^{(in)}$ depends strongly on the distribution of all s_j up to time t. For example, given a fixed function s, $0 < s < \min_i M_i$, $I_j^{(in)}$ will be larger if $s_j = s$ and $s_k = 0$, $k \neq j$, than if, say, $s_j = s_m = s$ and $s_k = 0$, $k \neq j, m$.

Imagine that $\pi_1^{(e^j)}(t)$ occurs in a quiescent strength field. s_1's growth will be augmented by reverberation with w_1. We suppose that M_W, p_{iW}, p_{Wi}, etc., are chosen so that when $\Sigma_k w_k$ is small and only s_1 is large, s_1 can generate a reverberatory internal input function of the order of magnitude of the largest transmitted strength input generated within $\mathcal{F}$ by s_1. We also restrict the sizes of M_W, etc., in such a way that when many points in $\mathcal{F}$ simultaneously have large strength values, their reverberatory inputs from p_W will all be greatly reduced. A convenient measure of this reduction in a special case is the following.

Fix a standard external input function I and field parameters $M_i = M$, M_W, p_{ij}, p_{kW}, etc., in such a way that $\mathcal{F}$ is initially homogeneous, that $p_{iW} = p_{Wi} = p_{Wj}$, etc. Set all initial strength values

equal to zero and all initial c_{ij} values equal to γA_{ij}, where γ is a fixed constant in $(0, 1)$. Deliver I at t=0 to any k points in $\mathcal{I}$. Measure the maximum ω_k of w_i for any one of these points. Define

$$\mathcal{R}_k(I) = \frac{\omega_k}{\omega_1}$$

$\mathcal{R}_k(I)$ measures the decrement in reverberatory inputs resulting from the multiplicity k of the simultaneous input array. Another measure is

$$\bar{\mathcal{R}}_k(I) = \frac{\bar{\omega}_k}{\bar{\omega}_1}$$

where

$$\bar{\omega}_k = \int_0^\infty w_i(t)dt$$

and p_i is one of the points receiving an input. Choose one of these measures and interpret the above remarks on reverberatory decrement in terms of it. Also let the recovery rate of the various w_i be sufficiently rapid under $\pi_1^{(e)}(t)$ that M_W will not be too strongly exhausted by w_1 growth when transmitted strength from p_1 begins to pass through the various nodes N_{ij}.

With these restrictions on p_W and its field in mind, suppose that initially c_{1j}, $j \neq 1, k$, are of equal size, that $c_{1k} > c_{1j}$, $j \neq 1$, and that $t_{1m} = v > 0$, $m \neq 1$. Let $\pi_1^{(e)}(t)$ occur in a quiescent strength field. As transmitted strength from p_1 begins to reach the other p_j, s_k will early majorize the s_j, $j \neq 1, k$. A rapid reverberation with p_W will increase this advantage while p_k is still receiving transmitted strength. As p_k rapidly carves out the greater share of M_W, the reverberatory internal inputs to the p_j, $j \neq 1, k$, will become increasingly small, and their s_j values will be comparably decreased relative to s_k.

This effect closely resembles that achieved when $\tilde{c}_{ki} = c^*_{ki}$. For (1) nonuniformities in the set $\{c_{1j}: j \neq 1\}$ lead to parallel nonuniformities in the induced $\{s_j: j \neq 1\}$ over and beyond those expected by simple c_{ij} transmission, and (2) the effect of the total transmitted strength from p_1 on new strength growth in $\mathcal{T}$ will be modulated by the state of a single source, the total pool M_W, according to the size of the transmitted value from p_1, just as the c^*_{1j} values modulated the strength transmitted from the single source p_1. Also, (3) if $c_{12}(0) = c_{13}(0) = \bar{c} > c = c_{14}(0) = c_{15}(0) = \ldots = c_{1n}(0)$ and $\pi_1^{(e)}(t)$ occurs, s_4 will be smaller after transmission from p_1 than if $c_{12}(0) = \bar{c} > c = c_{13}(0) = c_{14}(0) = \ldots = c_{1n}(0)$ even though, restricting attention to $\mathcal{T}$, p_4 receives an equal amount of excitation from p_1 in both cases. c_{14} growth in the first case will be less than c_{14} growth in the second case as a result. This effect is the analog of the competition between the p_j to achieve a $\tilde{c}_{ij} = c^*_{ij}$ growth determined by the relative rates of growth of the c_{ij}. It is the central fact underlying the possibility of achieving linear asymptotes and "all-or-none" effects in the simple consonant field. It reveals a strong tendency for the larger strength functions to be accentuated while the smaller strength functions are suppressed. We call such a tendency the sharpening of the strength distribution.

31. Introduction to Transmission Thresholds

The use of renormalizing points also gives rise to entirely new possibilities. Suppose that $t_{1k} < t_{1j}$, $j \neq k$; $k, j \neq 1$ and let $\pi_1^{(e)}(t)$ occur. Transmitted strength flows from p_1 to the p_j in such a way that s_k will begin to grow before the s_j, $j \neq k$, do. s_k will begin reverberating with p_W before the s_j can, and by the time the s_j values are large enough to generate sizeable internal inputs, E_W will already be partially depleted by p_k. Again the s_j, $j \neq k$, values will be relatively small. Suppose that provisions exist to hasten transmission over lines whose $\{c_{1k}, k \neq 1\}$ values are relatively large. Then transmission over these c_{1k} will

generate s_k's that deplete M_W before other s_j begin to reverberate with p_W. The sharpening of s_k is hereby facilitated even more than in the case $t_{1k} = v$, $k \neq 1$. The field before us can generate renormalizations based on temporal facilitation only if we let $t_{ik} = g(c_{ik}; \alpha_1, \alpha_2)$, where α_1 (α_2) is a vector of geometrical (input) parameters, and g is a monotone decreasing function of c_{ik}. A more intrinsic way to provide for temporal facilitation of this kind is to introduce a nontrivial transmission threshold, τ.

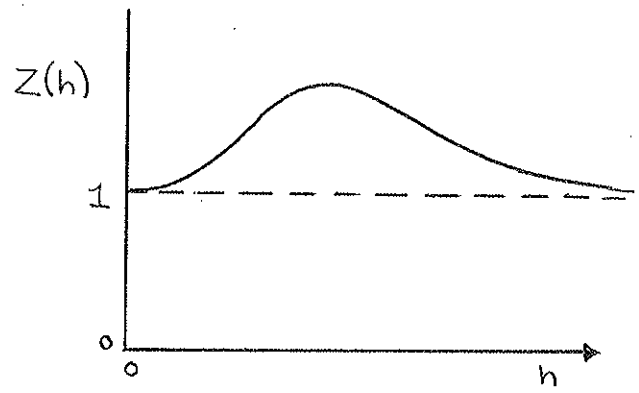
By this we mean that p_j does not transmit to p_W until $s_j \geq \tau > 0$. If $c_{1k} > c_{1j}$, s_k will exceed τ before s_j does, which allows us to draw the same conclusions as we did when $t_{1k} < t_{1j}$, with three improvements: (1) t_{1k} need not be taken as an ad hoc function of c_{1k}, (2) a threshold has an intrinsic dynamical meaning, and (3) the time required for s_j to exceed τ varies inversely with the degree of excitation of p_j. Thresholds provide a continuously modulated kind of temporal facilitation, sensitive to field activity, that is far more subtle than the merely geometrical condition $t_{1k} < t_{1j}$. Such thresholds will be studied in a later section. Now it suffices to observe that once renormalizing field extensions are allowed, we can construct geometrical carriers whose dynamical behavior is self-improving.

32. Introduction to Temporal Masking

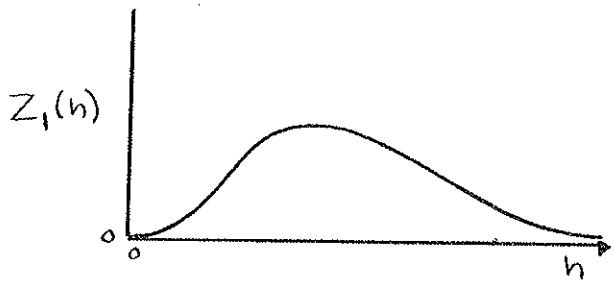
The next example shows that ongoing field activity can suppress the dynamical effects of new external perturbations. Let $(\mathcal{L}_i; \pi)$ denote the maximum of s_i achieved under the input regime π. Deliver $\pi_1^{(e)}(t)$, $\pi_2^{(e)}(t+h)$ in rapid succession. If w_1 is still large when s_2 begins to grow, then s_2's growth will be strongly curtailed by the paucity of internal inputs to p_2. Thus $(\mathcal{L}_2; \pi_1^{(e)}(t), \pi_2^{(e)}(t+h)) << (\mathcal{L}_2; \pi_2^{(e)}(t+h))$. This is one sense in which the event $\pi_2^{(e)}(t+h)$ hardly occurs at all, dynamically speaking, when it immediately follows $\pi_1^{(e)}(t)$. Indeed, a

negligible record of $\pi_2^{(e)}(t+h)$ shall be left in the residual line structure, since s_2 is completely overshadowed by s_1, and future inputs shall be distributed over the field lines almost as if $\pi_2^{(e)}(t+h)$ had never occurred. We say that $\pi_1^{(e)}(t)$ temporally masks $\pi_2^{(e)}(t+h)$. The analogy between this masking effect and the "masking" of errors in situations revealing "all-or-none" effects should not be overlooked.

The temporal masking of $\pi_2^{(e)}(t+h)$ by $\pi_1^{(e)}(t)$ depends sensitively oh h. When h=0, neither of the two events will have an advantage over the other, although both will generate smaller strength functions. As h is taken increasingly large, the disadvantage of $\pi_2^{(e)}(t+h)$ will first increase, since p_2 will be faced with a small E_W when s_2 is taking its largest values. We can, however, take h so large that E_W has recovered from $\pi_1^{(e)}(t)$ depletion before time t+h. Letting $Z(h) = (\mathcal{Q}_1; \pi_1^{(e)}(t), \pi_2^{(e)}(t+h))/(\mathcal{Q}_2; \pi_1^{(e)}(t), \pi_2^{(e)}(t+h))$, we therefore have a curve of the form



Letting $Z_1(h) = Z(h)-1$, this graph becomes



$Z_1(h)$ is one measure of the relative masking of p_2 by p_1. When $h=0$, both p_1 and p_2 are equally masked, and $Z_1(h)=0$. Another useful measure of relative masking is given by replacing $(\delta_i;\pi)$ by $(\int_0^\infty s_i(t)dt;\pi)$.

33. Intrinsic Renormalizability

The augmentation of 7 by p_W has demonstrated the possibility of introducing normalizations of transmitted quantities into the field in an intrinsic way. This normalizing property of the field is so important that we give it a name: the intrinsic renormalizability of embedding fields. The introduction of thresholds and temporal masking are not the only important ideas which are derivatives of intrinsic renormalizability. All processes involving attention, differential recognition thresholds and reaction times, the adjustment of motivational states, etc., depend critically on some aspect of this general psychological principle. Much of the remainder of this paper shall be devoted to the presentation of increasingly skillful constructions of fields which exhibit the intrinsic renormalizability property.

34. Blocking in p_W

Letting $w = \Sigma_k w_k$,

$$dw/dt = \lambda(M_W - w)(\theta \Sigma_k s_k(t-t_{kW})p_{kW}) - \delta w.$$

By analogy with the p_i, w may be called the strength function of p_W, with input function $\theta \Sigma_k s_k(t-t_{kW})p_{kW}$. By contrast with an l_{ij}, the line structure involving l_{Wi} does not partake of the total transmittable w, but only of that portion w_i that has been carved from the available E_W by transmitted point strength from p_i. l_{Wi} has its

source in a variable subregion of p_W of total extent w_i and it is this subregion which reverberates with p_i. The size of this subregion does not change in perfect unison with individual p_i activity, for the w_i are subject to growth and decay lags. No term of the form $(M_W - w)w_i$ is included in the differential equation for w_i, corresponding to the assumption that the subregion of p_W of size w_i is wrought from M_W entirely by incoming strength from p_i. The effects of internal excitatory lines $p_{WW}c_{WW}$ are assumed to be of negligible proportions relative to the rate of uptake of M_W regions by direct transmission from 7. p_W thus represents a structure whose internal parts are not so finely scupted as those of 7. After some p_i activates a subregion of M_W, this region is subsequently blocked from activation by any other p_j until the original activation by p_i decays. A description of the details of this blocking process are impossible to see in p_W, since all of the finer dynamical features of processes are lumped together whenever a single point is used to represent an extended region.

The extension of 7 by p_W does suggest an interesting heuristic comparison nonetheless. Mammalian brains abound in collections of neural material in all levels of local organization. Neocortex is often arranged in sheets of well-developed cellular columns, while subcortical nuclei, such as thalamic nuclei, exhibit much more profusely branched internal fiber tracts. Cortex and thalamus are also intimately connected by dense fiber tracts. From our discussion of the properties of 7 and p_W, it is more likely than 7 be closely related to cortical behavior and p_W to thalamic behavior than vice versa. If this is true, then we should in particular, seek a blocking mechanism in the thalamus that is induced by neural excitation transmitted to the thalamus from the cortex. Much more would be true if such an analogy could be rigorously established, of course. Every theoretical conclusion drawn from the appropriate abstract embedding field would carry over to specific regions of the brain.

This analogy between neural and embedding structures will be tightened as we continue to extend $\mathcal{F}$.

35. Lateral Inhibition in Renormalizing Fields and in the Thalamus

A necessary first step in this extension process is to replace p_W by a renormalizing field with more than one point. If $\mathcal{F}$ has n points, it is natural to replace p_W by n points $\{q_i : i=1, 2, \ldots, n\}$. It requires little more than reflexes now to decide to endow these points with a line structure $\{\hat{l}_{ik}\}$ and transmission latencies $\{\hat{t}_{ik}\}$ of their own. Continue by interconnecting each pair (p_i, q_i) with bidirectional lines. Aiming for initial simplicity, let the transmission times between each such pair be the same. This symmetry requirement is especially appropriate when $\mathcal{F}$ is taken to be initially homogeneous. Call the field of the q_i $\hat{\mathcal{F}}$.

How can renormalization be accomplished by an n point field like $\hat{\mathcal{F}}$? It is clear that a desirable minimal condition is the following: Let $\pi_i(t)$ be a representative input event to p_i alone. Let $\pi_i(t)$ occur when all $s_j(t)=0$. Consider the set

$$\Omega_i = \{\omega : s_i(\omega) < (\mathcal{S}_i; \pi_i(t)),\ \omega \geq t\}$$

Assuming that $\pi_i(t)$ is chosen so that s_i has only one maximum, let $V(\pi_i)$ be the length of the interval Ω_i. The condition

$$2T + \max_{i,k} \hat{t}_{ik} < V(\pi_i) \qquad (\alpha)$$

is one kind of prerequisite for renormalizability.

The desirability of a condition of this type may be seen by again letting $\pi_i(t)$ occur when all $s_j(t) = 0$. s_i will be transmitted to the other points of $\mathcal{F}$, which will begin to reverberate with $\hat{\mathcal{F}}$. Condition (α) assures that the strength functions of $\hat{\mathcal{F}}$ will interact with one

another by transmission within $\hat{\mathcal{F}}$ and thereupon renormalize the strength field of $\mathcal{F}$ by reverberation while the p_j are still receiving $\mathcal{F}$-induced transmissions. $2T + \max_{i,k} \hat{t}_{ik}$ is the least majorante of the total time required to transmit strength from a point in $\mathcal{F}$ to a point in $\hat{\mathcal{F}}$, thence to another point in $\hat{\mathcal{F}}$, and finally back to $\mathcal{F}$. Similar considerations generate a requirement like $\hat{t}_{ik} < t_{ik'}$ for otherwise the renormalizations induced by even local perturbations of $\mathcal{F}$ would involve large segments of $\mathcal{F}$. If conditions like these are not satisfied, the renormalizability pattern will be quite chaotic. It is most important when studying the dynamical evolution of a field to properly set the relative velocities and rates of growth and decay within distinguished subfields of the field.

Now that the geometry of $\hat{\mathcal{F}}$ is relatively well defined, it is appropriate to inquire into $\hat{\mathcal{F}}$'s dynamical structure. Recall that in the case where $\hat{\mathcal{F}}$ consists of one point, the effect of producing a high s_i value in a quiescent field is to induce a growth in w_i. w_i thereupon blocks the growth of all other w_j be exhausting E_W. In the present situation, this blocking effect can only be achieved if the activity of a given q_i, when transmitted to the q_j, $j \neq i$, suppresses the activity in these q_j. The extent of this suppression must parallel the degree of activation of q_i. Since the strength transmitted within the $\hat{I}_{ij}$ is a good measure of q_i's activation, we expect that increasing strength transmission from q_i to q_j will increase the suppression of q_i's activity. Such a suppression process is called <u>lateral inhibition</u>. A useful system exhibiting such inhibition which arises directly from the old embedding equations is

$$\text{(i)}\quad ds_i/dt = \alpha(M_i - s_i)(r\Sigma_j s_j(t-t_{ji})p_{ji}c_{ji} + \theta\, w_i(t-\hat{T}_i)\hat{p}_{ii} + I_i) - \beta s_i,$$

$$\text{(ii)}\quad dw_i/dt = \hat{\alpha}(\hat{M}_i - w_i)(\hat{r}w_i(t-\hat{t}_{ii})\hat{p}_{ii} + \hat{\theta}\, s_i\,(t-\breve{T}_i)\,\breve{p}_{ii})$$

$$- \hat{\beta}\, w_i(1 + \gamma\Sigma_{j\neq i}\, w_j(t-\hat{t}_{ji})\hat{p}_{ji}),$$

with the usual equations for the c_{ij}'s. w_i is the strength function of q_i and the "$\wedge$" distinguishes symbols of $\hat{7}$ from the associated symbols of 7. $\hat{p}_{ii}(\breve{p}_{ii})$ is the p value for the line from q_i (p_i) to $p_i(q_i)$, and $\hat{T}_i(\breve{T}_i)$ is the transmission lag for $\hat{7}\rightarrow 7(7\rightarrow\hat{7})$ transmission. The term

$$\text{(iii)} \qquad -\hat{\beta} w_i(\gamma \Sigma_{j\neq i}\, w_j(t-\hat{t}_{ji})\hat{p}_{ji})$$

in (ii) contributes actively to the decay of w_i, in addition to the spontaneous decay term $-\hat{\beta}w_i$, and increasing the transmitted excitation form q_i to q_j does increase the suppression of w_i. In the excitation term

$$(M_i - s_i)\,(r\Sigma_j\, s_j(t-t_{ji})p_{ji}c_{ji})$$

of (i) the various transmitted contributions $rs_j(t-t_{ji})p_{ji}c_{ji}$ combine additively and influence only the inactive portion (M_i-s_i) of M_i in determining ds_i/dt. Similarly, in the suppression term (iii) of (ii) the various transmitted contributions $\gamma w_j(t-\hat{t}_{ji})\hat{p}_{ji}$ combine additively and influence only that portion of M_i, namely w_i, which is active in determining dw_i/dt. The excitatory and inhibitory contributions thus play dual roles with respect to the local dynamics of q_i. With this duality in mind, it is easy to add further inhibitory and excitatory components to 7 and $\hat{7}$ in a similar fashion, and to introduce a multiple line structure as well. The addition of these extended embedding equations to our study suggest that we call the old equations a system of purely excitatory type. The system given by (i), (ii) and the usual equations for c_{ij} growth exhibits both excitatory and inhibitory effects, and we therefore call it a system of mixed type. The duality between excitatory and inhibitory processes will make it easy for the reader to consider for himself many inhibitory effects drawing upon our previous excitatory comments.

Equations of mixed type are a natural extension of the ideas implicit within the $c_{ij} = c^*_{ij}$ approximation. The remarkable fact has hereby emerged that the purely macroscopically defined desire to assure that perfect learning sometimes be possible--in part expressed by the $\tilde{c} = c^*$ approximation--contained within it, appropriately viewed, the necessity of the existence of dual fields $\hat{7}$ and the existence of lateral inhibitory processes in these fields, both of which are macroscopically invisible. We recall our comparison between 7 and the cortex and $\hat{7}$ and the thalamus to conclude with the conjecture that broadly distributed lateral inhibitory mechanisms for renormalizing cortical activity must exist within the thalamus.

36. Paired Associates

Before probing more deeply into questions of neurological interpretation, we return to a more thorough study of the source of these ideas, verbal learning. The serial character of a verbal learning situation is operationally characterized by a cyclic sequence of inputs timed with fixed intratrial interval. We now suggest that the first-order dynamics of the typical paired-associate situation are identical with those of the serial case. That is, in first approximation, differences in the two learning situations are generated wholly by differences in the input sequences. This claim does not, for example, imply that the strategies which subjects apply within free intratrial periods will be completely identical in both situations; it has already been observed that differences in strategy are representable by embeddings of a higher order of complexity than those needed to initially study simple list learning. Nor are fatigue effects and other slowly varying phenomena, such as the emergence of pacing effects, include in this claim. It shall nonetheless be shown that many familiar and distinctively paired associate phenomena are direct consequences of the same embedding equations

given for the serial case, but under a paired associate input paradigm.

Suppose that the pairs are of a simple and homogeneous character. For specificity, let them be composed of matched one-syllable adjectives and let only one item be presented at a time. To avoid the usual difficulties in "stimulus-response" terminology, call the "stimulus" item the first item and the "response" item the second item of any given pair. Initially, let every item be different and assign a single point to each adjective. This assignment will bejustified at a later time. Suppose that a first item is displayed, a guess of second item is made, and then the first and correct second items are displayed successively. As usual, rotate the order between successive trials, and use some standard presentation rate; in fact, set the field constants to realize a two-second intratrial interval throughout. The typical input sequence to a pair (p_i, p_{i+1}) is $\pi_i^{(e)}$, $\pi_?^{(f)}$, $\pi_i^{(e)}$, $\pi_{i+1}^{(e)}$. In the following discussion, we shall often assume that only the simple line structure prevails; hence the approximation $\tilde{c} = c^*$ will be applied for convenience. It shall become clear that this simplifying assumption on the lines is actually quite generally realized in the paired associate paradigm.

Suppose now that an experimental input has just been delivered to a first item p_i, where $i = 2k+1$, $0 < k$. Let the line structure be such that the response criterion is not satisfied, so that no feedback input is forthcoming. After a brief period, $\pi_i^{(e)}$ again occurs. This strong double impetus to s_i growth over a fairly long time interval will allow input induced asymmetries in the past strength field to decay considerably, thus enhancing $c_{i,i+1}$ growth when $\pi_{i+1}^{(e)}$ occurs. In this sense, the paired associate paradigm distributes practice even when the presentation rate is rapid. By distributing practice in this way, the double iteration of $\pi_{2k+1}^{(e)}$ will often also yield relatively strong backward connections between pairs of the form p_{2k}, p_{2k+1}. Iterating inputs at a point is very much like coming to the end of a list. In short, the paired associate paradigm tends to facilitate the condition $c^*_{2k,2k-1} + c^*_{2k,2k} + c^*_{2k,2k+1} \simeq 1$ from the very start. Since this high dynamical

contraction of the point field includes relatively strong one-step connections for the backward pairs (p_{2k}, p_{2k+1}), a detectable interference effect might result from changing pair orders. Similarly, if conditions conduce to the evokation of an error r_k, the sequence $\pi_i^{(e)}, \pi_k^{(f)}, \pi_i^{(e)}$ will tend to enhance the incorrect c^*_{ik}, c^*_{ki} couple more than in a comparable serial situation. Even given the correction procedure, the fact that the field is dynamically contracted to a small number of high c^* values can cause the subject considerable learning difficulties. For the existence of nearly equal point strength values at more than one point, no matter how high these values individually are, often goes hand in hand with the failure to satisfy the response availability criterion. Due to the repetitive correction procedure, however, the subject often overcomes these localized interference difficulties. When this occurs, the sequence $\pi^{(e)}_{2k-1}, \pi^{(f)}_{2k}, \pi^{(e)}_{2k-1}, \pi^{(e)}_{2k}$ leads to a high, and relatively symmetric, couple $c^*_{2k-1,\,2k}, c^*_{2k,\,2k-1}$ that conduces to good recall scores. Notice in particular that when $c^*_{2k-1,\,2k} \approx 1 \approx c^*_{2k,\,2k-1}$, an input to either point will cause a "dipole" of strength reverberation that rather efficiently preserves the couple of c^* values and leads to the evokation of the response associated with the other point. Backward learning here is excellent.

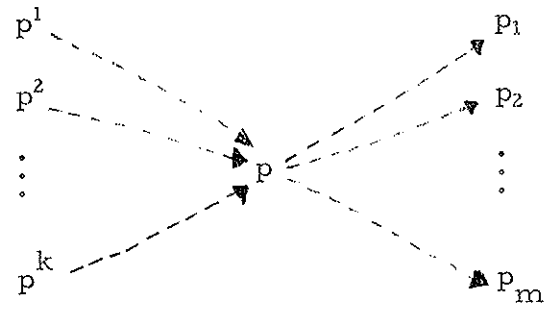
The rapid contraction $c^*_{2k,\,2k-1} + c^*_{2k,\,2k} + c^*_{2k,\,2k+1} \approx 1$ is mirrored in a comparable contraction of the multiple line structure. The rotation of pair orders insures that only c_{Iu} with I of the form $I = (2k, 2k\pm1)$ can hope to significantly influence learning. Since higher-order multiple connections will be of negligible importance, and since the input onset times are fairly regular, the c^* approximation becomes a useful theoretical approximation.

37. Item Availability

The setting for studies of item "availability" is often a paired associate field. These studies consider similarities in the dynamical

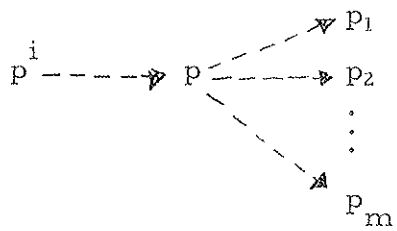
behavior of certain associations which have comparable positions in lists of inputs. For example, in a homogeneous paired associate field with no multiple crossings, all first items F_1 behave in a similar dynamical way, as do all second items F_2. The members of each F_i are therefore expected to be equally available in terms of their accessibility to recall. Suppose, by contrast, that we are given a list in which the point p_1^3 serves as a second item for three first items and as a first item for just one second item, while p_3^1 is a second item for one first item and a first item for three second items. p_1^3 and p_3^1 are not generally equally available in terms of accessibility to recall. Sources of differential availability can be explicitly studied in embedding fields, and many of our previous remarks can be interpreted in terms of this concept. We discuss the concept explicitly to demonstrate certain instructive difficulties which arise in its use and which have been pervasive in psychological theorizing.

Availability of items is usually operationally defined by first diagraming the collection of items which serve as the first items and as the second items of a given verbal unit. For example, if $p^1, p^2, \ldots, p^k$ are the first items, and $p_1, \ldots, p_m$ are the second items for p, then the simplest way to picture the geometrical substrate of the availability concept relative to p is to draw

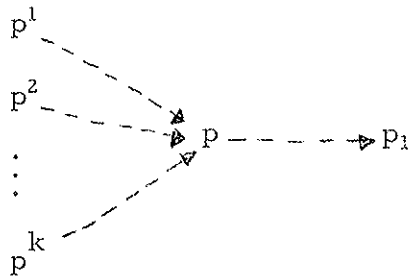


The dotted arrows distinguish the fact that $p^i \dashrightarrow p$ and $p \dashrightarrow p_j$ represent short sequences of input orders from the fact that $p^i \longrightarrow p$ $p \longrightarrow p_j$ represent two unidirectional connections of points by

directed field lines. Every field is thus given geometrically by no fewer than a pair of directed graphs, one outlining the field line structure while the other presents the ordering of external inputs, which we can call, by analogy, the input line structure. In terms of this picture, a list of simple type is one for which, given any p, the diagram for p is of the form $p^{i} \dashrightarrow p \dashrightarrow p_j$. It is a list with no multiple crossings. When this is true only for fixed p, the list is said to be simple at p. When the diagram is of the form



the list is backward simple at p. The list is forward simple at p when its diagram is of the form



Here attention is restricted to forward simple lists, since the present concern is not with probability matching.

With these concepts in mind, it follows immediately that the classical notion of item availability is highly limited. In particular, whenever it is important to consider point interactions of length greater than one, say $p^{i} \dashrightarrow p_j \dashrightarrow p^{k} \dashrightarrow p \dashrightarrow p_1 \dashrightarrow p^{m}$, the diagram, which is supposed to exhibit functionally important input connections, must be extended until it includes a description of practically the entire geometrical configuration of the input sequence,

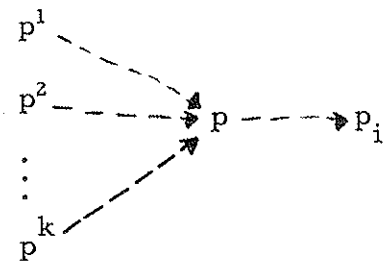
even though we choose to restrict attention to a fixed p. Variations in successive differences of input onset time with list position often induce variations in a unit's availability to recall. A mere "$\dashrightarrow$" gives no insight into variations of this kind. To discuss item availability in general settings, therefore, it is necessary to consider all aspects of the input paradigm, whence the classical methods for studying availability become useless in the general case. The present approach is free from these difficulties since it introduces a dynamical continuum which is sensitive to all variations of input ordering in space and time.

If the usual notion of availability is useless in general, we must ask why it has been used with greatest success in the special case of paired associate learning? This is understandable from our earlier observation that classical paired associate learning presents us with a highly concentrated normalized line structure from the very start. The availability diagram can therefore be restricted to one-step diagrams of the type illustrated above without strongly violating the dynamical facts. The regularity of input onset times in paired associate learning also permits us to average out many temporal factors across points, so that one line in the diagram actually does represent a bond of a certain strength whenever it join an element of F_1 (F_2) to an element of F_2 (F_1). Moreover, the normalized lines of a paired associate field are also very concentrated, so that the availability diagram and the diagram of normalized lines coincide in many places. It is for these reasons that the study of availability diagrams in paired-associate situations has been so useful. The concept of availability amounts to the intuitive guess that in paired-associate learning, one can draw one-step diagrams without ignoring too many theoretical variables. It is a guess about the character of the dynamical contractions found in these special learning situations without the foreknowledge that dynamical contractions exist.

Points which have identical availability diagrams are usually supposed to be equally available. Availability itself is operationally specified by some test which measures a unit's accessibility to recall. If availability is interpreted as "response availability" in the sense of this paper, then the familiar proposition that associations between two points p_1 and p_2 are symmetric if and only if p_1 and p_2 are equally available is simple to understand in many cases. Here availability means that the probability with which $\pi_1^{(e)}$ generates r_2 equals the probability with which $\pi_2^{(e)}$ generates r_1. Symmetry of associations in an embedding field means that $c^*_{12} = c^*_{21}$ whenever the c^* approximation is operative. If all other normalized line values are quite homogeneous and symmetry of associations is interpreted in this way, then the statement is entirely obvious when we deliver inputs to an initially quiescent strength field. If some $c^*_{1i} >> c^*_{2k}$, $k=1, 2, \ldots, n$, the statement is false, since the equality of excitation induced by $c^*_{12} = c^*_{21}$ is entirely offset by asymmetrical excitations carried by l_{1i}. In paired associate learning, however, we have seen that pairs of nearly symmetric first and second items are frequent asymptotes. Broad secondary excitations will not arise in paired associate learning with the same frequency with which one finds them under other standard input paradigms. The assertion that symmetric associations and equal availability go hand in hand is usually most fruitfully studied with paired associate paradigms. Again we have found a concept which becomes correct only under a special circumstance, the strong dynamical contraction of field functions throughout the learning session.

It might nonetheless be felt that "response availability" is not the only legitimate general concept of availability. Another view of availability amounts to the specification of a way to control an item's availability to recall. Given a forward simple point p, one augments the availability diagram of p in such a way that p is a second item

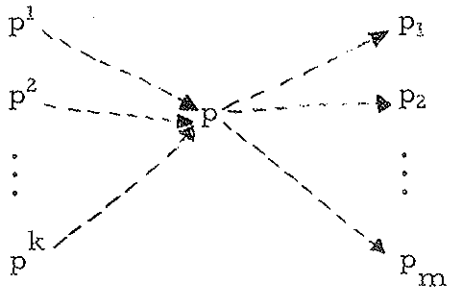
for several points:



By choosing the p^i appropriately, one can control the degree to which p is evoked, and thus rendered available, under any correction paradigm like $\pi^{(e)}_{2k-1}$, $\pi^{(f)}_{?}$, $\pi^{(e)}_{2k-1}$, $\pi^{(e)}_{2k}$. If we choose a situation for which an availability diagram is not a valid tool, then such a discussion of availability completely disintegrates. If we choose a situation for which such a diagram is a valid approximation, however, then the normalized line structure will closely parallel the availability diagram, and those items which possess the highest availability will also possess the most concentrated lines. Under effectively isolated inputs, the transmissions induced by the most concentrated lines satisfy the response criterion most easily, and all inputs must be effectively isolated for an availability diagram to be valid. We conclude that the most available items have the greatest response availability whenever item availability is well-defined. The notion of item availability is an example of a concept which is at best weakly determined in its broadest domain of definition and becomes describable in terms of "hidden variables" when it is restricted to a domain of definition for which it has a definite sense.

38. Markov Models Do Not Suffice

Another instructive difficulty attaches to the concept of item availability. Diagrams like



have suggested, by analogy with probabilistic reasoning, that the various p^i, p_j, and p be viewed as abstract states of some kind and the arrows as the geometrical substrate of a Markovian transition structure. Using this analogy, one is led to suppose that the probability of "mediated" associations, or associations involving a directed chain consisting of more than one arrow, can be computed by a formula like the Chapman-Kolmogorov equation in terms of one-step associations, or probabilities of transition. In general, the evaluation of mediated associations in terms of sums of products of one-step associations is impossible. In studying error correction, for example, we saw that simultaneously large c_{12}^* and c_{23}^* values can induce direct c_{13}^* growth according to nonlinear laws which depend strongly on the rapidity of the array of inputs to p_1, p_2, and p_3. The difficulty which arises here is pervasive throughout that part of the psychological literature which strives to find Markov models for general experimental situations. The program is doomed to failure. The process is not Markovian in this simple sense.

The temptation to try such models is theoretically understandable, however, quite apart from calling upon the desire to try a simple idea first. In every situation one does have a structural carrier, which is pictorially the same as a diagram of probability states and transitions. And in the interesting case of a paired-associate paradigm, the dynamics are not as far from being Markovian

as they are under other paradigms. We have already mentioned that a strongly contracted line structure is available for paired-associates from the start, and the paired associate paradigm distributes practice in such a way that the strength field is always highly concentrated as well. The higher-order transitions are thus actually built up in more of a step-by-step manner from the one-step transitions here than in many other settings. Especially when the intratrial interval is long are the first-order properties of the Markovian analogy qualitatively suggestive of the true dynamical situation. The familiar negatively accelerated theoretical Markovian distribution curves which agree so nicely with experimental data in paired-associate learning of this kind are thus qualitatively compatible with the dynamics of embedding fields as well. Another reason why the Markov approximation is sometimes successful is that the functionals of the experimental situation which are used as measuring devices often do not uniquely determine a single dynamical path. These functionals are localized both in time and in the point field carrying the total process. This localization corresponds to a coarse-graining of the space of dynamical paths. The nonlinear binding within a single dynamical path is partially averaged out by measuring only from localized regions in the space of paths. Such experimental situations are "noisy". So many experimentally uncontrollable factors are introduced in them that one is reduced to a study of asymptotics which do not yield central insights into the structure of the individual process.

In summary, "transitions" must be replaced by "transmissions." "Probabilities of transition" must be replaced by line functions whose dynamical significance exists in potentiality, and is realized only by regenerative strength functions which themselves strongly depend on the temporal pattern of external events. Just what a "state" or "point" represents in microscopic neurological terms is a question which requires extended consideration, and to which attention shall be increasingly turned as the formal structure of the

field is enriched.

39. A Principle for General Input Sequences. Stimulus and Response Learning

Suppose that input presentation rates satisfy the following conditions: (1) They are sufficiently rapid to substantially preclude variable individual subject strategies. (2) They are not so rapid that learning becomes impossible. (3) They are sufficiently regular that such higher-order expectations as input pacing are not abruptly disturbed in a given trial sequence, and may therefore be averaged out of the data. (4) They are not so prolonged that slowly varying noncognitive effects like fatigue become significant in the later trials. Under these conditions, our discussion has suggested that the distinction between paired associate learning and serial learning, in first approximation, becomes one merely of specifying different input sequences. By directly extrapolating from this fact, we now suggest the following

Principle: Under the above presentation rate prescriptions, all differences in subject performances may be understood, in first approximation, wholly as a manifestation of the special dynamical effects induced by the particular input sequence that is presented. That is, the embedding laws are an invariant of this enormous class of admissible input sequences.

For example, in a paired associate situation with nonsense syllable first items, one characteristically speaks of the process of stimulus learning as proceeding during the first few trials of learning. From the present vantage point, one sees this process as simply the gradual formation of strong multiple (including simple) line connections within the rapidly presented triad of stimulus syllables at the same time that lines form between these syllables in the usual way. The triad itself has many of the properties of a serial list—though a

short list, to be sure—and so one might expect an input to the last item of the triad to be more efficacious in producing a correct response than an input to either of the other two items of the triad. For a very rapid presentation of stimulus syllables, differential decay effects might be sufficiently small to further suggest that the lines which lead from the first item, not the second, will be the second most effective in generating responses, and so on. The stimulus triad, in other words, behaves like a short list. The point is that stimulus learning hardly deserves a special name as a distinguishable theoretical process; from the purview of the dynamics of an embedding field, it is a manifestation of just another input sequence. Similar remarks hold, of course, for the phenomenon of response learning.

While on the matter of sequences, it is an embarrassment to many contemporary theories that short lists, say of the form A-R (presented rapidly in succession), are often not learned on the first trial, when a paradigm of immediate recall is applied. This example alone is sufficient reason to worry despairingly about the old theories, and has justly added fuel to the controversy between "all-or-none" and "continuous" theories. In the present setting, simply deliver an input to p_A and then one to p_R. Notice that c^*_{AR} and c^*_{RA} rise rapidly while the other c^* values decay. Now deliver an input to p_A (p_R) alone. Strength transmission highly favors p_R(p_A) via c^*_{AR} (c^*_{RA}), and R (A) occurs just so long as the response criterion number is not impossibly high. Note further that if R is presented so long after A occurred that s_A has already decayed, then no benefits will be forthcoming to c^*_{AB} and c^*_{BA} growth and no learning occurs, as is surely the case if the subject chooses not to deliver internal inputs in rapid succession to p_A in the interim period. Once again an embedding field representation of such strategies must await a future discussion. Thus the most trivial example imaginable shows the poverty of a psychological theory for which a running time variable does not have a

central theoretical position. Moreover, this example of two-item lists reminds us that the embedding equations are sensitive to variations in list length and to the number of response alternatives in general. Without such sensitivity, the generalization to complicated lists would be hopeless.

40. Correction vs. Non-Correction Asymptotes

Before passing completely from the paired associate case, the correction technique contained therein suggests a remark concerning a broad class of verbal correction vs. non-correction processes. Namely, in a non-correction situation, if a fixed r_j is given in response to an input to p_i, c^*_{ij} will grow if the strength field is not broadly activated. On the next input presentation to p_i, h_j will thus be favored even further by transmission through c^*_{ij}. Iterating this process, it is clear that c^*_{ij} will approximate 1 asymptotically, so that r_j will almost always follow inputs to p_i. In a non-correction situation, the asymptotic response probabilities will cluster near zero and one. In a correction situation, on the other hand, let a distribution of several alternative second inputs follow experimental inputs to a fixed point p_i on successive trials. The c^*_{ij}, $j = 1, 2, \ldots, n$, values will oscillate in a manner that mirrors the distribution of second inputs closely. The response distribution is, in turn, strongly controlled by the c^*_{ij} distribution. On the trial after a particular c^*_{ik} is facilitated, if input conditions remain uniform over input positions, r_k will be comparably facilitated. If the distribution of second input alternatives is presented randomly over trials without long strings of repetitions of any one second input, then the mean response distribution will closely reflect the distribution of inputs to the various second input alternatives, and a situation resembling probability matching will

be approached once initial biases in the line structure are overcome.

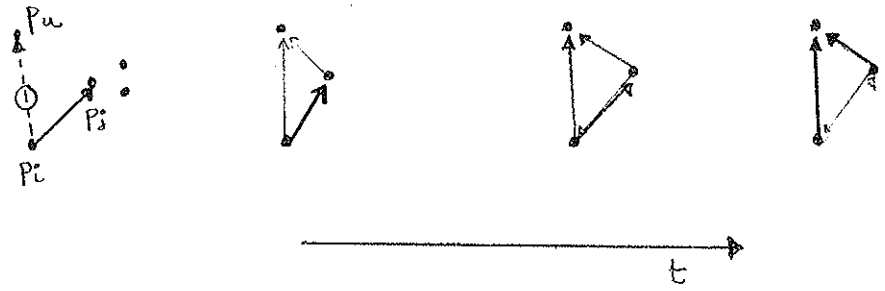
41. Mediated Associations. Response Facilitation and Response Interference

To the present, little has been said concerning the distribution of initial values $c_{ij}(0)$ at the beginning of the experiments considered. For simplicity, it has usually been assumed that all $c^*_{ij}(0)$ and all $c_{ij}(0)$ are approximately equal, $i \neq j$. A notable exception arose in our study of response clustering. Such an initially homogeneous distribution of lines is experimentally realized for lists composed of verbal units of similar type; for example, of simple adjectives which have been matched on a variety of tests of meaningfulness, familiarity, association values, and the like. The uniform initial condition is clearly a very special one, however, and the powerful effects of initial biases have been described in many experiments.

If the ordered pair (p_i, p_j) has recently been significantly more frequent in a subject's verbal experience than the pair (p_i, p_k), one might easily expect that $c_{ik}(0) << c_{ij}(0)$ just so long as $M_j \approx M_k$ and $A_{ij} \approx A_{ik}$. The effects of such a non-uniform distribution of initial values are not difficult to describe qualitatively and are in the same spirit as our above remarks, especially those on error correction and response clustering. Such a description is included so that the reader may achieve greater facility in carrying through embedding field arguments for himself.

A simple example is the following one. Let $p_{jj}=0$ and ignore all c_{jj} throughout. Restrict attention to the $\tilde{c} = c^*$ approximation for simplicity. Suppose that $c_{ij}(0)$, i and j fixed, is relatively high as a result of some kind of effective past pairing, that all $c^*_{km}(0)$, $k \neq i$, $m=1, 2, \ldots, n$, are approximately equal, and that $M_i=M$ and $A_{ij}= A(1-\delta_{ij})$. Now let $\pi^{(e)}_i$, $\pi^{(e)}_u$ occur at some

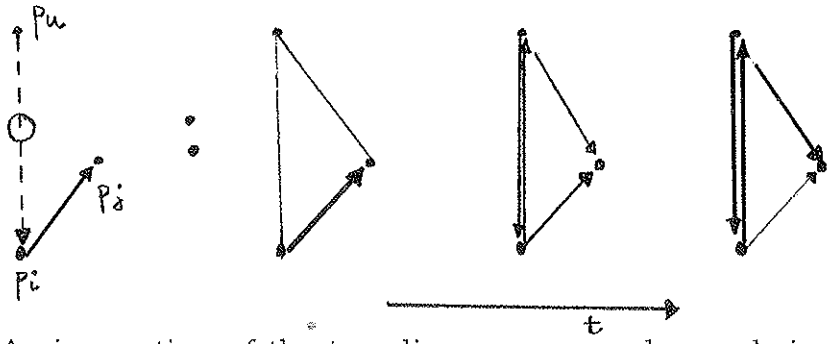
standard presentation rate, where $u \neq i, j$. Given $\pi_i^{(e)}$, a relatively large transmitted point strength value will reach p_j from p_i and will thence be transmitted towards p_u before $\pi_u^{(e)}$ occurs. Thus the growth of c_{iu}^* will be supplemented by the growth of c_{ju}^* as well. An input to a given item hereby generates line connections (though of lesser magnitude) between a strongly line connected item and third item that receives a contiguous second input. A "mediated" association has been induced. After successive presentations of $(\pi_i^{(e)}, \pi_u^{(e)})$, however, c_{ij}^* will diminish since c_{iu} continues to grow. Thus the initial secondary transmission effect will fade as trials proceed. Nonetheless the second-order c_{ju} value will sustain itself for awhile in a manner compatible with the decay laws and the general line leveling effects of strength scatter to other portions of the point field. A suggestive diagram representing this process is the following one:



$p_i \longrightarrow p_j$ represents the large initial field line value and $p_i \dashrightarrow p_u$ gives the direction of the input sequence. " $--\ominus--\rightarrow$ " means that this sequence is repetitive.

Now suppose that the above initial conditions prevail under the reversed input sequence $\pi_u^{(e)}, \pi_i^{(e)}$. Here, both c_{ui} and c_{uj} will grow considerably, although c_{uj} will be generally smaller than c_{ui}. Nonetheless, compared to the situation which evolves under the initial condition $c_{ij}(0) \approx 0$, c_{uj}^* will be comparatively large. Now as $\pi_u^{(e)}, \pi_i^{(e)}$ is repeated, c_{iu}^* will continue to grow until c_{ij} receives very little second order augmentation, whence asymptotically c_{ui}^* will grow to a high value. The

corresponding diagram is:



An inspection of the two diagrams reveals an obvious difference between the two situations before the line effects represented by the third stage of the drawings fade. In the first case, the strongest lines lead from both p_i and p_j to p_u, forming a fan of strongest lines. In the second case, a dipole of strong lines forms between p_i and p_j, while c^*_{uj} first grows and then fades. If in a recall situation $\pi^{(e)}_j$ occurs, the higher c^*_{ju} value in the first situation will lead to an r_u response more surely than in the second situation, whence "response facilitation" will occur as a result of training on $\pi^{(e)}_i$, $\pi^{(e)}_u$. Such a facilitation effect also holds when the first situation is compared with the case for which all initial line values are uniformly low and the input sequence of the first situation occurs. By contrast, compare situation two with the situation having uniform initial lines after both sets of initial conditions have been subjected to training under $\pi^{(e)}_u$, $\pi^{(e)}_i$. In situation two, the occurrence of $\pi^{(e)}_u$ will be followed by greater "response interference" in producing r_i than in the initially uniform case; for in situation two, c^*_{ui} is relatively low and c^*_{uj} is relatively high compared to the initially uniform case, due to the greater secondary transmission through l_{ij} in situation two. Moreover, the total time taken to reach a fixed criterion in a retraining task under $\pi^{(e)}_j$, $\pi^{(e)}_u$ in a situation resembling the last stage of situation one might well be diminished, relative to the uniform case, due to these facilitative effects.

Similarly, the time required to achieve a fixed criterion on retraining under $\pi_u^{(e)}$, $\pi_i^{(e)}$ in a situation resembling the last stage of situation two will be longer than the time needed to reach this criterion when the lines are initially uniform.

Such facilitative and interference effects with respect to measures on training and on retraining trials have often been experimentally demonstrated, but they seem to have eluded the simple explanation which we have just given. Other effects of this type can be read off from the diagrams. More complicated effects can be similarly treated. The reader might attempt to explain the dynamics of the above two situations combined with a situation of stimulus learning, say of nonsense syllables. From the present unifying perspective, these divers effects are all in first order the result merely of choosing different initial settings of initial values and different input sequences.

42. The Decomposition of Words

In any discussion of verbal units which are not equated as to familiarity, meaningfulness, and the like, a fundamental question arises: Given an arbitrary verbal unit p_i, how does one assign the value M_i? Certainly a familiar one-syllable adjective deserves a high value, as does a consonant. But what of the triple ABC? Is there a sense in which a $p_{(ABC)}$ and an $M_{(ABC)} > 0$ exist above and beyond the p_k and M_k, k = A, B, C? If not, then how can one justify the strong intuitive desire to assign a single point to familiar adjectives of more than one syllable? Indeed, formally at least, if one separates each syllable of a three-syllable adjective by a slight pause in pronunciation, a situation approximating the pronunciation of a nonsense syllable is achieved. From this simple observation, we see easily that a familiar three-syllable adjective may be viewed as a triad of points p_1, p_2, p_3, where

p_i is the i^{th} syllable, for which $c^*_{i,i+1}$ is close to 1 for $i = 1, 2$. The multiple line values interlacing these points will also be large, with a marked forward bias. Henceforth, the appropriate multiple line analog of a remark pertaining to simple lines will often be suppressed for convenience.

This procedure generalizes to a large number of verbal units. Given a complicated word, one seeks to decompose it, firstly on an a priori intuitive basis, into subportions which are perceived to be simple or atomic. The criterion of simplicity depends on the modality through which the word is presented to the subject. Choose the auditory modality for convenience and consider only subjects who have had no formal education in written language. Then a natural criterion for the atomicity of a candidate verbal form is that it be easily pronunciable but that no subportion of it more complicated than a single letter appear as a natural pronunciable unit in the subject's vocabulary. Single letters are also taken to be atomic units if they are presented singly. This criterion of atomicity satisfies the condition that the subject neither utter nor hear verbal forms that are not built up of strings of atoms if he lives in a society of his peers in knowledge. Such a condition is manifestly a stability condition. It is necessary to discuss the stable case when introducing ideas of atomicity, for units which are atomic in one situation need not be atomic in another.

It is amusing that this definition of a fundamental atomic unit resembles the definition of a prime number if all single letters are viewed as algebraic units and the relation that a verbal form contain a certain subform be taken as a type of divisibility property. We shall see that this analogy breaks down strongly when all units are well-learned. For otherwise we should have the rather discouraging theorem: all numbers are primes.

Continue to suppose that the subject under investigation has only auditory and vocal experience with a language that is well-known to him. A representation of the formal and non-denotative aspects of the structure of any atomic verbal unit in his vocabulary can be given by a point p_i with an associated M_i. The formal construction of standard words from such a collection of points amounts roughly to the specification of finite connected and directed strings of c_{ij} values which are so arranged with respect to their renormalization structures that in the approximation by $\tilde{c}_{ij} = c^*_{ij}$, the c^* values from a given unit to its successor in a directed chain are very high. Several important properties of this structure should be noted before passing to a neurological interpretation of its individual units.

Observe that once the first few points of a sequence of high c^* values have been excited, the embedding equations will force the strength distribution to configure itself along the path followed by the most highly weighted lines until the end of the sequence is reached. During such a period, hardly any verbal alternatives will be available to the subject. A comparable effect occurs when highly familiar sequences of words occur. Although the line structure tying together the atomic parts of individual words is commonly stronger than that interlacing different words, multiple line effects will rapidly reduce the number of possible verbal alternatives as the strength field moves deeper into a highly familiar word sequence. This reduction process is one of the fundamental ways whereby the motion through familiar syntactical forms interacts with individual words to select a string of words forming a coherent sentence.

The existence of such a collection of contracted strings of atomic units, strings of these strings, and so on, enormously simplifies the task of the communicating organism. Once a string is entered, the intrinsic structure of the field carries off the

evokation of the entire verbal form, no matter how complex its original parts might have been before they were connected. The organism needs a mechanism for choosing new verbal forms for emission at comparatively discrete times relative to the rate of ongoing verbal emission. Similarly, in the process of listening to language, the mere entrance of a message as an external input into some well organized string will enable the organism to predict with high probability the sequence of verbal forms most likely to immediately follow, just so long as the source and the listener have had similar language experiences. The most probable form to follow will automatically be excited more than other possible forms over the lines in the string whether or not the communicator continues. In both the processes of emitting and of receiving messages, as the strength distribution representing the motion through language forms flows along, the higher order line structure continuously modulates it in such a way that the motion between verbal forms proceeds quite smoothly and, from the point of view of the communicating organism, effortlessly. This smooth non-linear modulation process, as in the special case $ABC \rightarrow D$, depends on an interaction structure which is sensitive to dynamical time averages over many subsets of a point field of simple type, and which reorganizes this field in terms of these averages.

43. The Fundamental Dynamical Units of Language

One consequence of this view of the process is that the fundamental perceptual unit in language is not a phoneme or, for that matter, any other unit derived by a merely formal decomposition of language units into simple parts. It is rather the smallest highly contracted string that is excited by external inputs in the particular experimental situation under study. To fix any standard vocabulary of fundamental units at a single time and then suppose that this

vocabulary remains a complete one through time is to utterly misunderstand that it is a central role of neural dynamics to constantly reduce complicated units into a form which can be handled just as simply as the original units so that further units can in turn be constructed from these.

44. Chunking and the Magic Number Seven

G. A. Miller introduced the term "chunking" to suggest that the simple information theoretical analysis of information transfer does not hold when the units being analysed can be combined in various ways. Our analysis of strings shows how this combination of units takes place. The relevance of information theory to the analysis is clear from the appearance of the information functional in the definition of response availability functions, and our previous discussion of concentration sets shows how availability functions arise naturally in studying the dynamics of a field. In considering information theoretical analyses of learning situations, the first important fact to observe is that large simultaneous strength values over many points insure that $A_i < \Gamma$ for all i, so that no responding occurs. To insure that responding does occur, one must temporally stagger inputs in order that the strength field concentrate about a small number of points at any time. In an information theoretical analysis of response data, this is interpreted to mean that the channel capacity has a rather small upper bound, which empirically turns out to lie in the range of seven. As new strings of points are formed, however, the strength field induced by an input to the beginning of the string remains highly concentrated until its end, and this small concentration set implies better responding than we would expect if the units from which the string is composed were not

so strongly connected. Because of the close connection between concentration sets, A_i, and $\overline{H}$, an information theoretical analysis also shows that the units of the field seem to have been collapsed into larger units, as indeed they have when strings are formed.

When the single letter A is presented visually or auditorily to a standard subject, A is an atomic verbal unit. Yet when ANT is presented auditorily as a word, the sound representing the entire word generates the fundamental unit of the situation. ANT is not embedded in the standard adult under auditory presentation as a disjoint collection of points p_A, p_N, p_T. On the other hand, when ANT is presented visually, an observer is capable of eliciting the sounds A, then N, then T, as well as the word sound for ANT. Each of A, N, T, ANT is a fundamental unit in this situation. The unique flexibility of the visual process, whereby a given stimulus can be decomposed first into subportions of one kind (A, N, T), then into subportions of another kind (ANT), and so on, without any one decomposition interfering with the next, is due to a subtle interaction of geometrical and temporal factors. A study of eye movements is a prerequisite to understanding these factors, and to this we will turn in a later section. The analysis of the symbols A, N, T, and ANT as single sounds under auditory presentation is due to a geometrical difference between the auditory and visual processes, but does not involve drastic differences in local dynamics. The process whereby a string is formed is qualitatively the same for both modalities.

Another consequence of this view of the fundamental units of language is that during active listening to speech with intermittent noises, a subject's recall of the position of the noise relative to the speech flow will be rather poor. The dispersion of his guesses will

be broad. This suggested to Broadbent that the subject is "listening" to familiar groups of words and not to single words. This is exactly what we expect from our observation that once a linear string of words is entered, it will contract the strength surface to follow this string to its end. The entire string is a fundamental unit; thus the subject cannot easily coordinate superimposed noise forms with its smaller parts. Similarly, the theorist is tempted to ignore the internal structure of the string and to consider only how much strength it receives, how much it emits, how long the process takes, and in what position the string as an indivisible entity lies relative to the other strings in the field.

45. What Is a Choice?

From these remarks, it is clear that the classical concepts of a "choice structure" suffer from the same difficulties which were attributed to the attempts to attack learning situations with simple Markov models. A "choice" is simply not an act whose underlying dynamics may be contracted to a single point in time after which a transition to a new "state" occurs; one does not dynamically "choose this, then that." Rather the process is a continual reconditioning of the global strength field until a suitable response availability criterion is met. When this occurs, the response emerges into consciousness. The process whereby the "choice" is made proceeds continuously relative to the time scale used to measure the intervals that separate successive fulfillments of the availability criterion. The apparent discreteness of many "acts of choice", which are the manifestations of the satisfaction of the availability criterion, must not be confused with the choice process which precedes

these acts. This apparent discreteness is a sign of the stability of our perception of familiar behavioral forms--itself a result of the existence of highly contracted line structures--and little more.

The confusion of discrete choice times with a continuously modulated renormalization process interpolated between these times has caused errors in psychophysical theorizing, say with respect to reaction time, where it is especially tempting to conclude from the frequent use of brief stimulus durations and widely separated stimulus onsets that one can decompose the process into a discrete set of times between which simple energy summations occur, an untenable view in the general case.

46. The Reduction of Conscious Acts to Reflex Control

Another useful observation related to the example of strings of atomic forms is the following. The most fundamental verbal units are hard to forget. It must therefore be supposed that the field extensions of the points involved in highly structured strings are so arranged that in the absence of external perturbations, the line values of the string are preserved in some way. The way is suggested by the fact that when these line structures are activated, either by external or by internal inputs, they induce local walks which help to guide their own natural dynamical evolution. The line structure representing oft-repeated acts gradually concentrates and therefore can efficiently sustain itself. Acts whose performance at an earlier time required the active attention of the organism become and remain increasingly automatic after frequent repetitions. They are reduced successively from voluntary acts to reflex acts which insure their own dynamical evolution.

The continual reduction from behavioral forms of higher conscious type to forms of lower reflex type--from "composite numbers" to "prime numbers"--is a pervasive property of the evolutionary emergence of neural structures, and is strikingly in evidence in the increasing encephalization of higher phylogenetic organisms. A subtly timed line-renormalization process and the complicated hierarchical geometry of higher neural mechanisms are joint partners in this emergence, neither one capable of introducing behavioral order without the other.

47. A Comment on Perceived Time Quantization

An interesting possibility derived from studying strings of atoms begins with noticing that the rate at which choices are made and the perception of the rate of the passage of time are closely related phenomena. From the recognition that choices themselves are the expression of certain compressions of a strength field, it follows that an intermittent compression of large sectors of the global strength field might generate a parallel impression of compressions in the time structure. If these compressions are sufficiently rapid relative to those portions of the strength field which are internally--and thus intrinsically--timed, a perceived quantization of the time structure might follow. If it were possible, on the other hand, to mobilize the entire strength field to fluctuate in perfect global unison, no way of estimating the extent of this fluctuation would remain for the organism. The problem of judging the magnitude of quantities by means of naturally occurring sensory modalities must depend on the relative motion of nonglobal strength flows. The temporal evolution of such a flow appears as the motion of a kind of intrinsic coordinate system and the relative motion of at least two such systems is necessary to

make a judgment. No distinction between the "coordinates" of these flows and the actual dynamics can naturally be made; the entire flow is a unified entity, or field. Two such fields cannot participate in process of judging relative magnitudes unless they nontrivially interact, and the field quantities, or the "intrinsic coordinates" of the field, are themselves altered by this interaction. The proper experimental domain from which to view such judgmental processes is psychophysics. The bridge from verbal to psychophysical events can thus be constructed from the abstract concept of a string of atomic forms. It is a bridge which shall be much easier to cross after the notion of an atomic form is further analysed.

48. A Correspondence Principle for Enbedding Fields

What has thus far been accomplished in this analysis is, relative to the specific input-output system of auditory verbal comprehension and speech, a collection of language forms has been decomposed into a set of atomic subforms which are bound together by a hierarchical line structure and which participate in interactions with a rich collection of field extensions.

A fundamental question still remains unanswered: What theoretical process justifies the decomposition of all verbal forms into atomic forms and forbids the decomposition of atomic forms into still finer sets of points? What distinguishes an atomic form from a composite form? Within the scope of the theory as it has been developed to now the answer can only lie in the line structure. If an atomic form is composed of more than one point, then the manner in which its constituent points are mutually interconnected must be so stable in time that it is possible without disturbing the rapidly varying dynamics of the system in first approximation to collapse both the structural carrier and the functions associated with this carrier into a single point which represents a spatial average of finer points, and a smaller number of functions which represent dynamical averages over the finer points. Proceeding in the other direction, if it is assumed that the total number of objects in any structural carrier is

finite and bounded, then the decomposition of atomic forms into smaller units must eventually terminate. There must exist a universal, or enveloping, structural carrier $\mathcal{L}^*$ from which all other carriers are generated by taking suitable spatial averages. Similarly, associated with the universal carrier is a universal set of functions $\mathcal{S}^*$. $\mathcal{L}^*$ and $\mathcal{S}^*$ generate a universal embedding field $\mathcal{F}^* = (\mathcal{L}^*, \mathcal{S}^*)$. $\mathcal{F}^*$ represents a brain.

This passage to $\mathcal{F}^*$ in principle accomplishes the transition from a "state space" or set of abstract points and their interactions to an enveloping dynamical system evolving over a natural geometry. The concrete realization of this passage will require considerable effort, but it need not proceed in vacuo, for certain features of pre-averaged psychological fields must be invariant under passage to $\mathcal{F}^*$. We have, in fact, a correspondence principle for embedding fields. When the lines of a collection of dynamical forms over $\mathcal{F}^*$ become stable, it is possible to contract these forms to points and to study fields $\mathcal{F}$ in which only the more rapidly fluctuating line structures appear explicitly. Corresponding to this purely formal contraction is the fact that the most stable line structures are the ones whose dynamics proceed most automatically in the organism and the ones to which the organism least attends consciously. The variables which the conscious theorist decides to ignore are the same variables that the conscious organism ignores. The dimension of fields $\mathcal{F}$ thus has a deep behavioral significance. Such central principles as intrinsic renormalizability, lateral inhibition, and the like are retained when we pass between $\mathcal{F}^*$ and all levels of pre-averaged fields $\mathcal{F}$, and the embedding equations for $\mathcal{F}$ must hold in a statistical sense over $\mathcal{F}^*$. The passage from pre-averaged fields $\mathcal{F}$ to $\mathcal{F}^*$ is the passage from macroscopic psychological variables to microscopic neurological variables of which we spoke in the Introduction, and can now begin to realize.

49. Control Subforms and Excised Subfields

A useful definition for discussing degrees of pre-averaging in a field is the following. If the field $\mathcal{F}_1 = (\mathcal{L}_1, \mathcal{S}_1)$ is derived from $\mathcal{F}_2 = (\mathcal{L}_2, \mathcal{S}_2)$ by averaging $\mathcal{F}_2$ in the sense described above, then $\mathcal{F}_2$ is called a dynamical refinement of $\mathcal{F}_1$. This fact is denoted by $\mathcal{F}_1 \subseteq_d \mathcal{F}_2$ or by $\mathcal{F}_2 \supseteq_d \mathcal{F}_1$. In particular, $\mathcal{F}^* \supseteq_d \mathcal{F}$, where $\mathcal{F}$ is any field. $\mathcal{F}_2 \supset_d \mathcal{F}_1$ means that $\mathcal{F}_2$ dynamically refines $\mathcal{F}_1$ and is not equal to $\mathcal{F}_1$.

We now apply the correspondence principle to a collection of atomic forms in $\mathcal{F} \subset_d \mathcal{F}^*$, where $\mathcal{F}$ is a homogeneous field. By the homogeneity of $\mathcal{F}$, whatever be the particularities of the evolution of an $\mathcal{F}$-form as a strength flow within $\mathcal{F}^*$, it must be true that any two such forms must flow through a parallel series of hierarchical steps within $\mathcal{F}^*$. Secondly, it must be possible for the atomic forms of $\mathcal{F}$ to generate one another, considered in $\mathcal{F}^*$, in very much the same way as single points transmit to one another in $\mathcal{F}$. For example, suppose that the realization of a given form is a complicated muscular act like the production of a simple language sound, for which a regulated sequence of preparations in lips, tongue, jaw, pharynx, diaphragm, etc., is required. It is clear that if one form Ω_1 of such complexity is ever to become capable of generating another form Ω_2 with any satisfying degree of stability, then it is impossible that all of the complexities of Ω_2 be simultaneously generated by all of the complexities of Ω_1 in equal measure. Rather, a proper subportion $\bar{\Omega}_2$ of Ω_2 must be more critical than the rest of Ω_2 in setting off the entirety of Ω_2. The dynamical evolution of $\bar{\Omega}_2$ cannot correspond to such a complicated strength flow over $\mathcal{F}^*$ that Ω_1 can generate it only with difficulty. Moreover, the activation of this privileged subportion $\bar{\Omega}_2$ of Ω_2 should generate a strength flow over structures which represent increasingly fine preparations for the utterance of the sound. Hierarchical geometrical arrangements play an important role here.

Call the privileged subportion $\bar{\Omega}_i$ of Ω_i hereby isolated a control subform of Ω_i. Such control subforms merge continuously with the entire dynamical trajectory that generates a language sound, and are isolated from

this trajectory only as a conceptual convenience. The existence of these subforms is a determining factor in making possible the $\mathcal{F}$-representation of a flow in $\mathcal{F}^*$ in terms of a small discrete set of points. The discreteness of the $\mathcal{F}$-representation is a consequence of the fact that the flows necessary to evoke two forms evolve in parallel hierarchical steps and the times, considered in $\mathcal{F}^*$, during which the leading subforms of these flows are initially activated are discrete in $\mathcal{F}^*$. In summary, the relative simplicity and homogeneity of structure of control subforms are necessary for the stable generation of one atomic flow by another. The representation of atomic flows as single points in a homogeneous $\mathcal{F}$ expresses the strong similarity of the steps in the hierarchical evolution of two flows subserving the same modalities.

The behavior of a field $\mathcal{F}$, $\mathcal{F} \subset_d \mathcal{F}^*$, need not be a precise average of that of $\mathcal{F}^*$. Rather, it represents $\mathcal{F}^*$ up to a type of equivalence relation defined within the class μ (B) of all $\mathcal{F}$'s sharing the same atomic forms B. Each field in μ (B) represents all of those properties of atomic flows B in $\mathcal{F}^*$ which are distinguishable in first approximation after pre-averaging. For example in a field $\mathcal{F}$ whose points are verbal atomic forms, we ignored in first approximation those hierarchical preparations necessary to evoke any sound which are common to all the forms considered. These common hierarchical preparations are spatially indistinguishable. It was also possible to cut away those portions of the flows whose line dynamics are stationary in time in order to concentrate on the more variable portions. These stationary parts cannot be temporally distinguished over many trials. The fields $\mathcal{F}$ are thus not merely averages of $\mathcal{F}^*$. They are averages over excised subfields of $\mathcal{F}^*$, as the existence of control subforms already suggested. The exact form of the excision is not immediately easy to judge, precisely because the macroscopic data, which performs the excision for us, does not distinguish all the elements of the $\mathcal{F}^*$ fine structure clearly. It is intuitively clear, however, that the fields with which this study began are maximal excisions of $\mathcal{F}^*$, and were therefore a natural point at which to begin.

It cannot be too strongly exphasized that the possibility of passing to excised and pre-averaged fields, which so very much simplifies the task of the

theorist, is not merely a mathematical convenience. The choice of fundamental pre-averaged field units is made by an organism who himself behaves according to field laws. The possibility of naturally passing to fields $\mathcal{F}$, $\mathcal{F} \subset_d \mathcal{F}^*$, is a direct reflection of the neurological events which bring increasingly complicated behavioral acts under greater reflex control and thereby simplify the organism's task in adapting to his environment. The a priori judgment of what units are critical in constructing a macroscopic field exactly parallels the dynamical process which allows us to ignore oft-repeated behavior sequences, a process which carries on during our hours of theorizing. In this sense, intuition is here its own judge.

50. Core Trajectories

The fact that the embedding fields thus far studied provide a type of average of neural activity in $\mathcal{F}^*$ has the following consequences: A strength field is analog of a field of neural potentials, a line structure takes the role of such objects as fiber tracts and endbulb arborizations, and the points represent collections of cell groups. In order to say more about the M_i functions, these correspondences shall be used to perform a heuristic construction within $\mathcal{F}^*$.

Suppose that the perception of an atomic verbal unit has been induced auditorily in $\mathcal{F}^*$. The magnificent complexity of the neural activity which is correlated with this perception suggests the adoption of a policy of deliberate naïveté at first in order to be able to draw qualitative conclusions about this activity. Restrict attention to the distributions in potential corresponding to the verbal unit as they flow to the primary auditory cortices. These distributions are transmitted through a wide variety of nerve fiber structures to extensive cortical and subcortical regions after they pass through the primary cortices. This entire process traces out a space-time trajectory of excitation and inhibition over $\mathcal{F}^*$. To each auditory presentation of a given verbal unit σ, we can associate a trajectory $\mathcal{T}(\sigma)$ of this kind. For specificity, one can think of presenting the verbal unit σ auditorily to a resting and awakened subject a number of times under a standard experimental condition. Supposing that the onset of

experimental inputs is always measured from $t=0$, we then take the means of $\mathcal{S}^*$ for each space-time point in $\mathcal{L}^* \times [0,\infty)$ over the various trials to compute $\mathcal{T}(\sigma)$.

Now define the ϵ_1-structural carrier $\mathcal{P}_{\epsilon_1}(\sigma)$ of σ as that region of $\mathcal{T}(\sigma)$ whose means exceed ϵ_1. Compute the variances of the potentials generated at each space-time point in $\mathcal{L}^* \times [0,\infty)$ over the various σ presentations. Suppose that there exist certain space-time points whose variances are very large relative to the rest of the variances, say larger than ϵ_2. Exclude these points from the ϵ_1-structural carrier of σ, and denote the contracted carrier by $\mathcal{P}_{\epsilon_1,\epsilon_2}(\sigma)$. Choose ϵ_1 and ϵ_2 in such a way that $\mathcal{P}_{\epsilon_1,\epsilon_2}(\sigma)$ is contracted until it includes only those space-time points whose means (variances) are judged to be sufficiently large (small) to warrant their being considered dynamically essential structures in the trajectory. The resulting trajectory is called $\tilde{\mathcal{T}}_{\epsilon_1,\epsilon_2}(\sigma)$, or simply $\tilde{\mathcal{T}}(\sigma)$.(*)

In this manner, given any atomic verbal point p_i, a pair (D_i, f_i) is chosen consisting of a structural space-time carrier $D_i = \tilde{\mathcal{T}}(p_j)$ and a real-valued, mean excitation function f_i with domain D_i, which provide an idealized representation of the neural events which correspond to p_i considered in $\mathcal{T}^*$. We suppose that the (D_i, f_i) have been chosen so as to completely determine p_i. The map $p_i \longrightarrow (D_i, f_i)$ is therefore one-to-one into the space of all such possible pairs. Call (D_i, f_i) the core of p_i.

(*) The assumption that we can judge the means and variances of individual space-time points in $\mathcal{L}^* \times [0,\infty)$ independently in forming $\tilde{\mathcal{T}}(\sigma)$ is a special assumption which can be improved only at a later time. One can already see, however, that ratios like ϵ_2/ϵ_1 are critical in determining $\tilde{\mathcal{T}}(\sigma)$.

Given two points p_1 and p_2, we now choose a measure of distance between their respective cores. Extend f_1 to $\bar{f}_1$ on $D_1 \cup D_2$ by letting

$$\bar{f}_1(w) = \begin{cases} f_1(w), & w \in D_1 \\ 0, & w \notin D_1 \end{cases}$$

Similarly extend f_2 to $\bar{f}_2$ on $D_1 \cup D_2$.(*) The desired distance function is simply

$$d_{ij} = d(p_i, p_j) = \int_{D_1 \cup D_2} |\bar{f}_1 - \bar{f}_2| \, dq.$$

It is assumed in the notation $D_1 \cup D_2$ that the time variable starts running in both D_1 and D_2 at the effective onset time of the two trajectories. More explicitly, we arrange the carriers D_i so that f_i projected onto the time axis of D_i has $t=0$ as the left-hand endpoint of its support. d_{ij} is not a proper metric for general functions $\bar{f}_i$ because $d_{ij} = 0$ whenever $D_1 \cap D_2 = \emptyset$. In no physiological case does $D_1 \cap D_2 = \emptyset$ arise, however.

dq is not yet well defined. Indeed, we have not yet said explicitly what cellular regions are the spatial components of the points of $D_1 \cup D_2$. Since we are in $\mathcal{F}^*$, each point is, strictly speaking, a single cell. Nonetheless, it is difficult to achieve a stable mean of excitation over single cells. We can therefore suppose instead that each cellular component of $D_1 \cup D_2$ is a small connected region of gray matter, considered as a region embedded in Euclidean 3-space. The proper choice of region is not an entirely arbitrary matter, for cells subserving completely different functions lie juxtaposed in Euclidean norm. We therefore assume that du is measured over localized regions of cells of a given type. The functions f_i are space-time averages taken over many trials whose spatial components are the small regions depicted.

(*) In general, 0 on $w \notin D_1$ must be replaced by the equilibrium potential, but we are being naive, so the equilibrium potential has been scaled to zero.

51. Macroscopic Equations as Averages over $\bigcup_i \mathcal{T}(p_i)$

M_i is a measure of the average of the M values taken over the points D_i of the core trajectory of p_i, or at least over that subregion $\mathcal{T}(p_i)$ of the core which is the control subform of the core of p_i. p_{ij} must be decomposed into $p_{ij} = R_i \mathcal{P}_{ij}$, where R_i measures the effective total mass of the points in $\mathcal{T}(p_i)$. R_i is given by $R_i = M_i n_i$, where n_i is the total number of points in $\mathcal{T}(p_i)$. A finer determination of R_i is $R_i = \int_{\mathcal{T}(p_i)} M dq$. $\mathcal{P}_{ij}$ gives the average line density connecting $\mathcal{T}(p_i)$ to $\mathcal{T}(p_j)$. Although as a macroscopic variable, R_i is determined entirely by counting point quantities, R_i is not independent of the microscopic line structure. The points which are singled out to form $\mathcal{T}(p_i)$ can change with time. It is the deviations from initial line values within $\mathcal{T}^*$, induced by repetitive $\pi_i^{(e)}$ presentations, that determine which points within $\mathcal{T}^*$ form $\mathcal{T}(p_i)$, and which are therefore considered in computing R_i. Since M_i and $\mathcal{P}_{ij}$ are computed over $\mathcal{T}(p_i)$ and $\mathcal{T}(p_j)$, they too depend on variations in the microscopic lines through time.

Certainly the choice of a single point strength function to represent the activity of an entire core trajectory sacrifices a great deal of information by discarding the possibility of studying the distribution of excitation within a trajectory. Indeed, if $D_i^{(1)}(t)$ is the spatial projection of the trajectory of p_i at time t, $D_i^{(1)}(t) = \{w: f_i(t, w) > 0\}$, then $s_i(t)$ roughly measures

$$\int_{D_i^{(1)}(t)} f_i(t, w) du(w)$$

The inability to study the distribution of f_i restricted to $D_i^{(1)}(t)$ is matched by the collapse of all information concerning the distribution of strength transmission within each $D_i^{(1)}(t)$, and transmission from $D_i^{(1)}(t)$ to $D_i^{(1)}(t+w)$ can be studied only in the mean. All of these transmission factors were necessarily incorporated into a single self-excitation term $(M_i - s_i)(r s_i(t - t_{ii}) p_{ii} c_{ii})$

in our first set of dynamical equations. We now see that the term t_{ii} means that all internal transmissions take the same time to get from one part of the core trajectory to another. It is more plausible in general that transmission time lags be distributed according to a function $T_{ii}(w)$, $w \in D_i^{(1)} \equiv \bigcup \{ D_i^{(1)}(t): t \in [0,\infty) \}$. For fixed $k > 0$, the fraction of all points in $D_i^{(1)}$ for which $T_{ii}(w)=k$ is given by

$$\frac{\int_{\{w:\ T_{ii}(w)=k\}} \lambda_{ii}(w)du(w)}{\int_{D_i^{(1)}} \lambda_{ii}(w)du(w)}$$

where $\lambda_{ii} : D_i^{(1)} \to [0,\infty)$ is a structural density such that

$$p_{ii} = \int_{D_i^{(1)}} \lambda_{ii}(w)du(w).$$

A first step in passing from maximal excisions to $\mathcal{T}^*$ is to replace $s_i(t-t_{ii})p_{ii}$ by the transmission integral

$$\int_{D_i^{(1)}} s_i(t-T_{ii}(w))\ \lambda_{ii}(w)du(w).$$

The sets $\{ w:\ T_{ii}(w)=k \}$ can lie within $D_i^{(1)}$ in a complicated way. The transmission integrals are, however, quite insensitive to these topological complexities and depend only on the total size of the various dynamical quantities. Just as the points of $\mathcal{T}$ are not merely unions of subsets of $\mathcal{T}^*$, the passage from a field $\mathcal{T}$ of atomic forms to $\mathcal{T}^*$ cannot be achieved by merely replacing macroscopic dynamical terms by integrals, unless the f_i distribution over $\{ w:\ T_{ii}(w)=k \}$ sets is of a very special kind. When a single point represents an atomic form, the most that can be said is that a given form excites another form to a certain extent. No question of topologically bound perturbations within a point can possibly arise, and all topologically bound local variables

must have been homogenized out of existence. A precise sense can be given to this homogenizing process in the following way.

52. The Discrete V-Process: Spontaneous and Induced Changes of State

Let us be given the total embedding space M_1 associated with the point p_1. Represent M_1 by some smooth region $R_1 \subset \mathbb{R}^2$ of area M_1. Since only the area of this region is relevant, choose R_1 to be a square with sides $\sqrt{M_1}$ units long for specificity. Subdivide R_1 into a large number, say $\mu_1 M_1$, small subsquares $R_1 = \{ R_{1j}: j=1, 2, \ldots, \mu_1 M_1 \}$. Between every ordered pair (R_{1j}, R_{1k}) of subsquares, draw a directed line $l_{1j;1k}$. Now represent another point p_2 by a square R_2 of area M_2 such that $R_1 \cap R_2 = \emptyset$. Similarly define $R_2 = \{ R_{2j}: j=1, 2, \ldots, \mu_2 M_2 \}$ and the collection $\{ l_{2i;2j} \}$ of directed lines. Define collections of lines $\{ l_{1j;2k} \}$ and $\{ l_{2j;1k} \}$ in the obvious way.

Suppose now that every subsquare in $R_1 \cup R_2$ can be in precisely one of two states at any time, an active state or a passive state. For notational convenience, define an activity function V, $V: (R_1 \cup R_2) \times [0, T] \rightarrow \{0, 1\}$, such that

$$V(R_{ik}, t) = \begin{cases} 1 \text{ iff } R_{ik} \text{ is active at time } t \\ 0 \text{ iff } R_{ik} \text{ is passive at time } t \end{cases}$$

where $[0, T]$ is the time interval over which we consider the process. Let

$$s_i(t) \equiv \frac{1}{\mu_i} \cdot \int_{R_i} V(\lambda, t)\, dm(\lambda)$$

where $m(u)$ is the normalized counting measure. That is, the strength function s_i, evaluated at time t, gives the total area of the regions in R_i that are active at time t. Notice that s_i is a step function. μ_i is chosen so large, however, that the individual $\frac{1}{\mu_i}$ contributions are very small, whence s_i shall turn out to be very close to being a continuous and even smooth function.

One can interpret all remarks concerning derivatives and the like of s_i in the distribution sense, but such a venture is rendered particularly unnecessary by the fact that an improved interpretation of s_i will soon be given. From this definition of s_i, a dictionary of old terms can be recast in the new setting:

1) Transmission of Point Strength:

Each $l_{ik;jv}$ receives from R_{ik} a value $r p_{ik;jv}\left[\frac{V(R_{ik},t)}{\mu_i}\right]$, which pases through the node $N_{ik;jv}$ at time $t + T_{ik;jv}$ where it is further transformed to $r p_{ik;jv} c_{ik;jv}(t + T_{ik;jv})\left[\frac{V(R_{ik};t)}{\mu_i}\right]$. Thus R_{jv} receives a collection of

$$r p_{ik;jv}(V(R_{ik}, t-T_{ik;jv})/\mu_i)$$

values at every time t. Assume that these values act additively upon the recipient structure R_{jv}, just as in the macroscopic case. Then

$$r \ \Sigma_i \ \Sigma \left\{\left(\frac{p_{ik;jv} c_{ik;jv}(t)}{\mu_i}\right); R_{ik} \in \mathcal{A}_i(t-T_{ik;jv})\right\}$$

where $\mathcal{A}_i(t)$ $(\mathcal{A}_i'(t))$ is the set of active (passive) regions of R_i at time t, is the total transmitted input received at R_{jv} at time t.

2) The simplest interpretation of the appearance of a term $s_i(t-t_{ij})$ in the macroscopic equations, independent of k and v, is that $T_{ik;jv} = t_{ij}$. A more general interpretation will soon be given.

3) The effective embedding space $E_i(t) = \alpha (M_i - s_i(t))$ is, up to a proportionality constant, the total area of the passive regions at time t. The excitation term in the law for s_i growth expresses the fact that the rate with which passive regions become active at any time t is proportional to the product of the number of such regions and the size of the transmitted input received by each such region at t. Moreover, inputs to active regions have no effect on them. The growth law therefore says that the probability that a passive region $R \in \mathcal{A}_i'(t)$ becomes active at time t is proportional to

the total input received by R at t and, conditioned to $\mathcal{A}_i^!(t)$, is independent of the history of inputs received at R at all times $\tau < t$ since R last became passive. Moreover, the various passive regions in $\mathcal{A}_i^!(t)$ became active independent of each other.

4) The decay law $D_i = -\beta s_i$ says that the probability that a given active region $R \in \mathcal{A}_i(t)$ becomes passive at time t is constant through time, that R is independent of all input influences at times $\tau < t$ since R last become active, and that the elements of $\mathcal{A}_i(t)$ become passive independent of one another.

From this point of view, the active $\longrightarrow$ passive and passive $\longrightarrow$ active transitions are quite symmetric, if 1 in $D_i = -\beta(1-s_i)$ is interpreted to be a "virtual" input of constant value 1. That D_i represents a "spontaneous" decay process means that the total input $I(t)$ in $D_i = -\beta I s_i$ is a constant. When we deal with a field of mixed type, $D_i = -\beta s_i$ is replaced by a term like $D_i = -\beta s_i(1+\gamma \Sigma_{j\neq i} s_j(t-t_{ji})p_{ji}c_{ji})$. Here the total input is $I(t) = 1+\gamma \Sigma_{j\neq i} s_j(t-t_{ji})p_{ji}c_{ji}$, which is no longer constant and thus no longer purely "spontaneous." The decay process here is broken into two parts: "spontaneous" decay, and decay that is "induced" by $\Sigma_{j\neq i} s_j(t-t_{ji})p_{ji}c_{ji}$. The excitatory term $r\Sigma_j s_j(t-t_{ij})p_{ij}c_{ij}$ in $(M_i-s_i)(r\Sigma_j s_j(t-t_{ji})p_{ji}c_{ji})$ represents an "induced" growth process from this point of view.

We are thus given two states, $\mathcal{L}_1$ and $\mathcal{L}_2$, and a set of elements (or subregions) which can be in precisely one of these states at any time. $\mathcal{L}_1$ represents the state of being "passive" while $\mathcal{L}_2$ represents the state of being "active." Passage of a given element from one state to the other is independent of the behavior of all other elements sharing the same original state. A critical asymmetry in the transitions is that the transition of a given element from state $\mathcal{L}_2$ to $\mathcal{L}_1$ is either "spontaneous," in the sense that it obeys a time-invariant or stationary input law, or "induced" by fluctuating inputs impinging on the element. Transition from $\mathcal{L}_1$ to $\mathcal{L}_2$ is always actively "induced" by transmissions from other elements when they were in state $\mathcal{L}_1$ and by other excitatory external perturbations. This kind of

asymmetry in the transition laws between the "lower" state $\mathcal{L}_1$ and the "higher" state $\mathcal{L}_2$ is a pervasive property of embeddingequations. That $\mathcal{L}_1$ is an equilibrium state follows from it, and in a free embedding field, all elements pass gradually into the equilibrium state $\mathcal{L}_1$. When no self-excitations exist, this passage is entirely spontaneous. Otherwise, it occurs at a slower rate, but asymptotically it is inevitable.

This asymmetry in the transitions between $\mathcal{L}_1$ and $\mathcal{L}_2$ calls to mind the discussion of the absorption and emission of radiation by which Einstein explained Planck's law. If we are given two quantized energy states E_1 and E_2 with $E_1 < E_2$, then $\mathcal{L}_i$ is formally analogous to E_i, i=1, 2. Spontaneous $\mathcal{L}_2 \longrightarrow \mathcal{L}_1$ transitions are analogous to spontaneous $E_2 \longrightarrow E_1$ transitions with the emission of light. Induced $\mathcal{L}_2 \longrightarrow \mathcal{L}_1$ transitions correspond to $E_2 \longrightarrow E_1$ transitions induced by the presence of light of given frequencies, and $\mathcal{L}_1 \longrightarrow \mathcal{L}_2$ transitions correspond to $E_1 \longrightarrow E_2$ transitions induced by the absorption of radiation. In the absence of $E_1 \longrightarrow E_2$ transitions, all atoms in state E_2 pass to the lower energy state E_1, just as in a free embedding field, all elements in $\mathcal{L}_2$ pass into $\mathcal{L}_1$.

From the discussion of $E_i(t)$in (3), we see that the rate at which active regions are added to R_i due to excitation from R_j at time t is, letting $\mu = \mu_i = \mu_j$ for simplicity:

$$\psi_{ji}(t) = \alpha \int_{R_j}\int_{R_i} \left[\frac{1-V(R_{iv}, t)}{\mu}\right]\left[r\left(\frac{V(R_{jk}, t-T_{jk;iv})}{\mu}\right)p_{jk;iv}c_{jk;iv}(t)\right] dm(k)dm(v)$$

$$= \alpha r \int_{R_j}\int_{R_i} (1-V(R_{iv}, t))\left[V(R_{jk}, t-T_{jk;iv})p_{jk;iv}c_{jk;iv}(t)\right]\left(\frac{dm(k)}{\mu}\right)\left(\frac{dm(v)}{\mu}\right)$$

since this double integral adds up the contributions to excitatory growth over R_i at time t. Similarly, the old macroscopic variables p_{ji} and c_{ji} should appear as integrals of microscopic densities of the form

$$p_{ji} = \int_{R_j}\int_{R_i} \hat{p}_{jk;iv}\left(\frac{dm(k)}{\mu}\right)\left(\frac{dm(v)}{\mu}\right),$$

and

$$c_{ji}(t) = \int_{R_j}\int_{R_i} \hat{c}_{jk;iv}(t)\left(\frac{dm(k)}{\mu}\right)\left(\frac{dm(v)}{\mu}\right),$$

where $\hat{p}_{jk;iv}$ and $\hat{c}_{jk;iv}$ are undetermined densities. These functions, together with

$$s_i(t) = \int_{R_i} V(R_{ik}, t)\left(\frac{dm(k)}{\mu}\right)$$

and

$$M_i = \int_{R_i}\left(\frac{dm(k)}{\mu}\right)$$

must, by the correspondence principle, fulfill the relation

$$\psi_{ji}(t) = \alpha(M_i - s_i)(rs_j(t-t_{ji})\, p_{ji} c_{ji}),$$

which is the old macroscopic law for the growth of the collection of active regions. Written in full, with $dm_1(k) = dm(k)/\mu$, this demand becomes

$$\int_{R_j}\int_{R_i} (1-V(R_{iv}, t))V(R_{jk}, t-T_{jk;iv})p_{jk;iv}c_{jk;iv}(t)dm_1(k)dm_1(v) =$$

$$\left[\int_{R_i} (1-V(R_{iv}, t))dm_1(v)\right]\left[\int_{R_j} V(R_{jk}, t-t_{ji})dm_1(k)\right]\left[\int_{R_j}\int_{R_i} p_{jk;iv}dm_1(k)dm_1(v)\right].$$

$$\left[\int_{R_j}\int_{R_i} c_{jk;iv}(t)dm_1(k)dm_1(v)\right]$$

where we have made the now natural choice of densities $\hat{c}_{jk;iv} = c_{jk;iv}$ and $\hat{p}_{jk;iv} = p_{jk;iv}$. The macroscopic s_i growth process thus implies the statistical independence of certain microscopic densities. This is one sense in which passing from $\mathcal{T}^*$ to single point control form representations homogenizes the dynamics of $\mathcal{T}^*$. Statistical independence of this type means that each of the functions s_i, p_{ij}, and c_{ij}, representing processes which occur over disjoint regions of the structural carrier, are distributed in a chaotic way relative to another. Notice that $T_{jk;iv} = t_{ji}$ is not required. All that is necessary is that the phases of the transmission lags be so distributed around t_{ji} that when the integrals are split into independent parts, the mean of $V(\cdot,\cdot)$ may be evaluated over all R_{jk} at a single time. The equation for c_{ji} growth gives another example of statistical independence of this kind in the same way.

The present example shows that all microscopic densities for macroscopic equations give the same macroscopic process if they respond in the same way to the sum total of input excitation converging on each point. In this sense, macroscopic equations only weakly determine the microscopic process. We call the process considered above the V-process, after the activity function V.

53. The Graded G-Process: Dynamical Hierarchies

A more useful microscopic realization of the excitatory macroscopic embedding equations closely parallels the interpretation originally given to these equations. Let Ω_i be the area of a square R_i in $\mathbb{R}^2$, and divide R_i into a large number, say n_i subsquares $R_i = \{R_{ik}\}$ each of area h_i, so that $\Omega_i = n_i h_i$. Similarly define Ω_j for R_j, with $R_i \cap R_j = \emptyset$, $\Omega_j = n_j h_j$, $R_j = \{R_{jk}\}$ lines $\{l_{ik;jv}\}$ and nodes $\{N_{ik;jv}\}$. In this situation, it is again assumed that a random process is evolving, but this process will not be definable in terms of transitions between two levels $\mathcal{L}_1$ and $\mathcal{L}_2$. Rather, a continuum of levels $[0,\lambda]$, where $M_i = \lambda h_i$, $\lambda > 0$, will be considered for

every R_{ik}. The old macroscopic embedding equations for p_j will be interpreted to be the integrated form, over the entire collection R_j, of a graded process proceeding within each R_{jk} which is definable by a set of stochastic differential equations governing the densities of the macroscopic integrals. For example, if only R_j transmits to R_{ik}, we write

$$\text{(i)}\quad ds_{ik}/dt \propto (M_i - s_{ik})\Big(r \cdot \int_{R_j} s_{jv}(t-t_{jv;ik})p_{jv;ik}c_{jv;ik}\, dm(v) + I_{ik}\Big) - \beta s_{ik},$$

and

$$\text{(ii)}\quad dc_{ik;jv}/dt = \gamma^{+}_{ik;jv}(A_{ij} - c_{ik;jv})s_{ik}(t-t_{ik;jv})s_{jv} - \gamma^{-}_{ik;jv}c_{ik;jv}(M_i - s_{ik}(t-t_{ik;jv}))(M_j - s_{jv})$$

in close analogy with the original macroscopic case. Here, the input function I_{ik} is only a measurable function, and (i) and (ii) become stochastic differential equations for the densities s_{ik} and $c_{ik;jv}$. Both this process and the V-process satisfy the same macroscopic equations, so that the macroscopic process does not determine microscopic dynamics in this case. The two microscopic realizations generate the same macroscopic process and are quite different conceptually.

In equation (i), just as in the macroscopic case, s_{ik} is a level of excitation in R_{ik} whose maximum is $M_i = \lambda h_i$. The rate with which this excitation grows at time t is proportional to the product of the linear measure $M_i - s_{ik}$ of the number of excitation levels above s_{ik} and the total transmitted excitation received at time t. Each s_{ik} is itself an average over a random process proceeding within R_{ik}. s_{ik} is in first approximation to be thought of as an excitation spread uniformly over R_{ik} with density (s_{ik}/h_i). The fluctuations in this local R_{ik} process are so small compared to the fluctations between the various s_{jm} that they can be ignored in first approximation.

This random process within each R_{ik}, which we call the G-process, or graded process, is in all essential formal respects like the V-process over an entire collection R_i. For it is a process with independent increments of a fixed order of magnitude which represent the step-wise activation of a large finite population of elements. The essential difference between the two microscopic descriptions is that the G-process emphasizes that the total macroscopic process is the resultant of hierarchically ordered microscopic interactions. There are two levels of dynamical graining within the integrated G-process. Over each R_{ik} proceeds a nontrivially graded dynamical process. The existence of a large collection R_i of R_{ik}'s serves to replicate this process so often that its stability is assured in spite of random fluctuations in external inputs and in field transmissions to each R_{ik}.

Thus the growth of each s_{ik} depends on R_j transmission through

$$\int_{R_j} s_{jv}(t-t_{jv;ik})p_{jv;ik}c_{jv;ik}\,dm(v)$$

which is an average of excitation densities over the entire set R_j. The determination of the densities (s_{ik}/h_i) is also governed by an averaging process, which takes place within R_{ik}. s_{ik}/h_i is the expectation value of this process, and the deviations of local excitations within R_{ik} from s_{ik}/h_i are of a much smaller magnitude than the deviations of the various s_{ik} from their mean. The total G-process thus exhibits two simultaneously ongoing averaging processes, one far more finely grained than the other. Through the use of hierarchies of differently grained, simultaneously ongoing averaging processes, of which the present example is one of the simplest, neural interactions achieve an enormous ensemble stability using a comparatively small collection of macroscopic units.

In the present situation, local averages constantly readjust themselves in an intrinsic way according to the influence of the entire distribution of these

averages. A similar situation earlier arose in discussing the effect of multiple line structures on a strength field of relatively uniform type. The G-process is a microscopic analog of that situation, and shows how well the hierarchical character of neurological geometry and of neurological dynamics are suited to one another. In fact, the similarity between the two types of hierarchy is so close that the possibility of finding an appropriate line structure for the ultrafine excitation dynamics within each R_{ik} becomes highly plausible. Such a line structure will already have a chemical interpretation.

Although the macroscopic equations do not completely determine microscopic densities, we see that the V- and G-processes differ only in the specification of the underlying hierarchy of averaging processes which generates them. The insensitivity of the macroscopic process to differences in hierarchy of this kind makes good behavioral sense. In the emission of verbal units, for example, we are not aware of any duality or more complicated hierarchical partition of verbal forms corresponding to the hierarchical dynamics and geometry generating these forms. Nature has skillfully introduced hierarchies to improve the stability of our daily affairs without denying us the sense of a smoothly flowing interaction with our environment.

54. Volume Conductors, Axons, Endbulbs, and Transmitters

Each R_{ik} of the G-process represents a bona fide geometrical entity with a dynamical behavior of its own. We call such an entity a cell, or, more properly, a caricature of a cell. R_{ik}, as a cell, is a body in $\mathbb{R}^3$ bounded by a closed surface called a membrane. The fact that (s_{ik}/h_i) is supposed to represent a homogeneous excitation density within R_{ik} means that R_{ik} is a perfect volume conductor and that (s_{ik}/h_i) is the excitatory potential per unit area, up to a scaling factor, of R_{ik}. The perfection of R_{ik} as a volume conductor must always be measured relative to the next coarser level of graining within the system, which is here between various cells R_{ij}. The lines passing from one cell to another are collections of still finer lines,

each terminating in nodes as before. These finer lines are called fibers, or axons, and the finer nodes endbulbs, again with unambiguous intent. When each $N_{ik;jv}$ is considered as a collection of bodies in $\mathbb{R}^3$, the correspondence principle requires that these bodies be distributed along the membrane of R_{jv}. Moreover, the transmission contributions from all of the $N_{ik;jv}$ terminating on R_{jv} combine additively to excite R_{jv}. This excitation process must therefore occur by way of local processes at the node terminals impinging on the R_{jv} membrane, and these local processes must induce potential changes that are immediately propagated throughout the volume of R_{jv} to determine the new uniform excitation density (s_{jv}/h_j) by an intracellular averaging process.

Since the individual cells are still very small objects under the eye of the present description, $p_{ik;jv}$ and $c_{ik;jv}$ are thought of as averages over the bundles of fibers and endbulbs passing from cell R_{ik} to cell R_{jv}. As with the various s_{ik}, the process giving each average $c_{ik;jv}$ over the collection $N_{ik;jv}$ of endbulbs is a finely grained one compared to the distribution of the various $c_{ik;jv}$. The formal similarity between $A_{ji} \sim c_{jv;ik}$ and $M_i \sim s_{ik}$, and between $\sim \beta s_{ik}$ and $\sim \bar{\gamma}_{jv;ik} c_{jv;ik}$ shows that $c_{jv;ik}$ represents a level of excitation chosen from a continuum of possible levels. Letting each $N_{jv;ik}$ be represented by a square of area h_{ij} such that $A_{ij} = \tau h_{ij}$, $\tau > 0$, the excitation density over $N_{ik;jv}$ is $(c_{ik;jv}/h_{ij}) \in [0,\tau]$. When each $N_{ik;jv}$ is considered as a collection of nodal bodies in $\mathbb{R}^3$ at the end of the axons from R_{ik} to R_{jv}, this excitation density is to be thought of as being spread over all of the bodies. Since the various nodal bodies contribute additive excitations to R_{jv} from the single source R_{ik}, it is clear that the exact distribution of excitation over the $N_{ik;jv}$ nodes is important only if the fluctuations within the $l_{ik;jv}$ lines are not chaotic.

If the excitation density within R_{ik} at time t is $s_{ik}(t)/h_i$, then $r s_{ik}(t) p_{ik;jv}$ is transmitted over the axons $l_{ik;jv}$ and reaches $N_{ik;jv}$ at time $t+T_{ik;jv}$. This excitation mobilizes a process within $N_{ik;jv}$ that is strongly coupled to the quantity measured by $c_{ik;jv}$. This coupling generates

a local process which emits a quantity that passes out of $N_{ik;jv}$ to the R_{jv} membrane. The effect of this local process on R_{jv} is to cause the potential $s_{jv}(t+T_{ik;jv})$ to grow at a rate proportional to $rs_{ik}(t) \cdot p_{ik;jv} c_{ik;jv}(t+T_{ik;jv})$.

Let $N_{ik;jv}(t)$ denote $N_{ik;jv}$ considered at time t, and let $\overline{N}_{ik;jv}(t)$ denote the joint $N_{ik;jv}$-R_{jv} membrane complex at time t. We use this terminology to discuss the excitatory term

$$\gamma^{+}_{ik;jv}(A_{ij}-c_{ik;jv})s_{ik}(t-T_{ik;jv})s_{jv}$$

determining $c_{ik;jv}$ growth. A_{ij} gives the total capacity of an activation process taking place within $N_{ik;jv}$: A_{ij} measures the total population of elements subserving this activation process. $(A_{ij}-c_{ik;jv}(t))/h_{ij}$ is the density of inactive elements of this process within $N_{ik;jv}(t)$. These elements cannot be activated in the absence of transmitted excitation from the cells R_{ik} and R_{jv}. The excitation from R_{ik} is transmitted over $l_{ik;jv}$ and reaches $N_{ik;jv}(t)$ in the amount $rs_{ik}(t-T_{ik;jv})p_{ik;jv}$. R_{jv}, in turn, induces a process which affects $N_{ik;jv}(t)$ by quantity proportional to s_{jv}. All of the processes involving transformations from R_{ik}- and R_{jv}-excitations to quantities used by $N_{ik;jv}(t)$ take some time to be activated, but we suppose that this time is so small compared to $T_{ik;jv}$ that it can be ignored in first approximation. Joint simultaneous excitation from R_{ik} and R_{jv} at $N_{ik;jv}(t)$ is necessary for the activation of the inactive elements, which exist in $N_{ik;jv}(t)$ with density $(A_{ij}-c_{ik;jv}(t))/h_{ij}$. The processes induced by this joint simultaneous excitation are coupled in such a way that the probability that an inactive element is activated is proportional to $(rs_{ik}(t-T_{ik;jv})p_{ik;jv})s_{jv}$. Since the inactive elements are activated independently, the rate at which inactive elements become activated is $\gamma^{+}_{ij;kv}(A_{ij}-c_{ik;jv})s_{ik}(t-T_{ik;jv})s_{jv}$.

The active elements of $N_{ik;jv}$ become inactive according to a process that is dual to the inactive $\longrightarrow$ active process. These elements exist in $N_{ik;jv}(t)$ with density $c_{ik;jv}(t)/h_{ij}$. They become inactive at a rate that

depends on how strongly the coupling condition governing inactivate $\longrightarrow$ active transitions is violated. One measure of the extent to which this $N_{ik;jv}(t)$ coupling is violated, and the one which we have been using, is proportional to $(M_i - s_{ik}(t-T_{ik;jv}))(M_j - s_{jv}(t))$. Since the active elements become inactive independent of one another, the rate with which active inactive transitions occur is $\gamma_{ij;kv} c_{ik;jv}(M_i - s_{ik}(t-T_{ik;jv}))(M_j - s_{jv})$. This decoupling process means that both the R_{ik}- and the R_{jv}-induced $N_{ik;jv}(t)$ activation processes must be inoperative for active $\longrightarrow$ inactive transitions to occur. If the absence of only one of these activation processes is sufficient to induce active $\longrightarrow$ inactive transitions, then these transitions occur at a rate proportional to

$$M_i M_j - s_{ik}(t-T_{ik;jv})s_{jv} = (M_i - s_{ik}(t-T_{ik;jv}))s_{jv} + (M_j - s_{jv})s_i(t-T_{ik;jv}) + (M_i - s_{ik}(t-T_{ik;jv}))(M_j - s_{jv})$$

If the rate of inactivation depends on R_{ik}- and R_{jv}-uncoupling to different degrees, then this rate is proportional to

$$\xi_{ik;jv}(\alpha_1, \alpha_2, \alpha_3; t) \equiv c_{ik;jv}\left[\alpha_1(M_i - s_{ik}(t-T_{ik;jv}))s_{jv} + \alpha_2(M_j - s_{jv})s_i(t-T_{ik;jv}) + \alpha_3(M_i - s_{ik}(t-T_{ik;jv}))(M_j - s_{jv})\right].$$

$\xi_{ik;jv}(\alpha_1, \alpha_2, \alpha_3; t)$ includes all the other cases by proper setting of $(\alpha_1, \alpha_2, \alpha_3)$. If joint R_{ik}-R_{jv} uncoupling is not a necessary precursor to $c_{ik;jv}$ inactivation in certain processes, then it is possible that there exist processes for which joint R_{ik}-R_{jv} coupling is not a necessary precursor to $c_{ik;jv}$ activation. Thus the $(A_{ij} - c_{ik;jv}, c_{ik;jv})$ and the $(s_{ik}(t-T_{ik;jv})s_{jv}, \ldots, (M_i - s_{ik}(t-T_{ik;jv}))(M_j - s_j))$ vectors can be coupled in

all possible ways, with appropriate coupling coefficients, in more general processes. If the active-inactive duality for $c_{ik;jv}$ is insufficient to describe certain $\bar{N}_{ik;jv}$ processes, the next step is to consider several $c_{ik;jv}$ functions $\{c^{(n)}_{ik;jv}: n=1, 2, \ldots, N\}$ which are coupled to one another and to the vector $(s_{ik}(t-T_{ik;jv})s_{jv}, \ldots, (M_i - s_{ik}(t-T_{ik;jv}))(M_j - s_{jv}))$ in the appropriate way. We then study systems

$$ds_{ik}/dt = X_{ik}(s_{ik}, \{s_{jm}(t-T_{jm;ik})\}, \{c^{(n)}_{jm}\}, \ldots),$$

$$dc^{(m)}_{ik;jv}/dt = Y^{(m)}_{ik;jv}(s_{ik}(t-T_{ik;jv}), s_{jv}, \{c^{(n)}_{ik;jv}\}, \{c^{(n)}_{ik;jv}(t-T_{nm})\} \ldots).$$

These more exotic couplings do not, however, appear directly in psychological data. They are averaged out in the ensemble behavior. The strong coupling combination $(A_{ij} - c_{ik;jv}, c_{ik;jv}; s_{ik}(t-T_{ik;jv})\, s_{jv}, (M_i - s_{ik}(t-T_{ik;jv}))\cdot(M_j - s_{jv}))$, with which we began this study, and the weaker coupling $(A_{ij} - c_{ik;jv}, c_{ik;jv}; s_{ik}(t-T_{ik;jv})s_{jv}, M_iM_j - s_{ik}(t-T_{ik;jv})s_{jv})$ seem to be the simplest and most symmetric couplings that arise as ensemble densities.

The "elements" which are involved in the $N_{ik;jv}(t)$ and $\bar{N}_{ik;jv}(t)$ processes must be interpreted in a general sense. An "element" need not correspond to a stable physical object, but can refer to an abstract structure which changes its physical state through time. For example, $c_{ik;jv}(t)$ can represent the degree to which provision for the production of a chemical transmitter within $N_{ik;jv}(t)$ has evolved within the endbulbs as a result of coupled R_{ik} and R_{jv} excitation derivatives in $\bar{N}_{ik;jv}(w)$, $w \leq t$. These provisions for transmitter production need not be identical with the transmitter itself. Suppose nonetheless for a moment that $c_{ik;jv}(t)/h_{ij}$ does measure the density of transmitter that has accumulated in $N_{ik;jv}(t)$. Then the fact that the effective excitation of R_{jv} by R_{ik} - transmission is proportional to $rs_{ik}(t-T_{ik;jv})p_{ik;jv}c_{ik;jv}(t)$ means that a quantity of transmitter

must be subtracted from the amount of $c_{ik;jv}(t)$ in $N_{ik;jv}(t)$, for part of this transmitter is actively released from $N_{ik;jv}(t)$ into the subsynaptic cleft. Suppose, in particular, that the amount of transmitter released is proportional to the rate with which $s_{jv}(t)$ growth is augmented by $l_{ik;jv}$ transmission. Then we must subtract an additional quantity proportional to $s_{ik}(t-T_{ik;jv})p_{ik;jv}c_{ik;jv}(t)$ from the equation determining the behavior of $c_{ik;jv}$. We therefore find an equation of the form

$$dc_{ik;jv}/dt = (A_{ij}-c_{ik;jv})Q_1 - c_{ik;jv}Q_2$$

$$- \nu s_{ik}(t-T_{ik;jv})p_{ik;jv}c_{ik;jv},$$

where Q_1 and Q_2 are coupling terms.

On the other hand, suppose that $c_{ik;jv}$ is taken to represent a cellular transmitter potentiality which controls the production of available transmitter, but is not itself the quantity of available transmitter. Let the amount of available transmitter be proportional to the potentiality $c_{ik;jv}$, with perhaps a small lag between changes in the control process and in transmitter production. A process which controls transmitter production in this way can be realized by broadly distributing a large number of transmitter producing mechanisms of a single type throughout $N_{ik;jv}$. Each such mechanism, when it is activated, produces transmitter in packets of uniform size at a fixed rate. The various mechanisms can act independently of one another, and $c_{ik;jv}(t)$ measures, up to a scaling factor, the total number of active mechanisms in $N_{ik;jv}(t)$. If it is now assumed that the available transmitter is released in an amount proportional to the R_{ik}-induced excitation impinging on $N_{ik;jv}(t)$, then once again a quantity proportional to $s_{ik}(t-T_{ik;jv})p_{ik;jv}c_{ik;jv}(t)$ is transmitted to R_{jv}, but now the $c_{ik;jv}(t)$ supply is not depleted by this transmission process and the old equations hold without change.

How does the process of transmitter release occur? Let $N^b_{ik;jv}$

denote that part of the membrane surface of $N_{ik;jv}$ which impinges upon R_{jv}. The above reasoning strongly suggests that the transmitter is broadly distributed over $N^b_{ik;jv}$, for the determination of $c_{ik;jv}$ is closely related to a coupling of R_{ik}-R_{jv} activity. The density of the transmitter at $N^b_{ij;kv}$ is therefore nearly proportional to $c_{ik;jv}/h_{ij}$. A homogeneity argument shows that the excitation density transmitted from R_{ik} to $N_{ik;jv}(t)$ must also be broadly distributed over $N^b_{ik;jv}(t)$. The fact that the product $s_{ik}(t-T_{ik;jv})c_{ik;jv}(t)$ controls the release of transmitter may thus be interpreted as follows: The excitation of local regions of $N^b_{ik;jv}(t)$ releases quantities of transmitter which are jointly proportional to the concentration of transmitter packets and to the degree to which the activated membrane allows the contents of transmitter packets to pass through. The degree of activation of regions of this membrane which are chosen sufficiently large that they contain a large number of transmitter releasing mechanisms is, in turn, proportional to $s_{ik}(t-T_{ik;jv})$. The transmission term $s_{ik}(t-T_{ik;jv})c_{ik;jv}(t)$ is therefore the expectation value of a random process, with two statistically independent components, which is going on in $N^b_{ik;jv}(t)$ and the cellular surround. $N^b_{ik;jv}$ is a large structure compared to the size of a single transmitter packet unit and many such units exist which contribute to the random process. Microscopically, this process resembles a Poisson process with two time-dependent intensity parameters, one governing the concentration of available transmitter packets and the other governing the concentration of transmitted excitation density.

The statistical independence of the $c_{ik;jv}(t)$ and the $s_{ik}(t-T_{ik;jv})$ membrane processes reveals a coarseness of dynamical graining within $N_{ik;jv}$ that is perfectly suited to the fact that $N_{ik;jv}$ activates a volume conductor R_{jv} whose s_{jv} function is itself the resultant of an averaging process at precisely the same level of dynamical graining. The $R_{ik} \longrightarrow N_{ik;jv} \longrightarrow R_{jv}$ interaction, given by $s_{ik}(t) \longrightarrow s_{ik}(t+T_{ik;jv})c_{ik;jv}(t) \longrightarrow s_{jv}(t+T_{ik;jv}+\epsilon)$, $\epsilon << T_{ik;jv}$, is thus a phenomenon of <u>parallel dynamical graining</u>. This parallelism means that, when we consider the whole cell

as a dynamical averaging mechanism, the neurological hierarchy reduces to a single hierarchical level which is ideal for the stable propagation of excitatory or inhibitory perturbations of a given dynamical type between cells. Many variables attached to local regions of $N_{ik;jv}$ contribute to the production of this parallel transmission process, but in first approximation, these variables average out in the emsemble behavior of whole cells. The way in which these local cellular variables average out in the study of cell ensembles is analogous to the way in which certain parts of a core trajectory become suppressed in the study of macroscopic interactions of atomic verbal forms. Both omissions are examples of an intrinsic excision property in the neural geometrico-dynamical hierarchy.

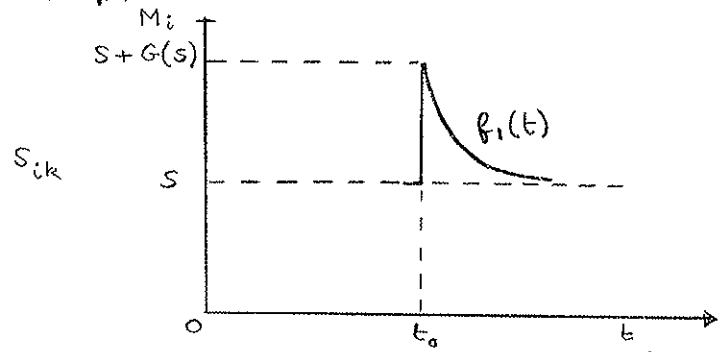
55. Spikes and An Invariance Principle for Transmission Between Cells

When R_{ik} has excitation density $s_{ik}(t)$, $N_{ik;jv}(t+T_{ik;jv})$ receives transmitted excitation over $l_{ik;jv}$ that enters into $N_{ik;jv}(t+T_{ik;jv})$ processes in an amount proportional to $s_{ik}(t)$. Fluctuations in R_{ik} activation are thus communicated over $l_{ik;jv}$ by a graded excitation process, and changes in $s_{ik}(t)$ effect proportional changes in the magnitude of the $N_{ik;jv}(t+T_{ik;jv})$ processes dependent on R_{ik} excitation. Since changes in $R_{ik}(t)$ reach $N_{ik;jv}(t+T_{ik;jv})$ without a decrement and with a fixed transmission lag $T_{ik;jv}$, they must be transmitted in nondecaying waves of excitation over $l_{ik;jv}$. These waves are called spikes. It is not *a priori* necessary that all of these excitation waves have the same shape. Some can be higher than others or wider than others. All that is important is that $N_{ik;jv}$ receives enough spikes so that the laws governing the ensemble behavior of the $c_{ik;jv}$-control mechanisms and the transmitter release from $N_{ik;jv}$ are preserved. One way of achieving this is to require that every steady state potential level in R_{ik} is associated with a steady frequency of spiking along $l_{ik;jv}$ with spikes of a given form. The rate of spiking alone is not

sufficient to determine this system unless the spikes generated at all steady potential levels have precisely the same form.

Suppose, for example, that associated with an increment of size Δs_{ik} in R_{ik}-potential is a non-trivial decrease in spike height of size ΔG_{ik}, while the rate of spiking is proportional to $s_{ik}(t)$. Also suppose that the $N_{ik;jv}$ processes are determined by the total potential transmitted to R_{jv} from R_{ik} in unit time. Since the height of every spike decreases with increasing potential, while the rate of spiking is proportional to the potential, the breadth of each spike must increase with $s_{ik}(t)$ in order to preserve the $R_{ik} \longrightarrow N_{ik;jv}$ map $s_{ik}(t) \longrightarrow rp_{ik;jv}s_{ik}(t-T_{ik;jv})$. Thus the mean decay rate of spikes generated at higher potentials would necessarily be slower than the decay rate of spikes generated at lower potentials. Moreover, if the shape of spike, at all potential levels, can be given by a curve depending on a small number of parameters, then the spike decay rate can be computed as a function of the steady potential. A simple example of this possibility is the following.

Given a steady potential level s, let the spike height $G(s)$ be proportional to $M_i - s$: $G(s) = a(M_i - s)$, $a < 1$. Approximate the form of a spike by $f(t)$ in the simple diagram



Here $f(t)$ has an almost instantaneous climb from s to its maximum $s + G(s)$ followed by an exponential decay. Thus

$$f(t) = \begin{cases} s+G(s)e^{-k(s)(t-t_o)} & t \geqslant t_o \\ s & t < t_o \end{cases}$$

We must solve for $k(s)$. We have supposed that $N_{ik;jv}$ activity is determined by the total integral of R_{ik}-induced activation per unit time. We measure time here relative to the rate of ensemble fluctuations in $N_{ik;jv}$, and assume that every spike decays so quickly that practically all of its mass fits into one time unit in the $N_{ik;jv}$ time scale. Since the rate of spiking is proportional to the steady state potential, the $R_{ik} \longrightarrow N_{ik;jv}$ law requires that the total mass of an individual spike be independent of s, or

$$\int_{t_o}^{\infty} G(s)e^{-k(s)(t-t_o)}dt = \text{constant} = \lambda$$

Hence

$$\int_{o}^{\infty} e^{-k(s)t}dt = \lambda/G(s) = \lambda/a(M_i-s),$$

and

$$k(s) = G(s)/\lambda = a(M_i-s)/\lambda .$$

The method holds, of course, for arbitrary $G(s)$. If the rate of spiking $w(s)$ is not proportional to s, and the $R_{ik} \longrightarrow N_{ik;jv}$ law still holds, the total mass of individual spikes must vary with s in a compensatory way, and

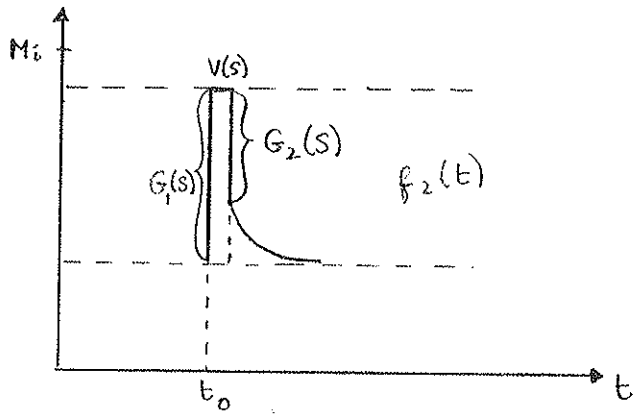
$$\int_{o}^{\infty} G(s)e^{-k(s)t}dt = \lambda s/w(s),$$

so that

$$k(s) = G(s)w(s)/\lambda s. \qquad \text{(I)}$$

Whenever spikes of the form $f_1(t)$ are characteristically found in a given situation, all of the quantities in formula (I) are directly measurable. Since formula (I) was derived under the assumption of particular $R_{ik} \longrightarrow N_{ik;jv}$ laws, the satisfaction of (I) in this situation becomes a check of whether or not the $R_{ik} \longrightarrow N_{ik;jv}$ law holds. $R_{ik} \longrightarrow N_{ik;jv}$ laws are not themselves as easily measured, for their significance becomes clear only by simultaneously studying R_{ik} and $N_{ik;jv}$ activity. Formulas such as (I) are thus most helpful whenever they can be applied, for they provide simple and direct estimates of what the transmission law must be.

Another example is



Here $f_2(t)$ climbs almost immediately from s to its asymptote $G_1(s)+s$, lingers at asymptote for a time $v(s)$, and then quickly falls $G_2(s)$ units, where it enters into a period of more gradual exponential decay. Again suppose that the $R_{ik} \longrightarrow N_{ik;jv}$ law is given as above, and that the spiking rate is $w(s)$. Then

$$\lambda s/w(s) = \int_{t_0}^{\infty} f_2(t)dt = G_1(s)v(s) + (G_1(s)-G_2(s)) \int_0^{\infty} e^{-k(s)t}dt$$

$$= G_1(s)v(s) + (G_1(s)-G_2(s))/k(s),$$

and

$$k(s) = (G_1(s)-G_2(s))/(\lambda s/w(s)-A(s)), \qquad \text{(II)}$$

where $A(s) = G_1(s)v(s)$ is the area of the initial rectangular spike. Whenever spikes of $f_2(t)$ form are characteristically found, (II) becomes

a check of the $R_{ik} \longrightarrow N_{ik;jv}$ transmission law.

Suppose that the $R_{ik} \longrightarrow N_{ik;jv}$ transmission law is $s_{ik}(t) \longrightarrow rp_{ik;jv}s^2_{ik}(t)$. This squaring operation can emerge at various stages of the transmission process: either when local R_{ik} strength is transformed into spikes, or when spikes take their effect on $N_{ik;jv}$ activity, or both. Suppose for specificity that the squaring takes place during the transmission process itself and before $N_{ik;jv}$ is reached. Then s in (I) and (II) must be replaced by s^2, and we find instead of (I),

$$k(s) = G(s)w(s)/\lambda s^2 \qquad (I'),$$

and instead of (II):

$$k(s) = G_1(s)-G_2(s))/(\lambda s^2/w(s)-A(s)) \qquad (II').$$

For f_1 spikes, therefore, if $k(s)/G(s)w(s)$ varies like $1/s$, the transmission law $s_{ik}(t) \longrightarrow rp_{ik;jv}s_{ik}(t)$ is favored, while if $k(s)/G(s)w(s)$ varies like $1/s^2$, the transmission law $s_{ik}(t) \longrightarrow rp_{ik;jv}s^2_{ik}(t)$ is favored. Similarly, for f_2 spikes, the variation of

$$w(s)\left[A(s) + (G_1(s)-G_2(s))/k(s)\right]$$

as s^m suggests the $s_{ik}(t) \longrightarrow rp_{ik;jv}s^m_{ik}(t)$ transmission law, while its variation as any function $g(s)$ suggests the $s_{ik}(t) \longrightarrow rp_{ik;jv}g(s_{ik}(t))$ law.

We can turn the method of these simple examples around to get a general method for finding transmission laws. Given any transmitting cell, it has a fixed $R_{ik} \longrightarrow N_{ik;jv}$ transmission law, which we possibly do not know. We begin with the nicest cells possible: those for which a particular spike shape $f(s,\cdot)$ corresponds to every steady state potential s, at least in the mean. For these cells we can directly measure (a stable average) spike height $G(s)$ as a function of steady-state potential s. We also fit

a curve $f(s,\cdot)$ which represents the temporal potential gradient of a standard spike that is transmitted over $l_{ik;jv}$. We do this with the simplest possible curves, since all that matters is $N_{ik;jv}$'s ensemble behavior. Corresponding to this coarseness of graining is the fact that a standard spike is an average of many electrode readings. We wish to find the $R_{ik} \longrightarrow N_{ik;jv}$ transmission law using this data. The search for this transmission law often reduces to the specification of a functional F of $(f(s,\cdot), w(s))$ such that $F(f(s,\cdot), w(s)) =$ constant, where $w(s)$ is the directly measurable rate of spiking per unit time and the unit time is measured relative to the reaction rates of the $N_{ik;jv}$ ensemble to $l_{ik;jv}$ transmission. The simple condition of this kind in our examples had as its F

$$F(f(s,\cdot), w(s)) = \frac{w(s)}{g(s)} \cdot \int_0^\infty f(t;k_1(s), k_2(s), \ldots, k_n(s))dt,$$

where $g(s) = s^m$ and the $k_i(s)$ are parameters determining f as a function of s. The examples show that if we can find such an F, for all s, then we are well on the way to knowing the $R_{ik} \longrightarrow N_{ik;jv}$ transmission law governing the situation. Knowing $G(s)$ often helps in the search for F, as in the examples, both since $G(s)$ helps to characterize many smooth curves which have just one maximum, and since we expect $N_{ik;jv}$ to respond to a homogeneous average of the $f(s,\cdot)$'s.

This discussion admits the possibility that different cells can sustain spike generating mechanisms which differ in detail even though they preserve important underlying ensemble invariances in the $R_{ik} \longrightarrow N_{ik;jv}$ transmission process. These differences in detail, such as differences in rates of spiking at fixed potential levels, differences in spike heights and the decay rates of individual spikes, and so on, compensate one another to preserve these underlying invariances.

The discussion also suggests the possibility that the $R_{ik} \longrightarrow N_{ik;jv}$ transmission law $s_{ik}(t) \longrightarrow rs_{ik}(t)p_{ik;jv}$ is not universal in all transmitting

nerve cells. Why should this law not be replaced by $s_{ik}(t) \longrightarrow rs_{ik}^2(t-T_{ik;jv})p_{ik;jv}$, for example? A condition which is universally applicable to all transmitting cells, and which restricts the class of possible transmission laws, is the following.

Principle: Whatever be the transmission law $s_{ik}(t) \longrightarrow K(s_{ik}(t))$, it must be such that the excitation densities of the free embedding field which it generates asymptotically approach their equilibrium potentials on a global scale. The equilibrium potential with which we have been dealing to now has been zero. The transmission law $K(s_{ik}(t)) = rs_{ik}^2(t)p_{ik;jv}$ can indeed be made to satisfy this principle by properly choosing the rate constants α, β, $\gamma^+_{ik;jv}$, and $\gamma^-_{ik;jv}$. Whenever we have a free embedding field, these rate constants and the transmission law must be jointly chosen to satisfy the principle. Otherwise, the effects of old inputs to the field will never fade and embedding dynamics will reduce to a mass of confusion.

56. The K_1 Transmission Law: Dynamical Indistinguishability Implies Geometrical Indistinguishability

$K_1(s_{ik}(t)) = rp_{ik;jv}s_{ik}(t)$ and $K_2(s_{ik}(t)) = rp_{ik;jv}s_{ik}^2(t)$ are both admissible under the previous principle. Is there any other way to distinguish these two laws? K_1 is unique in the following sense. Suppose that $R_{jv}(t)$ receives a total input

$$T_{jv} = r\sum_{i,k} p_{ik;jv}s_{ik}(t-T_{ik;jv}).$$

Partition the set $\{R_{ik}\}$ of all cells into maximal subsets $T^{jv}_1, \ldots, T^{jv}_N$ such that $R_{ik} \in T^{jv}_n$ if and only if $T_{ik;jv} = t_n$, $n=1,2,\ldots,N$. Write T_{jv} as

$$T_{jv} = r\sum_{n=1}^{N} \sum_{i(T^{jv}_n)} p_{ik;jv}s_{ik}(t-t_n).$$

where $i(W)$ is the set of indices corresponding to elements of W. Partition each T_n^{jv} into maximal subsets $\{T_{nm}^{jv},\ m=1,2,\ldots,k_n\}$, such that every point in T_{nm}^{jv} has received precisely the same total inputs through time and started out with the same initial value. Thus $s_{ik}(t) \equiv s(n,m;t)$ if and only if $R_{ik} \in T_{nm}^{jv}$. Write T_{jv} as

$$T_{jv} = r \sum_{n=1}^{N} \sum_{m=1}^{k_n} \Big(\sum_{i(T_{nm}^{jv})} p_{ik;jv} \Big) s(n,m;t-t_n)$$

$$= r \sum_{n=1}^{N} \sum_{m=1}^{k_n} P(n,m)\, s(n,m;t-t_n),$$

where $P(n,m) = \sum_{i(T_{nm}^{jv})} p_{ik;jv}$.

The partition $\{T_{nm}^{jv}\}$ of $\{R_{ik}\}$ divides all the cells into maximal classes such that all elements in any T_{nm}^{jv} are <u>dynamically indistinguishable</u> when considered in terms of their effects on the dynamics of R_{jv}. All elements of any T_{nm}^{jv} exhibit the same dynamical behavior at any time and take the same amount of time to transmit to R_{jv}. Calling this kind of indistinguishability <u>dynamical indistinguishability modulo</u> R_{jv}, the K_1 transmission law allows us to say: The partition $\{T_{nm}^{jv}\}$ is a partition of $\{R_{ik}\}$ into dynamically indistinguishable sets, mod R_{jv}, as well as a partition of R_{ik} into <u>geometrically</u> indistinguishable sets, mod R_{jv}.

Geometrical indistinguishability means the following. Whenever two points R_{ik} and R_{uw} are dynamically indistinguishable, mod R_{jv}, it is true in particular that their excitation densities are the same. We can thus lump R_{ik} and R_{uw} into a single point $R_{ik,uw}$, with M value M_i+M_u, without changing the local dynamics of R_{ik} and R_{uw}. If $K \neq K_1$ + constant, however, the $R_{ik}+R_{uw} \longrightarrow R_{ik,uw}$ lumping process, which does not affect local R_{ik} and R_{uw} dynamics or transmission lag times to R_{jv}, does distort the transmission pattern from the total field to R_{jv}. Unless

$\kappa = \kappa_1 +$ constant, the $R_{ik;uw} \longrightarrow R_{jv}$ transmission is not a simple composite of the $R_{ik} \longrightarrow R_{jv}$ and $R_{uw} \longrightarrow R_{jv}$ transmissions. Yet given any R_{jv}, when we look at any two cells R_{ik} and R_{uw} in the same T^{jv}_{nm} separately and consider their interactions with R_{jv} alone, R_{ik} and R_{uw} cannot be distinguished. Since their boundary membranes distinguish nothing about the behavior of the two cells, we should be able to dissolve this membrane conceptually and to join the two cells into a single larger unit without changing R_{jv}-dynamics. This is only possible if $\kappa = \kappa_1 +$ constant, and this constant must be zero so that the associated free embedding field converges to the equilibrium potential zero. $\kappa = \kappa_1$ is thus singled out as a privileged transmission law when we study volume conductors. By lumping together all $R_{ik} \in T^{jv}_{nm}$, for all n and m, we find an embedding field that is equivalent to the old field, modulo R_{jv}, whose points have M values $\Sigma \{ M_{ik} : R_{ik} \in T^{jv}_{nm} \}$. This field is called the <u>minimal field (of the second kind)</u>, <u>modulo</u> R_{jv}. All of our work should be carried out with the minimal field of a given situation, since this field distinguishes all of the features of the situation in the most parsimonious and intrinsic way. The use of a minimal field also reminds us that we should not expect to see individual spikes in a description of the interactions of large numbers of whole cells. Since the system behaves as if all dynamically indistinguishable cells are clustered into a single unit, the different phase relations of the individual spikes are averaged out over a given cluster and we receive the impression of a flow of strength from one cluster of cells to another. Introducing new input asymmetries introduces new membrane boundaries into the cluster of cells forming a given T^{jv}_{nm}, and the T^{jv}_{nm} partition must be refined. This refinement is directly analogous to the refinement of partitions required to maintain propagating symmetries in sets of points. It is augmented in the present setting by the rule: draw boundaries only when they distinguish dynamical differences.

We will not always be studying such simple objects as volume conductors. Yet we still have the following principle to guide us and to check against future examples:

Principle: Given any set of embedding field equations, the transmission law K is always chosen so that dynamically indistinguishable cells are also geometrically indistinguishable. This principle also applies to systems for which a simple superposition of individual input contributions does not hold. In the statement of the principle for such systems, K is replaced by (Q, K), where Q is the functional determining how individual input contributions combine to influence a given cell.

57. Macroscopic Fields are Equivalent to Statistically Independent, Excised Microscopic Subfields

The above discussion is an introduction to neural dynamics proper using embedding fields. This discussion shall be extended and refined in various directions in the following pages. It has been brought about by a consideration of purely macroscopic psychological data. All discussions of spiking laws, the control of transmitter accumulation and its release, etc., could have been phrased in different terms, but the laws themselves would nonetheless have followed from the macroscopic psychological equations and the correspondence principle without an independent neurological inquiry, as they did when I first considered them. Macroscopic psychology and microscopic neurology thus share a tight dynamical bond. The study of one of these levels of experience cannot fail to shed light on the other.

For completeness in discussing the G-process, we now check to see when the microscopic s_{ik} equations generate the macroscopic s_i equations.

$$s_i = \int_{R_i} s_{ik} dm(k),$$

$$I_i = \int_{R_i} I_{ik} dm(k),$$

so that

$$\frac{ds_i}{dt} = \frac{d}{dt}\int_{R_i} s_{ik}\,dm(k) = \int_{R_i}\frac{d}{dt}\,s_{ik}\,dm(k) = \alpha\int_{R_i}(M_i - s_{ik})\cdot$$

$$\left[\gamma\int_{R_j} s_{jv}(t-t_{jv;ik})p_{jv;ik}c_{jv;ik}\,dm(v) + I_{ik}\right]dm(k) - \beta\int_{R_i} s_{ik}\,dm(k),$$

which we require to be equal to

$$\alpha\left[\int_{R_i}(M_i - s_{ik})dm(k)\right]\left\{\left[\gamma\int_{R_j} s_{jv}(t-t_{ji})dm(v)\right]\left[\int_{R_j}\int_{R_i} p_{jv;ik}\,dm(v)dm(k)\right]\cdot\right.$$

$$\left.\left[\int_{R_j}\int_{R_i} c_{jv;ik}\,dm(v)dm(k)\right] + \int_{R_i} I_{ik}\,dm(k)\right\} - \beta\int_{R_i} s_{ik}\,dm(k).$$

Thus

$$\int_{R_i}(M_i - s_{ik})I_{ik}\,dm(k) = \int_{R_i}(M_i - s_{ik})dm(k)\int_{R_i} I_{ik}\,dm(k)$$

and

$$\int_{R_i}\int_{R_j}(M_i - s_{ik})s_{jv}(t-t_{jv;ik})p_{jv;ik}c_{jv;ik}\,dm(v)dm(k) =$$

$$\int_{R_i}(M_i - s_{ik})dm(k)\int_{R_j} s_{jv}(t-t_{ji})dm(v)\iint_{R_i R_j} p_{jv;ik}\,dm(v)dm(k)\iint_{R_i R_j} c_{jv;ik}\,dm(v)dm(k).$$

Where $s_{ik} = \eta\int_{R_{ik}} V = \eta\int_{R_{ik}} \tilde{V}$, and V is the density for a V-process over each R_{ik}. $\tilde{V}$ is any density equivalent to V with respect to the $\{R_{ik}\}$ partition. Once again, dynamical quantities taken over disjoint parts of the structural carrier which subserve different processes are statistically independent, and in the case of the terms $s_{jv}(t-t_{jv;ik})$, the independence involves a

joint variation of s_{jv} and $t_{jv;ik}$ values. We erect these facts into a Postulate: Macroscopic embedding equations induce equations for microscopic densities such that the densities subserving different dynamical subprocesses are statistically independent in the above sense. These densities are, at best, equivalent to densities in $\mathcal{7}^*$ over excised subfields of $\mathcal{L}^*$.

This brief excursion into microscopic dynamics has provided a better idea of the interpretation of microscopic variables and has shown that the correspondence principle is realized by successively passing to ever finer averaging processes which always exhibit an excision property. Instead of remaining at the microscopic level, we shall now turn again to macroscopic phenomena, primarily geometrical matters in fact, but from a point of view that will guide us in enriching the dynamics of both macroscopic and microscopic settings.

58. Reaction Time, Thresholds, and Relay Nuclei

Microscopically, M_{ik} is a measure of the volume of a fixed cell or cluster of dynamically indistinguishable cells. Macroscopically, M_i is an estimate of the average of the M_{jk} values of the cells which make up the control subform of a core trajectory. These latter cells need not be the same through time, even if the macroscopic embedding equations remain unchanged, just so long as field numbers such as M_j, p_{jk}, etc., and the times taken to transmit between points do not change. A system for which this is true is called stationary. The fields $\mathcal{7}$ are conceivably in continual motion within $\mathcal{7}^*$ without this fact being apparent in pre-averaged equations. Those $\mathcal{7}$ with which we have been concerned to now are thus not sensitive to the topology of $\mathcal{7}^*$. Rather, we merely have equations defined over an abstract surface (p_i). The passage from (p_i) to the surface generated by the points of $\mathcal{L}^*$ must introduce topological restrictions. It is behaviorally

important that the macroscopic equations for verbal data show a certain insensitivity to microscopic geometry, just as it was important that the macroscopic equations be insensitive to microscopic dynamical hierarchies. After all, a direct perception of neuroanatomy does not spontaneously arise in all speaking individuals, and we are spared many hours of withdrawn introspections by this particular perceptual deficiency. The introduction of the necessary topological restrictions shall now be made in gradual steps.

One topological oversight in the original equations is the following. If the natural dynamical object to study is approximately a core trajectory, then what does the splitting of the dynamics into an input function and a derived strength function mean? We know that the strength function roughly represents the activity of the control subform of the trajectory. The input function must therefore represent an average of the potentials generated from the peripheral receptors which pass through the various relay nuclei and to the primary receptive cortices, but not from beyond the structural carrier of the control subform. All core trajectories must share these first few steps in the relay of excitation from periphery to primary receptive cortices. The macroscopic determination of an input representing the presentation $\pi_i^{(e)}$ of the verbal form r_i cannot, therefore, in general be made independently of the distance

$$d_{ij} = \int_{D_i \cup D_j} |\bar{f}_i - \bar{f}_j| \, d_q$$

between the core trajectories (D_i, f_i) and (D_j, f_j). For as d_{ij} decreases, it is to be expected that the trajectories of p_i and p_j will increasingly share a common residual line structure, which will serve to guide the dynamical evolution of the total strength field in the service of both points. Since the line structure influences the evolution of a trajectory from the moment it is generated in the peripheral receptors, two general types of

questions must be answered in interpreting an atomic field:

1) How does the input vary as a function of the effective total mass R_i of a point?

2) How does the occurrence of $\pi_i^{(e)}$ affect those s_j for which d_{ij} is very small?

1) For definiteness, choose the input function

$$I_i^{(e)}(t) = \begin{cases} 0 & t < 0 \\ a_i t e^{-b_i t} & 0 \leq t \leq T \\ 0 & T < t \end{cases}$$

and let $M_i = M$, for all i. Suppose that all $\pi_i^{(e)}$ generate excitation gradients which have approximately equal total energies and frequency spectra on the peripheral receptors. Then it is reasonable to require that the time derivatives of all $I_i^{(e)}$, evaluated on their onset times -- which are assumed to be widely separated, are the same. For it is only after reaching higher neural stations that differences in residual line distribution strongly deform the excitatory trajectory through feedback and channeling effects. Consequently,

$$a = dI_i^{(e)}(0)/dt = a_i (1-b_i t) e^{-b_i t} \Big|_{t=0} = a_i ,$$

so that all $a_i = a$. It is intuitively clear that increasing R_i will not decrease the total input to p_i, whence

$$\bar{I}_i = \int_0^\infty I_i^{(e)} dt$$

is a monotone increasing function of R_i. Since $\bar{I}_i = a_i/b_i^2 = a/b_i^2$, b_i is a monotone decreasing function of R_i. The time at which $I_i^{(e)}$ reaches

its maximum is called the resonance time of $I_i^{(e)}$. $I_i^{(e)}$ resonates at $t=1/b_i$, which occurs sooner for smaller R_i. Similarly, the maximum value of $I_i^{(e)}$, a/eb_i, grows with R_i, as does the total mass $\int_o^{1/b_i} I_i^{(e)}dt = (1-2/e)/b_i^2$ delivered to p_i up to the resonance time.

A difficulty immediately arises in using this $I_i^{(e)}$, or any other input function depending on so few parameters. It is easy to imagine many situations in which the resonance time of $I_i^{(e)}$ should be a monotone decreasing function of R_i, while the other functionals of $I_i^{(e)}$ continue to vary with R_i in the same direction as they do above. For example, the time required to react to words often decreases as their familiarity increases, and familiarity certainly is not a monotone decreasing function of R_i. This difficulty is a direct consequence of the one-point representation p_i of a control form, which in turn requires a representation of $I_i^{(e)}$ as a function determined at a single source.

Letting Ω_t be the set of points in the $\mathcal{F}^*$-core of p_i which are first excited at time t, we see that the condition $a = dI_i^{(e)}(0)/dt$ imposes a restriction only on the initial strip Ω_o in $\mathcal{F}^*$. In $\mathcal{F} = (\{p_i\}, \mathcal{L})$ by contrast, this condition is extended to all of $\bigcup_{t\leq T} \Omega_t$, where T is the first time that the control form is excited by the input. All of the subtle temporal interactions within $\bigcup_{t\leq T} \Omega_t$ are thus collapsed into the uniformity of a homogeneously excited pool of cells, and $I_i^{(e)}$ measures the excitation of this collapsed pool.

The macroscopic fact that the reaction time for highly familiar words is small requires that there exist provisions whereby forms with large R_i can speed up the pace of their dynamical interactions. A large macroscopic R_i often goes hand in hand with highly concentrated (D_i, f_i) line structure, and by increasing the concentration of a line structure, we increase the highest strength maxima achieved by points in the field. Increasing the size of local excitation densities through increased strength transmission is thus correlated with increasing the rate with which transmission begins. In the equations we have been considering, both the onset

time of transmission and of excitation at any p_i are the same, and are the first time, B, that $s_i > 0$. B, however, is not changed by increasing s_i excitation. The onset time of local excitation cannot therefore generally equal the onset time of transmission to other points. This can be true only if a point begins transmitting after its strength value exceeds some value strictly greater than zero. The faster recognition time of familiar words is thus strongly dependent on the existence of a transmission threshold τ which exceeds the equilibrium potential of cells. Since we have always chosen the equilibrium potential to be zero, $\tau > 0$.

If we introduce nontrivial transmission thresholds into a macroscopic excitatory embedding field in the simplest way, the equation for s_i becomes

$$ds_i/dt = \alpha(M_i - s_i)(r\Sigma_j \max(s_j(t-t_{ji}) - \tau_j, 0)p_{ji}c_{ji} + I_i) - \beta s_i, \quad 0 \leq \tau_j < M_j,$$

which will reappear in our later work.

A homogeneously connected pool of cells cannot make effective use of a nontrivial transmission threshold. Thresholds only exhibit powerful effects when a temporal ordering exists in the line structure. The pathway from peripheral receptors to control form must therefore be broken up into disparate cellular collections, or nuclei, which are connected in chain-like sequences. Successive relay nuclei and nontrivial transmission thresholds are both required to even begin explaining why familiar words are recognized more rapidly than unfamiliar words.

Both the dynamical fact that nontrivial transmission thresholds exist and the geometrical fact that local chains of relay nuclei exist are familiar from neurological studies. What is significant here is that these facts, and part of their theoretical significance, can be derived by simple macroscopic psychological reasoning. Also of importance is the fact that here a dynamical or a geometrical property alone does not suffice to produce a desired effect. The dynamics and geometry must be properly coupled before the properties inherent in either one become behaviorally significant.

59. Inputs with Functional Coefficients

In some psychological studies, one wishes to preserve the association of a single point with a single atomic form for simplicity, and one is therefore reluctant to enlarge the carrier of $I_i^{(e)}$ to exhibit the interactions between relay nuclei, control forms, and the like. The simplification which is achieved by associating only one point to every atomic form is often accompanied by a complexification of the functions living over these points. For in psychological studies which involve delicate temporal interactions, the inadequacy of $I_i^{(e)}$ often requires the passage to input functions of the type

$$J_i^{(e)}(t) = \sum_{k=1}^{n} P_k(R_i, s_i\big|_{[0,t]}; t)e^{Q_k(R_i, s_i\big|_{[0,t]}; t)},$$

where P_k and Q_k are polynomials in t whose coefficients are functionals of R_i and $s_i\big|_{[0,t]}$, and $s_i\big|_{[0,t]}$ is the restriction of s_i to the time interval $[0,t]$. $Q_k \longrightarrow -\infty$ either if $t \longrightarrow \infty$ or if $s_i\big|_{[w,w+W]} \longrightarrow 0$ when $w \longrightarrow \infty$ and W grows sufficiently rapidly with w. Functional coefficients and other variables of weak type are necessary to exhibit the excitatory fluctuations in $\bigcup_{t\leq T} \Omega_t$ which are collapsed together in atomic fields. The functional coefficients "lift" the collapsed Ω_0 into $\bigcup_{t\leq T} \Omega_t$. Such coefficients can be replaced, with an increase in insight, by strong first-order differential equations if one is willing to increase the number of dynamical variables and to extend the underlying point field to include such objects as relay nuclei. Since such extensions are our central concern, we shall hold the determination of the P_k's and Q_k's in abeyance.

60. Vector Input Functions

2) In order to take into account the fact of non-independence of core trajectories, we must allow $\pi_i^{(e)}$ to induce some s_j excitation whenever

d_{ij} is small. The simplest way to do this is to assume that to every ordered pair (p_i, p_j) is assigned a positive number a_{ij} which measures the effective overlap of trajectories. Suppose that a_{ij} has been determined in some way and that occurrence of $\pi_i^{(e)}(t_o)$ generates the experimental input

$$J_i^{(e)}(t, t_o) = \sum_{k=1}^{n} P_k(R_i, s_i \big|_{[t_o, t]}; t, t_o) e^{Q_k(R_i, s_i \big|_{[t_o, t]}; t, t_o)},$$

where $J_i^{(e)}(t, t_o)$ is $J_i^{(e)}(t)$ shifted so that its onset occurs at $t=t_o$. In particular, $J_i^{(e)}(t) = J_i^{(e)}(t, 0)$. Under these conditions, we require that

$$_iJ_j^{(e)}(t, t_o) = \sum_{k=1}^{n} P_k(\hat{R}_j, s_j \big|_{[t_o, t]}; t, t_o) e^{Q_k(\hat{R}_j, s_j \big|_{[t_o, t]}; t, t_o)}$$

is received at p_j, where $\hat{R}_j = R_i a_{ij}, j \neq i$. $_iJ_j^{(e)}(t, t_o)$ is called the p_j input induced by $\pi_i^{(e)}(t_o)$. Letting $\hat{R}_i = R_i$, $J_i^{(e)}(t, t_o) = {}_iJ_i^{(e)}(t, t_o)$.

The experimental input to a macroscopic point field induced by $\pi_i^{(e)}(t_o)$ is thus a vector function

$$I_i^{(e)}(t, t_o) = ({}_iJ_1^{(e)}(t, t_o), {}_iJ_2^{(e)}(t, t_o), \ldots, {}_iJ_n^{(e)}(t, t_o))$$

of the dimension n of the point field. The rationale behind the choice of this function is simple. It is imagined that, when viewed from the points excited by $\pi_i^{(e)}(t_o)$, each $p_j, j \neq i$, appears to have an effective total mass of $\hat{R}_j = R_i a_{ij}$. The evolution of s_j under this geometrical relativization then determines the functional coefficients P_k, Q_k in the vector input function in the usual way.

How can the a_{ij}'s be chosen? The geometry of the situation is so restricted that a choice like

$$a_{ij} = \mathcal{A}(R_i/R_j, d_{ij})$$

is almost inevitable, where $\mathcal{A}$ is a monotone decreasing function of d_{ij} and of R_i/R_j. An important property of such an a_{ij} is that $R_i = R_j$ implies

$$a_{ij} = \mathcal{A}(R_i/R_j, d_{ik}) = \mathcal{A}(1, d_{ji}) = \mathcal{A}(R_j/R_i, d_{ji}) = a_{ji}.$$

These choices of $I_i^{(e)}(t, t_o)$ and of a_{ij} are examples of a more general situation in atomic fields. We consider for convenience an idealized situation, never realized in an actual case, and compare this situation with more realistic ones. Thus, suppose that $\pi_j^{(e)}$ has never occurred in the past and that $\pi_i^{(e)}$ generates an idealized core trajectory $\mathcal{G}_i$. We shall compare this $\mathcal{G}_i$ with the trajectory induced by $\pi_i^{(e)}$ in a field for which both p_i and p_j have non-trivial R values. In such a field, $\pi_i^{(e)}$ will no longer generate $\mathcal{G}_i$ when d_{ij} is small, for the line residues of p_j will deform $\mathcal{G}_i$ to conform with the pattern of the p_j lines. a_{ij} is a density measuring the degree to which unit regions of p_j can deform R_i, and $\hat{R}_j$ is the effective total mass of these regions. The definition of a_{ij} and R_j thus involves the virtual variation of a trajectory.

How does it happen that a single density a_{ij} characterizes such a variation, which is conceivably very complicated? In general, it doesn't. The present situation is very special, however. Here, the set $\{ p_k \}$ consists of atomic forms of homogeneous type, all subserving a common dynamical role. Practically all local variables are averaged out in the atomic field because they are macroscopically indistinguishable. The single parameter R_i is left to distinguish all geometrical differences in the atomic forms. Relative deformation capacities of these forms must therefore be describable entirely in terms of the R_i, without examining the exact distribution of the various excitation distributions of the $\mathcal{T}^*$-cores. Since relative deformation capacities are determined entirely by the R_k's,

equal R_k's must imply equal deformations, whence $a_{ij}=a_{ji}$. As a function of (R_i, R_j), a_{ij} is a function of the ratio R_i/R_j alone because it measures a density that must be spread over a region whose size depends on the relative magnitudes of the p_i and p_j cores, and our only measure of such magnitudes is R_i/R_j. a_{ij} cannot be entirely independent of the relative positions of (D_i, f_i) and (D_j, f_j) in $\mathcal{7}^*$, so we are forced to study its dependence on d_{ij} as well. The pair of variables d_{ij} and R_i/R_j together represent a splitting of two trajectories into their relative distributions and their relative total masses. Although such a splitting is conceptually simple and involves few variables, one has less direct control over such variables as d_{ij} and a_{ij} than one has over more locally defined variables, of which one usually needs many. This is the same difficulty which we face when we use a general $I_i^{(e)}(t, t_o)$ vector function with functional coefficients. Lumping the total space $\mathcal{7}^*$ leads to fewer functions, but each of these functions is harder to deal with numerically.

One of the most characteristic features of the representation of atomic forms by single points is that it leads to the splitting of field variables of almost every kind. First, core trajectories are split into field points and external input sources. To understand how inputs work in a field that is split in this way, we introduced the concept of an ideal trajectory, evolving unperturbed by the existence of the line residues of other cores. A virtual deformation of this trajectory, induced by a replacement of the other line residues, gives rise to a vector input function whose components are determined by the magnitude of this deformation. Although corresponding to $\pi_i^{(e)}$ only one flow exists in $\mathcal{7}^*$, we split the total input into n components and imagine that each of the p_i receives its own component input. $\hat{R}_j = R_i a_{ij}$ provides a geometrical estimate of this splitting in the total input function. In an atomic field, a symmetry argument shows that one must always seek a geometrical condition like $\hat{R}_j = R_i a_{ij}$ to determine how the various input components differ from one another. A dynamical condition would require that we augment

the field with further points. Since the R_i themselves vary slowly with time, the coefficients of the components of the vector input function are themselves split into two kinds of variables: $s_j\big|_{[t_o, t]}$ is rapidly varying while R_j is slowly varying. This splitting is analogous to the splitting which contrasts s_i and c_{jk} with M_i and p_{jk}. Splitting 7* into one point representations of atomic forms thus requires a corresponding splitting in individual core trajectories, in the total input function, and in the component functions of this input function. When one geometrical or dynamical portion of the field is grained at a given level, all features of the field must follow suit.

61. Perceptual Readiness, Stimulus Generalization, and Stimulus Differentiation

The existence of vector input functions with more than one nonzero component has far-reaching consequences. Let the transformation W be defined by

$$W(r_i, t_o) = \left\{ p_j : {}_j\pi_i^{(e)}(\cdot, t_o) \not\equiv 0 \right\}.$$

W is not a well-defined point-to-point transformation. Rather it carries points into sets of variable size and thus violates the intuitive, though noncritical view that the presentation of a single $\pi_i^{(e)}$ should dynamically pick out its intended p_i, and p_i alone. What gives rise to the intuitive impression that a given verbal input is an input to precisely one verbal form? This impression is strongly tied to the fact that no more than one response unit can be emitted at any time in speech, and that most verbal presentations are so distinctive that line renormalizations do tend to quickly channel the input vector into the core which generates the correct response. By gradually permitting the input to become less distinctive, however, it becomes intuitively much more obvious that the effects of the input should

be broadly spread over the collection of points which lie close to the primary input point in 7^*. Good examples of such a spread occur when one must guess which sounds have been uttered at threshold intensities, or when one must complete ambiguous figures. The notion that "choices" are temporally localized acts and the notion of nontrivial vector input functions are thus strongly incompatible.

62. Anchoring

The set $W(r_i, t_o)$ is called the spray of $\pi_i^{(e)}(t_o)$, denoted by $sp_i^{(e)}(t_o)$, and we sometimes say that $\pi_i^{(e)}(t_o)$ sprays some point in W. A large number of phenomena can be subsumed under a general study of sprays. An interesting phenomenon involving sprays is a development of the fact that the existence of transmission thresholds greater than the equilibrium potential helps to explain the more rapid recognition of familiar items. Let us be given the three point structural carrier $\{p_i, p_j, p_k\}$ with $sp_i^{(e)}(t_o) = \{p_i, p_k\}$ and $sp_j^{(e)}(t_o) = \{p_j, p_k\}$. We wish to perform two independent experiments on this field, so we suppose that duplicate fields are given and that $\pi_i^{(e)}(t_o)$ occurs in one of them and $\pi_j^{(e)}(t_o)$ occurs in the other. Whenever we say that two events $\pi_i^{(e)}(t_o)$ and $\pi_j^{(e)}(t_o)$ occur simultaneously, or discuss various aspects of these two events, we will mean that they occur in different copies of the same field. Suppose that

$$k^{J_i^{(e)}}(t, t_o) > k^{J_j^{(e)}}(t, t_o),$$

$$i^{J_i^{(e)}}(t, t_o) = j^{J_j^{(e)}}(t, t_o),$$

and, trivially,

$$j^{J_i^{(e)}}(t, t_o) = i^{J_j^{(e)}}(t, t_o).$$

Consider the two sequences $\pi_{ik} = (\pi_i^{(e)}(t_o), \pi_k^{(e)}(t_o + \lambda))$ and $\pi_{jk} = (\pi_j^{(e)}(t_o), \pi_k^{(e)}(t_o + \lambda))$, and let u^{s_k} be the strength function of p_k in the

field corresponding to π_{uk}, $u = i, j$. λ is chosen so that $\max_{u=i,j} {}_{u}s_{k}(t_o+\lambda) \not\approx 0$. Also endow the field with a set of nontrivial transmission thresholds.

Under these conditions, $\pi_{i}^{(e)}(t_o)$ will augment ${}_{i}s_{k}$ growth more than $\pi_{j}^{(e)}(t_o)$ will augment ${}_{j}s_{k}$ growth. Since ${}_{i}s_{k}(t_o+\lambda)$ will therefore be greater than ${}_{j}s_{k}(t_o+\lambda)$ when $\pi_{k}^{(e)}(t_o+\lambda)$ occurs in both fields, ${}_{i}s_{k}$ will generally exceed ${}_{j}s_{k}$. Both the transmission threshold and the response criterion will therefore be reached sooner for ${}_{i}s_{k}$ than for ${}_{j}s_{k}$ after $\pi_{k}^{(e)}$ occurs. Translated into macroscopic terms, this means that the initial presentation of an item r_i that is more closely related to r_k than is r_j can decrease the time needed for r_k to reach consciousness and to be uttered in reply when $\pi_{k}^{(e)}$ later occurs. The fact that conditioning the field with different r_i and r_j can influence r_k emission in different ways as a result of differences in the intertwining of core trajectories shows that a covert dynamical "tug-of-war" takes place between items whenever even a single input is delivered. By conditioning the field with r_i in this example, we prepare the organism for the evokation of r_k even before $\pi_{k}^{(e)}$ occurs. This preparation appears to the psychological experimenter as a kind of perceptual readiness which can be manipulated by introducing special input sequences. This readying of the organism by conditioning the field is a direct analog of the formation of psychological sets. Both concepts involve the introduction of localized biases in the global instantaneous strength field which linger after the experimental manipulation has been terminated. If choice behavior really did occur at quantized instants in time, such conditioning processes would be impossible to understand.

Whenever $W(r_i, t_o)$ is a set with more than one element in it, it is proper to say that the stimulus "generalizes." "Stimulus generalization" is a process which is described by studying both broad line interactions between collections of points and the distribution of large spray sets in time. Dually, "stimulus differentiation" involves the contraction of the various concentration sets and of the spray sets associated

with a given input to a single point. In 7*, this joint study of concentration sets and of spray sets, which is required by the splitting of the field into separate points, is replaced by a global study of dynamical trajectories. We say that stimulus generalization increases in 7* when such a trajectory simultaneously excites points that transmit to increasingly large sets of response control subforms.

It is important to observe that the spray sets and the line concentration sets will not generally contract in precisely the same way. For the line concentration sets refer primarily to the embedded memory structure, while the spray sets include overlap effects due to mere topological propinquity of different input forms on the peripheral receptive layers and the response control cell groups.

63. Emotive Fields and Cognitive Balance

Another effect characteristic of sprays is the following. Let a point set $\{ p_1, \ldots, p_n \}$ be given with $p_{ij} = \delta_{j,i+1} + \delta_{j,i-1}$, $i=2, 3, \ldots, n-1$, $p_{1j} = \delta_{2j}$, $p_{nj} = \delta_{n-1,j}$. Such a point set is called a chain of loops. Let

$$sp_i^{(e)}(t_o) = \{ p_{i-1}, p_i, p_{i+1} \}, \qquad 2 \leq i \leq n-1,$$

$$sp_1^{(e)}(t_o) = \{ p_1, p_2, p_3 \},$$

and

$$sp_n^{(e)}(t_o) = \{ p_n, p_{n-1}, p_{n-2} \}.$$

Require that

$$_{i-1}J_i^{(e)}(t, t_o) = {}_{i+1}J_i^{(e)}(t, t_o) < {}_{i}J_i^{(e)}(t, t_o) = {}_{j}J_j^{(e)}(t, t_o),$$

$t_o < t$, $2 \leq i, j \leq n-1$, and that

$$ {}_1J^{(e)}_1(t, t_o) = {}_nJ^{(e)}_n(t, t_o) > {}_2J^{(e)}_1(t, t_o) = {}_{n-1}J^{(e)}_n(t, t_o) > $$

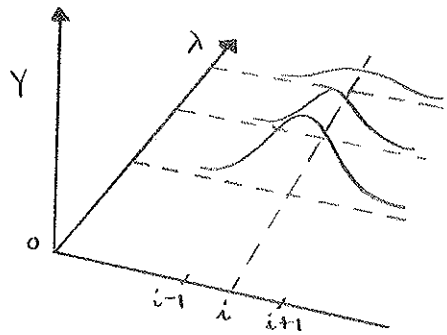
$$ {}_3J^{(e)}_1(t, t_o) = {}_{n-2}J^{(e)}_n(t, t_o). $$

Let ${}_iJ^{(e)}_j(\cdot, t_o) \equiv 0$ in all other cases. Also endow all line functions with a comparable symmetry relative to the endpoints $\{p_1, p_n\}$, and let $M_i = M$.

Let π_{ik} occur with i fixed and k varied in copies of the field. As $|i-k|$ decreases, ${}_is_k(t)$ increases. Whenever $|i-k| >> 2$, ${}_is_k(t) \approx s_k(t)$, where s_k is the strength function induced under $\pi^{(e)}_k(t_o+\lambda)$ alone. In fact, define

$$ Y(\lambda; k|i) = \left[\left(\int_{t_o+\lambda}^{\infty} {}_is_k(t)dt; \pi^{(e)}_i(t_o), \pi^{(e)}_k(t_o+\lambda) \right) / \left(\int_{t_o+\lambda}^{\infty} s_k(t)dt; \pi^{(e)}_k(t_o+\lambda) \right) \right] - 1. $$

Y measures the relative mean facilitation of p_k by a conditioning input to p_i λ time units earlier. Y looks like



A facilitative effect on the ${}_is_k$'s is thus restricted to local chains of loops of the form $\Lambda_i^{mr} = \{p_{i-m}, p_{i-m+1}, \ldots, p_i, \ldots, p_{i+r}\}$ where m and r depend on λ and on the total breadth of the spray sets and of the p_{ij} distribution. This situation is sometimes described by saying that p_i is an anchor for Λ_i^{mr}. Anchoring depends critically on topological factors, which are here expressed by locally chaining the p_{ij} distribution and the spray sets.

Anchoring has been studied in various discrimination experiments, notably with the discrimination of colors by pigeons. The points of this example thus exhibit behavior which resembles the behavior underlying the anchoring response distributions of a pigeon trying to discriminate colors. If we wish to extend this analogy between points and colors varying along a continuum of frequencies, should not $\{p_1, \ldots, p_n\}$ be replaced by $\{p_\lambda : \lambda \in [\lambda_1, \lambda_2]\}$, where $[\lambda_1, \lambda_2]$ is a band of frequencies within the visible spectrum of the pigeon? This is impossible, since only a finite number of points exist in $\mathcal{F}^*$. How can an infinite collection of frequencies be registered within a finite collection of receptive points? One is forced to admit that each point carries an infinite number of degrees of freedom which can be used to register differences in the frequency of light impinging on peripheral receptors. The strength functions are necessarily the carriers of this infinitude of degrees of freedom. The existence of a system of frequency modulation in each point thus follows directly from a dimensional argument. If all first-order visual afferents were identical and were attached to the retina in a homogeneous way, every steady state potential s of these cells would be associated with a connected band of frequencies, possibly containing just one point, which is transmitted to more centrally located cells by spikes obeying $R_{ik} \longrightarrow N_{ik;jv}$ invariance. It is easy to see that such a homogeneous distribution of points is not very efficient. Since every point carries equipment to distinguish infinitely many frequencies, the capacity to distinguish these frequencies with maximal efficiency must be introduced by properly distributing points relative to the visual receptor cells and to one another. In particular, the cells must be distributed in subsets that respond best to receptor cells which are maximally sensitive to localized frequency bands. How this localization is accomplished is again a topological question, but one of far greater complexity than that required to achieve merely qualitative anchoring effects by localizing field dynamics to short chains of loops. We will return to this question after making some further

geometrical preparations.

63. Emotive Fields and Cognitive Balance

Another field which describes non-verbal behavior is a field related to emotional expression. We suppose that there exist structures,(*) represented by points $\mathcal{H} = \{q_h : h \in H\}$, whose activation corresponds to a variety of emotional sensations within an individual. We idealize the situation for initial simplicity by supposing that each of the structures represented by one of these points is concerned with a particular emotional disposition and that pairs of such structures behave independently. We suppose further that the activation of these points may be induced by strength transmitted from the points of the verbal embedding field $\mathcal{T}$. Thus, to every p_i and q_h, assign numbers p_{ih} and p_{hi} in the familiar manner. It is not at all obvious at this stage whether the q_h carry an embedding dynamics that is identical with that of a p_i, but assume for simplicity that we have chosen q's for which this is completely true. That is, let M_h's be given and define functions e_h corresponding to the s_i and functions g_{ih}, g_{hi} corresponding to the c_{ij}. Further let $\tilde{g} = g$, and for simplicity suppose that an n point dual completion $\hat{\mathcal{T}}$ of $\mathcal{T}$ is given, which is entirely unconnected with the q_h (diagram 10). $A_{hh'} = A\,\delta_{hh'}$ by the assumption of independence of the q_h's. Assume that the times required for $\mathcal{T} \to \mathcal{T}$ transmission and for $\mathcal{T} \to \mathcal{H} \to \mathcal{T}$ transmission are determined in complete analogy with the transmission assumptions needed in n-point dual completions of $\mathcal{T}$. Suppose, somewhat naively, that our enumeration of the q_h is so complete that the portion of the subject's emotional repertoire which is related to $\mathcal{T}$ may be derived entirely from functions of the vector (e_h).

Consider the assignment of initial values to the g's. Large values of $g_{ih}(0)$ and $g_{hi}(0)$ correspond to frequent pairings of the verbal unit p_i with the emotive unit q_h. No amount of merely verbal activity can induce strong connections with $\mathcal{H}$. Surely the activation of many emotive dispositions

(*) hypothalamic!

arose originally from nonverbal sources, but it may be assumed that our study of the process has begun only after the subject's development has proceeded to the realization of a high order of symbolic influence on the emotive structure. Now simply apply the usual analysis of embedding effects. For example, let $\pi_i^{(e)}(t)$, $\pi_j^{(e)}(t+h)$ occur, for small h, where $p_{ih}g_{ih}$ and $p_{ij}c_{ij}^*$ are initially large and $p_{jh} > 0$. e_h will grow quickly due to strength transmitted from p_j, whence by the usual argument g_{jh} will grow as well. Thus, frequent pairings of words, one of which has a certain strong emotive import, will tend to increase the extent to which the second one has the same import. Similarly, an input to a word with a high emotive import e_h will, via e_h transmission to the field $\mathcal{F}$, increase the point strength values of points p_i for which $p_{hi}g_{hi}$ is large. To the extent that both g_{hi} and g_{ih} are large whenever one of them is, we see from this that even words which have a trivial history of temporal contiguity with each other but close relation with a given emotive disposition will tend to facilitate one another. This is one method whereby psychological sets are formed and their gradual transitions controlled during a running emission of language forms.

The often gradual character of these emotive transitions in language behavior will be especially well-achieved under two circumstances: first, if the e_h decay rate is slower than the s_i decay rate; and second, if emotionally significant words are not distributed too densely in a sequence of words. The first alternative may be replaced by the still more effective method of allowing strong self-excitations in the q_h. Self-excitatory interactions can sustain themselves for a long time even if decay rates in $\mathcal{H}$ are no smaller than those in $\mathcal{F}$, and can nonetheless be rapidly suppressed by lateral inhibitory action. The second alternative is a way of distributing practice in $\mathcal{H}$ relative to the relaxation times of $\mathcal{H}$-dynamics. From this point of view, $\mathcal{H}$ may be viewed as a type of measuring device hooked up in a non-trivial way to a <u>proper</u> subfield of $\mathcal{F}$. The projection of $\mathcal{F}$ onto $\mathcal{H}$

is reminiscent of a Hilbert space projection onto a proper subspace. The proper functioning of $\mathcal{H}$ in this measuring capacity requires that dynamical transitions within $\mathcal{H}$ proceed significantly more slowly than those in $\mathcal{7}$. The reciprocal line connections from $\mathcal{H}$ to $\mathcal{7}$ are constructed to insure this relative smoothing of transitions within $\mathcal{H}$ by modulating the strength field of $\mathcal{7}$ to conform with that of $\mathcal{H}$. If the rate of transition from point to point within $\mathcal{7}$ is ν but only $1/m$ of the points within $\mathcal{7}$ project to $\mathcal{H}$, then the effective rate with which inputs pass from $\mathcal{7}$ to $\mathcal{H}$ is just ν/m. The time between $\mathcal{7} \rightarrow \mathcal{H}$ inputs is used by $\mathcal{H}$ to excite points within $\mathcal{7}$ which are strongly line-connected to the $\mathcal{H}$ point receiving the last $\mathcal{7} \rightarrow \mathcal{H}$ input, at least in the simplest cases. Other things equal, the inputs to $\mathcal{7}$ which are most successful in generating responses will thus be to such $\mathcal{H}$-facilitated points. By constructing one's geometrical carrier carefully, highly sensitive context effects can be achieved using this type of interaction.

How do the point concentration sets of $\mathcal{7}$ and $\mathcal{H}$ look at any moment? Various situations revealing strong contractions of the $\mathcal{7}$-concentration sets have already been studied. One such situation occurs when very large inputs are delivered to a small collection of $\mathcal{7}$ points. Suppose that very large inputs are delivered to a few $\mathcal{7}$ points N which hold a strong emotional significance to the subject. Then $p_{hi}g_{hi}$ and $p_{ih}g_{ih}$ couples, with $p_i \in N$, are large for a small set of points $P(N)$ in $\mathcal{H}$. Let $\hat{N}$ be the set of points p_j whose $p_{jh}g_{jh}$ and $p_{hj}g_{hj}$ values are large for some $p_h \in P(N)$. $N \subset \hat{N}$. Suppose that the points in N represent important units or events in the subject's repertoire, so that their R_i values are large, and let $\mathcal{7}$ be equipped with a field extension that serves to sustain its embedded forms.

Presenting large inputs of this kind has far reaching effects on field behavior. Firstly, s_i and e_h, for $p_i \in N$ and $q_h \in P(N)$, will facilitate and sustain one another through reciprocal excitation. The strong intervention of $\mathcal{H}$ will also globally renormalize the spontaneous dynamics of $\mathcal{7}$ to $\hat{N}$. Focalized activation of N can also renormalize the dynamical effects of external inputs to other $\mathcal{7}$ points out of existence through the intervention

of blocking effects in $\hat{7}$. The net result is a dynamical system which closets itself in $\hat{N}$ and cannot be perturbed from its cyclic self-excitations by ordinary external inputs. Such effects are familiar in cases of severe psychological trauma and in many psychotic withdrawal syndromes. Here we have merely sketched in broadest strokes one way in which these unfortunate events may be studied dynamically. By extending our investigations to more elaborate and neurologically more accurate fields, it will prove possible to apply variational tools to the problem of how best to decompose the $\hat{N}$ contraction and to enable the subject to return to a more responsive life.

64. Net Affect and Complete Sets of Antagonists

In this direction, let us now remove the independence condition on the points q_h in a simple way. Imagine that a given pair $(q_h, q_{h'})$ of emotions is antagonistic in the sense that it is not the functions e_h and $e_{h'}$ which express the perceived emotional effect, but rather some monotone increasing function $\mathcal{E}$ of $|e_h - e_{h'}|$, say $|e_h - e_{h'}|$ for simplicity. We shall not explore the process that realizes $|e_h - e_{h'}|$ now, but shall take its existence as a given. In an admittedly vague terminology, one might find it intuitively instructive to let (q_h) consist of only two points, where q_1 = "liking" and q_d = "disliking." Then if a "+"-sign is attached to q_1 and a"-" sign to q_d, the function $N_{1d} = (\operatorname{sgn}(e_1 - e_d))\ \mathcal{E}\ (|e_1 - e_d|)$, where

$$\operatorname{sgn}\omega = \begin{cases} +1 & \text{if } \omega > 0 \\ -1 & \text{if } \omega < 0, \end{cases}$$

is more natural to consider than $\mathcal{E}\ (|e_1 - e_d|)$; it roughly measures the net affect of a given verbal situation at any time. Again let $\mathcal{E}(w) = w$ for simplicity, and suppose that $(\pi_i^{(e)}(t),\ \pi_j^{(e)}(t+z))$ occurs at a time t when

$p_{il}g_{il}$ and $p_{jd}g_{jd}$ are much larger than $p_{id}g_{id}$ and $p_{jl}g_{jl}$. In this situation, the average net affect associated with rapid inputs (z small) to the pair p_i, p_j will be less than that associated with each item under a slow presentation rate (z large). The antagonistic affects thus tend to cancel one another when they are contiguously presented. An alternating sequence of the type $(\pi_i^{(e)}(t), \pi_j^{(e)}(t+z), \pi_i^{(e)}(t+2z), \ldots)$, with z small, will have a comparable cancelling effect on the affects associated with p_i and p_j, but will by virtue of the greater total excitatory mass of the long sequence also tend to homogenize any affects associated with other points in $\mathcal{F}$.

Averaging effects between non-independent affects are related to the various attempts at developing balance theories to describe attitude structures. We therefore call N_{ld} a balancing function. If some p_i does not project significantly to q_l or q_d, it shall not directly participate in the balancing process. In some balance theories, say that of Heider and the graph theoretical developments inspired thereby, such nonparticipation is often expressed by omitting a line in the graph representing the cognitive structure. Here it is recognized that the fiber tracts or lines, might well exist, but their $p_{ih}g_{ih}$ products are relatively small. In some of the attempts to develop cognitive models, the cognitive units are taken as nodes in a graph and the lines represent the degree of attitudinal connection between these units. Here it is recognized that several types of points and lines are required. The cognitive units themselves form one field $\mathcal{F}$ which is reciprocally linked to another field $\mathcal{H}$ that subserves the task of preparing attitudinal averages. The lines are not attitudinal links which themselves carry some type of affect measure. Rather, they merely transmit certain dynamical quantities whose attitudinal significance evolves within the points of $\mathcal{H}$. The old theories thus collapsed properties of emotive points and transmission lines into lines suggesting an attitudinal connection between cognitive points. In these theories, it was not natural to add emotive points, since the cognitive points were merely formal labels for externally perceived

units which did not subserve any dynamical process of their own. Many of the difficulties in constructing attitudinal theories thus arose from a very incomplete understanding of the geometry of the attitudinal situation. The deficient geometry was, in turn, the natural counterpart of the practically non-existent perception of the underlying dynamics. In an embedding field context, many of these difficulties can be overcome by applying the same methods that hold for ostensibly non-attitudinal processes.

It is important to ask how a function like N_{ld} comes to modify the behavior of the entire organism. For example, N_{ld} or some counterpart thereof will perhaps express the degree of eventual transformation of strength values from $\mathcal{H}$ to the hormonal values related to emotional expression, which are then freed into the bloodstream. An alternative here arises: Either each of e_l and e_d generates some actuator $\Phi(e_l)$ and $\Psi(e_d)$, respectively, of its particular emotional dimension, after which these various actuators cancel one another in a nonneural frame, or e_l and e_d first act upon one another to produce a net distribution of excitation, say $\tilde{e}_l$ and $\tilde{e}_d$, after which some one of $\Phi(\tilde{e}_l)$ or $\Psi(\tilde{e}_d)$ comes into play most prominently in the organism as a whole. To the extent that the global appearance of both $\Phi(e_l)$ and $\Psi(e_d)$ will generate internally incompatible behavior in the organism, it is profitable that the release mechanism be constructed roughly upon the example of the second alternative.

The possibility therefore strongly emerges that there exist lines L_{ld} and L_{dl} over which the activity at each $\mathcal{H}$ point is rapidly transmitted to suppress the activity of its antagonist, by a form of lateral inhibition, and that equal activity in each antagonist should generate roughly an activity of zero as a rapidly achieved asymptote. Equally excited antagonistic pairs will hold one another in a state of active suppression so that the overt behavior of the organism does not become a mass of confusion. A similar condition of almost zero activation can be achieved for any number of equally excited antagonistic points in a homogeneous $\mathcal{H}$ field by suitably choosing the L_{ij} line functions. When the L_{ij} functions are so chosen, we say that the

point field of $\mathcal{K}$ comprises a complete set of antagonists. In the e_k functions of a complete set of antagonists is thus to be found a law of distribution for which initially equal values become zero asymptotically, even if the $\mathcal{I}$ inputs generating these equal values are not discontinued until long after their onset period. To the present, we know only one functional for which equal values give a zero value: the information functional. The expectation therefore arises that, in some fashion, embedding fields which approximate a complete set of antagonists give an asymptotic dynamical realization of some analog of the information functional.

65. Complete Sets of Antagonists and the Information Functional

This expectation gains further support from the fact that, in a complete set of antagonists, among all possible orderings of input arrays which use some fixed input form, an input to a single point will generate maximal e_k values. Similarly, let P be a proper subset of $\hat{P}$, and suppose that a fixed input array is delivered once to P with P considered as a proper subset of $\hat{P}$ and once to P alone. The e_k functions of P in both situations are identical, since the e_k functions of $\hat{P} \setminus P$ receive only inhibitory transmissions from P and thus never begin to transmit reciprocally to P itself.

These three properties of the dynamics of complete sets of antagonists are clear analogs of the following three properties of the informational functional

$$H = - \sum_{k=1}^{n} p_k \lg_2 p_k,$$

where $\sum_{k=1}^{n} p_k = 1$ and each $p_i \geq 0$:

1) $H(p_1, \ldots, p_n)$ achieves it largest value for $p_k = \frac{1}{n}$, all k.

2) $H(p_1, \ldots, p_n) = 0$ whenever some $p_j = 1$, and (3) $H(p_1, p_2, \ldots, p_n, 0, 0, \ldots, 0) = H(p_1, \ldots, p_n)$.

Indeed, considering the function

$$\overline{H}(p_1, \ldots, p_n) = 1 - \frac{H(p_1, \ldots, p_n)}{\lg_2 n},$$

These properties become:

1) $\overline{H}\left(\frac{1}{n}, \ldots, \frac{1}{n}\right) = 0.$

2) $\overline{H}(p_1, \ldots, p_n) = \max_{p_1^*, \ldots, p_n^*} H(p_1^*, \ldots, p_n^*) = 1$ whenever $p_i = 1$, $p_j = 0$, $j \neq i$, for some i.

3) $\overline{H}(p_1, \ldots, p_n, 0, \ldots, 0) = \overline{H}(p_1, \ldots, p_n)$.

The inputs to $\mathcal{H}$ play the role of the p_k, while the values of $\overline{H}$ are replaced by the asymptotic values of the e_k induced by lateral inhibition of antagonists.

To every isolated input I_k to g_k which we have thus far considered can be associated a t_k such that $I_k(t) \approx 0$ whenever $t \geq t_k$. If the asymptotic values of the e_k are taken to be $e_k(\infty) = \lim_{t \to \infty} e_k(t)$, all asymptotes will trivially be approximately zero and the comparison between $\overline{H}$ and the behavior of $\mathcal{H}$ becomes uninteresting. One way to naturally avoid this difficulty in interpreting the analog between strictly positive $\overline{H}$ values and e_k asymptotes is to consider classes of inputs for which the limits $\lim_{t \to \infty} I_k(t) = \omega_k$ exist and the ω_k are allowed to be strictly positive. Then the asymptotes $e_k(\infty)$ will exist and will sometimes form a nontrivial distribution. In this extension, the analog of p_k is $I_k(\infty)$ and of $\overline{H}$ is $\{e_k(\infty)\}$.

This consideration of asymptotic values at $t = \infty$ is a useful

conceptual device since it can be turned around to illustrate the following central fact: complete sets of antagonists have arisen in order to make possible the renormalization of the e_k values in finite time and thereby to eliminate the need to wait an infinite time interval while the I_k's and their induced e_k's reach stable asymptotes. Once repid lateral inhibitory action within $\mathcal{K}$ stabilizes the e_k, these asymptotic values will transfer their effects to another part of the structural carrier of the system. The termination of the stabilization process within $\mathcal{K}$, accomplished in finite time, thus represents the effectively infinite time, modulo $\mathcal{K}$, of the entire process. Lateral inhibition within complete sets of antagonists allows for an almost continuous flow of input excitation by computing stable net excitation averages quickly and passing on the results to the next processing station in the organism. This fact shows, in particular, that it is hopeless to seek a definitive neural correlate or a complete set of correlates of a global behavioral act by measuring on a microscopic neural scale. One must rather consider the entire flow of excitation over large collections of interconnected cells when dealing with behavioral events.

66. Apparent Time Quantization. Temporal Numerosity

An interesting observation which applies antagonistic inhibitory effects and the importance of considering the entire trajectory requires that we suppose that we are given a finite collection $\{\Omega_i, i=1,2,\ldots,n\}$, of fields such that whenever lines from Ω_i lead out of Ω_i, they lead to Ω_{i+1}. That is, we have a chain

$$\Omega_1 \longrightarrow \Omega_2 \longrightarrow \ldots \longrightarrow \Omega_n$$

of fields. Suppose that external inputs to this system arrive only at Ω_1 and that the emission of a given behavioral act requires the realization of a certain excitatory flow through the entire chain of fields. While this act

is being elicited, it is important that new inputs which would generate incompatible behavior not be allowed to arrive at Ω_1 in effective form. That is, extensive excitation within $\Omega_2 \rightarrow \Omega_3 \rightarrow \ldots \rightarrow \Omega_n$ must inhibit excitation within Ω_1. Some general ways whereby this can be accomplished have already been studied in fields $\hat{7}$ and $\mathcal{H}$. Assume that one of these ways applies in the present case. What is visible of this process from the point of view of an observer $\mathcal{O}$ delivering inputs to Ω_1?

For definiteness, suppose that the time needed to transmit from Ω_k to Ω_{k+1} is τ_k and that an input representing a strongly embedded trajectory of $\Omega_2 \rightarrow \ldots \rightarrow \Omega_n$ has been delivered to Ω_1. For a total time of approximately $\tau = \sum_{k=1}^{n} \tau_k$ after delivery, no incompatible form will be able to effectively excite Ω_1. In other words, $\mathcal{O}$ will think that time within $\Omega_1 \rightarrow \Omega_2 \rightarrow \ldots \rightarrow \Omega_n$ is <u>quantized</u> in intervals of length τ. But to an observer following the excitation wave from Ω_1, through the lines to Ω_2, and so on, time will still seem to flow <u>continuously</u>. Data has appeared in which just such an apparent quantization of time relative to events in Ω_1 seems to occur; for example, in subjects judging the temporal numerosity of successive light flashes delivered at high presentation rates. Here one finds that the assessment of temporal numerosity is almost linearly related to the rate of presentation of successive flashes, with a slope less than one, even when the flicker fusion rate is not exceeded. Such an effect is immediately evident in chains like $\Omega_1 \rightarrow \Omega_2 \rightarrow \ldots \rightarrow \Omega_n$, where the dynamical events in $\Omega_2 \rightarrow \ldots \rightarrow \Omega_n$ include the counting process itself. Delicate visual factors can also contribute to this effect, but it can be explained even in their absence. The present example thus shows that apparent quantizations can be easily determined entirely by the perspective of the observer as a measurer of macroscopic events and not as a reflection of a quantization in local excitatory dynamics. By special choices of the Ω_i and relaxations of the chaining requirement, such effects will appear in many guises below.

67. Conditioning in Complete Sets of Antagonists

The comparison of the mapping $\{I_K(\infty)\} \longrightarrow \{e_k(\infty)\}$ with $\{p_k\} \longrightarrow \{\bar{H}(p_1, \ldots, p_n)\}$ also suggests the following observation. We should find the same $\{e_k(\infty)\}$ whenever we are given two sets $\{I_k\}$ and $\{I'_k\}$ of $\mathcal{H}$-inputs such that (1) $\omega_k = \lim_{t \to \infty} I_k(t) = \lim_{t \to \infty} I'_k(t)$, and (2) the input functions achieve their asymptotes sufficiently slowly. For the mapping is one of asymptotic values only and should therefore be independent of all fluctuations in the inputs which take place over finite time intervals. In particular, the e_k asymptotes should be independent of the initial values of the I_k. This property is obviously not satisfied when complete sets of antagonists must produce stable averages in finite time. A strong conditioning of the stabilized e_k values on the initial distribution of excitation and on the total input distribution takes place. This conditioning of values is the analog of the following property of the information functional:

$$4) \qquad H(AB) = H(A) + H_A(B),$$

where A and B are two events, AB designates their joint occurrence, and $H_A(B)$ gives the conditional information of B given the event A.

In $\mathcal{H}$, A and B represent the occurrence of fixed collections of inputs to the same set Γ of points in $\mathcal{H}$. The event AB means that both sets of inputs are delivered to Γ simultaneously, while saying that B is conditioned on A means that the A inputs are delivered earlier than the B inputs. The various probabilistic notions pertaining to the combination of individual events must be considered in a temporal dimension as well as in a spatial dimension in $\mathcal{H}$. One cannot, as one often does in information theory, assume that all operations are merely conceptual and therefore free of binding by a specific time component. The notion that conditioning in $\mathcal{H}$ involves a temporal component is a natural consequence of the idea that antagonistic sets act to produce asymptotic averages for the

rest of the field.

In information theory, it is proved that $\overline{H}_A(B) \geq \overline{H}(B)$. This is interpreted in $\mathcal{H}$ simply as: the net effect of earlier A inputs to $\mathcal{H}$ is to raise the e_k values, perhaps only infinitesmally, from quiescence, and thus to prepare them to reach higher asymptotes under B than could be achieved without a preliminary A. The condition $H(AB) = H(A)+H(B)$ can therefore only occur when A sends inputs to Γ_A, B sends inputs to Γ_B, $\Gamma_A \bigcap \Gamma_B = \phi$, and no lines pass between Γ_A and Γ_B.

Since conditions (1), (3), and (4) together characterize H within the class of continuous functions, as Khinchin shows in his book on Information Theory, the analogy between complete sets of antagonists and $\overline{H}$ is a significant one.

One fact has been conveniently suppressed during this discussion. Even under equal inputs to a homogeneous complete set of antagonists, Φ - transmission from $\mathcal{H}$ will continue just so long as the e_k's are greater than zero, and they will remain greater than zero indefinitely after they are excited, even though lateral inhibition helps to decrease their values quickly. If is quite desirable, in the interest of minimizing transient $\Phi(e_i)$ activation, to seek an additional mechanism which can completely stifle transmission from $\mathcal{H}$ in finite time, and in fact quickly. This will be assured if we suppose that there exists a number τ_Φ, $0 < \tau_\Phi < \min_m M_m$, such that no Φ production occurs whenever $e_k \leq \tau_\Phi$. That is, we postulate the existence of a threshold τ_Φ, higher than the e_k equilibrium threshold, for the $e_k \longrightarrow \Phi(e_k)$ process. If τ_Φ is sufficiently large and the rate of growth of $\mathcal{H}$ -inputs is well-controlled, then the growing $e_1 = e_2 = \ldots = e_n$ values generated by equal inputs can laterally suppress one another before any e_k reaches the τ_Φ level, whence no Φ production will occur and the organism is spared several moments of behavioral confusion. Notice that some lateral inhibitory transmission must occur before e_k reaches τ_Φ to insure that τ_Φ is never reached. If a nontrivial transmission threshold τ_L for

lateral transmission exists, therefore, it must be strictly smaller than Υ_Φ, whence we have the inequalities $\Upsilon_\Phi > \Upsilon_L \geq \Upsilon$, where Υ is the equilibrium potential of the $\mathcal{C}$ points. Given $(\Upsilon, \Upsilon_L, \Upsilon_\Phi)$, the rate of growth of $\mathcal{C}$-inputs are adjusted so that lateral inhibition of equal e_k effectively stifles Φ transmission.

How can these formal conditions be realized in an actual field? A simple way is the following: To each point q_i, associate a point $\tilde{q}_i$. Endow the set $\{\tilde{q}_i\}$ with a field structure of its own, that is homogeneous when $\mathcal{C}$ is homogeneous, and denote the resulting fields by $\mathcal{C} = \mathcal{F}\{q_i\}$ and by $\tilde{\mathcal{C}} = \mathcal{F}\{\tilde{q}_i\}$. We call $\tilde{\mathcal{C}}$ the double of $\mathcal{C}$. Draw lines from q_i to $\tilde{q}_i$ for every i and define $\tilde{p}_{ii} > 0$ as the line connection numbers from q_i to $\tilde{q}_i$. Now simply suppose that the strength functions $\{\tilde{e}_i\}$ of the $\tilde{q}_i$, not the e_k, generate the Φ functions. Let the $q_i \longrightarrow \tilde{q}_i$ transmission threshold, $\Upsilon_{q_i \longrightarrow \tilde{q}_i}$, exceed the lateral inhibitory threshold for $\mathcal{C}$, and let all $\mathcal{C}$ thresholds and the $\tilde{e}_k \longrightarrow \Phi(\tilde{e}_k)$ threshold be at least as large as the respective equilibrium thresholds (diagram 11). The inequalities $\Upsilon_\Phi > \Upsilon_L \geq \Upsilon$ are thus realizable in no fewer than two geometrical transmission steps.

In summary, generating stable net averages in finite time of the behavior of an entire field $\mathcal{F}$ requires: (1) some analog of a complete set of antagonists, (2) a nontrivial distribution of transmission thresholds, and (3) restrictions on the possible line connections (as in the $q_i \longrightarrow \tilde{q}_i$ connection; $q_i \not\longrightarrow \tilde{q}_j$, $j \neq i$).

68. Response Availability and Complete Sets of Antagonists. Elimination of Functionals

The use of nontrivial thresholds and the $\bar{H}$ function together occurred in just one previous place: in estimating the response availability functions A_i relative to the response number (threshold!) $\Upsilon > 0$. It will be recalled that $A_i = s_i \bar{H}(s_1^*, \ldots, s_n^*)$, where the $s_i^* = s_i / \Sigma_k s_k$. In the analogy between

$\overline{H}(p_1, \ldots, p_n)$ and a complete set of antagonists, no such normalization of s_i to s_i^* was discussed. The absence of normalization of this type in a complete set of antagonists is compensated by the unnormalized s_i value in the definition of A_i. The availability functions thus emerge as a first-order way of expressing the fact that the s_i functions are transformed in a field resembling $\mathcal{C} \cup \widetilde{\mathcal{C}}$ before they generate a macroscopic behavioral verbal act, just as these functions are renormalized before generating a nonneural correlate of net affect. The artificial $\overline{H}(s_1^*, \ldots, s_n^*)$ normalization is replaced in the $\mathcal{C} \cup \widetilde{\mathcal{C}}$ field by ordinary dynamical interactions. This replacement illustrates the fact that embedding fields are capable of renormalizing themselves in an intrinsic way, whatever the total number of field points, in response to the interactions of only the recently activated points. Such a property is, of course, crucial to the proper functioning of any dynamical system whose task is to order a continually growing collection of embedded experientially induced forms.

Along with the dynamical improvement, we find that the original, and not very revealing, response point $p_{(r)}$ is replaced by an extended structural carrier of $\mathcal{C} \cup \widetilde{\mathcal{C}}$ type. Also, an abstract functional $\overline{H}(\cdot, \ldots, \cdot)$ is replaced through this geometrical extension by honest dynamics. It was already suggested that this would happen when we discussed input functions $\Sigma_k P_k e^{Q_k}$ with functional coefficients and exponents. In this case, the functionals "lifted" Ω_o to the extended structural carrier of $\bigcup_{t<T} \Omega_t$. It has also just occurred for the balancing functions N_{ld}, which have been replaced by complete sets of antagonists. We now erect the possibility of such a replacement into a working

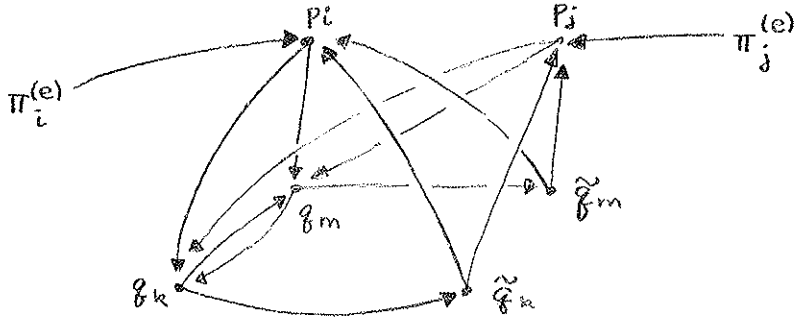
Postulate: Whenever it seems empirically necessary in a given structural carrier to work with functionals of dynamical quantities, there exists an extended carrier and new dynamical variables which eliminate the need to use functionals and for which the original field is, at best, a first-order approximation.

A final remark about the use of information functionals in

psychology can now be made. The success of information theory in telephone trunking problems, joined with the superficial analogy between trunking systems and central neurophysiology, has generated the application of information theoretical methods in various branches of psychology. Almost invariably, the information measures have been taken of macroscopic variables and, for every empirical agreement with information theoretical expectations based on trunking systems, a deviation has elsewhere appeared. The method is thus simultaneously promising and frustrating. Our discussion of the analogy between informational functionals and embedding dynamics shows why the hopes for the method were not whimsical, for analogies of information functionals arise whenever nontrivial lateral inhibitory effects are generated, and such effects are pervasive in neural interactions. At the same time, this discussion suggests some of the additional subtleties of embedding fields, including the continual fluctuations in the field of fundamental behavioral units and the delicate temporal factors in lateral inhibitory interactions, which transcend the information theoretical approach and generate inevitable partial frustration in its use. By extending our remarks on local dynamics, antagonists, thresholds, and inhomogeneous line distributions, it will be possible to see more clearly why the trunking and, simultaneously, the computer analogies of central processes break down in the most essential places.

69. Some Notation

We now pass to slightly more complicated fields which will gradually be made neurologically more realistic. Consider the following diagram:



To discuss it conveniently, some new notation must be introduced. Let $p = \{p_i\}$, $q = \{q_i\}$, $\tilde{q} = \{\tilde{q}_i\}$, and again write $\mathcal{F}(p) = \mathcal{F}(\{p_i\}) = \mathcal{F}\{p_i\}$ for the field whose points are $\{p_i\}$. Given any field $\mathcal{F}$, let $\mathcal{P}(\mathcal{F})$ be the point set of the embedding carrier of $\mathcal{F}$, $\mathcal{L}(\mathcal{F})$ the set of lines, $\mathcal{S}(\mathcal{F})$ the set of strength functions, $\mathcal{C}(\mathcal{F})$ the set of line functions, $\mathcal{M}(\mathcal{F})$ the set of M functions, and so on. In particular $\mathcal{P}(\mathcal{F}(p)) = p$. Let $\mathcal{F}_t$ denote the field $\mathcal{F}$ with its functions evaluated at time t. For all fields which have thus far been considered in particular examples, $\mathcal{P}(\mathcal{F}_t)$ has been independent of t. When $\mathcal{P}(\mathcal{F}_t)$ varies with t, new fundamental units are being formed. Two fields $\mathcal{F}$ and $\mathcal{G}$ are said to be disjoint if $\mathcal{P}(\mathcal{F}) \cap \mathcal{P}(\mathcal{G}) = \phi$, and to be disconnected (or unconnected) if no lines join an element of $\mathcal{P}(\mathcal{F})$ to one of $\mathcal{P}(\mathcal{G})$. Since all of our present equations have the property:

$$p_{ij}c_{ij}(t_o) = 0 \text{ implies } \qquad p_{ij}c_{ij}(t_1) = 0,\ t_1 \geq t_o,$$

we always assume l_{ij} is removed from the structural carrier whenever $p_{ij}c_{ij}(t_o) = 0$ for some t_o to avoid trivialities. A carrier with all such l_{ij}'s removed is called a minimal carrier (of the first kind). All carriers are henceforth automatically assumed to be minimal unless explicitly noted to the contrary.

Given any two fields $\mathcal{F}(p)$ and $\mathcal{F}(q)$, $\mathcal{F}(p) \cup \mathcal{F}(q)$ is the field whose points are $p \cup q$; that is $\mathcal{F}(p) \cup \mathcal{F}(q) = \mathcal{F}(p \cup q)$. When new points are added to a field, it is understood that the lines connecting these points with the old points are also added. In particular, when $p \cap q = \phi$, we write $\mathcal{F}(p) \oplus \mathcal{F}(q) = \mathcal{F}(p \oplus q)$ with "$\oplus$" instead of "$\cup$" to denote the process of combining fields. In particular $\mathcal{S}(\mathcal{F} \oplus \mathcal{G}) \neq \mathcal{S}(\mathcal{F}) \oplus \mathcal{S}(\mathcal{G})$, where the "$\oplus$" on the right hand side is the usual set-theoretical disjoint union. This lack of equality follows from the possible introduction of new interactions between $\mathcal{P}(\mathcal{F})$ and $\mathcal{P}(\mathcal{G})$ under

the " $\oplus$ " operation. Similarly $\mathcal{F}(p) \cap \mathcal{F}(q) = \mathcal{F}(p \cap q)$ and in general $\mathcal{S}(\mathcal{F}) \cap \mathcal{S}(\mathcal{G}) \neq \mathcal{S}(\mathcal{F} \cap \mathcal{G})$. The various set theoretical symbols are hereby introduced as commands to consider the fields induced by operating on the point carriers according to the customary set theoretical interpretation. In is more proper to write these relations as, say, $\mathcal{F}(p) \cap_{\mathcal{F}} \mathcal{F}(q) = \mathcal{F}(p \cap q)$ to distinguish between the two kinds of operations, but no confusion can result by writing "$\cap$" instead of "$\cap_{\mathcal{F}}$" for convenience.

Given two disjoint fields $\mathcal{F}$ and $\mathcal{G}$, we say that a field line function is <u>associated with</u> $\mathcal{F} \oplus \mathcal{G}$ if its first index labels a point in $\mathcal{P}(\mathcal{F})$ and its second a point in $\mathcal{P}(\mathcal{G})$. If every function associated with $\mathcal{F}_t(p) \oplus \mathcal{F}_t(q)$ is the same, we say that $\mathcal{F}_t(p)$ <u>factors</u> $\mathcal{F}_t(q)$, and write this relation $\mathcal{F}_t(p) \mid \mathcal{F}_t(q)$. If $\mathcal{F}_t(q) \mid \mathcal{F}_t(p)$ as well, we write $\mathcal{F}_t(p) \phi \mathcal{F}_t(q)$ for brevity.

If $\mathcal{F}_t(p) \mid \mathcal{F}_t(q)$ for all t under consideration, we write $\mathcal{F}(p) \mid \mathcal{F}(q)$. If only one function τ associated with $\mathcal{F}_t(p) \oplus \mathcal{F}_t(q)$ factors, we write $\mathcal{F}_t(q) \mid_{\tau} \mathcal{F}_t(p)$.

Suppose that $\mathcal{F}_t(p) \cap \mathcal{F}_t(q) = \phi$ and that $\Lambda_t : p \longrightarrow q$ is a 1-1, onto mapping with $\Lambda_t(p_i) = q_i$ which induces the natural maps

$$\Lambda_t^{\mathcal{S}} : \mathcal{S}(\mathcal{F}_t(p)) \longrightarrow \mathcal{S}(\mathcal{F}_t(q))$$

$$\Lambda_t^{\mathcal{C}} : \mathcal{C}(\mathcal{F}_t(p)) \longrightarrow \mathcal{C}(\mathcal{F}_t(q))$$

of all field quantities. In particular, $\Lambda_t^{\mathcal{S}}$ takes the strength function of p_i into the strength function of q_i, both evaluated at time t. When all of $\Lambda_t^{\mathcal{S}}$, $\Lambda_t^{\mathcal{C}}$, ..., are identity transformations, we say that $\mathcal{F}(p)$ is a <u>copy</u> of $\mathcal{F}(q)$ at time t and write $\mathcal{F}_t(p) \simeq \mathcal{F}_t(q)$. If only, say, $\Lambda_t^{\mathcal{S}}$ is the identity, we write $\mathcal{S}(\mathcal{F}_t(p)) \simeq \mathcal{S}(\mathcal{F}_t(q))$, and so on.

Suppose now that a third field $\mathcal{G}_t$ is given and a relation R pairing each of $\mathcal{F}_t(p)$ and $\mathcal{F}_t(q)$ with $\mathcal{G}_t$; for example, we write $R(\mathcal{F}_t(p), \mathcal{G}_t)$ to designate this relation in first case. Further suppose that Λ_t induces an identity transformation between those field functions of $\mathcal{F}_t(p)$ which are R-related to $\mathcal{G}_t$ and those of $\mathcal{F}_t(q)$ which are so related. We then write $R(\mathcal{F}_t(p), \mathcal{G}_t) \simeq R(\mathcal{F}_t(q), \mathcal{G}_t)$, or $\mathcal{F}_t(p) \simeq_R \mathcal{F}_t(q) \pmod{\mathcal{G}_t}$, and say that $\mathcal{F}_t(p)$ and $\mathcal{F}_t(q)$ are R-copies modulo $\mathcal{G}_t$. For example, if R denotes factorization, then the R-related quantities are the lines joining each of $\mathcal{F}_t(p)$ and $\mathcal{F}_t(q)$ with $\mathcal{G}_t$, and $\mathcal{F}_t(p) \mid \mathcal{G}_t \simeq \mathcal{F}_t(q) \mid \mathcal{G}_t$ means firstly that $\mathcal{F}_t(p) \mid \mathcal{G}_t$ and $\mathcal{F}_t(q) \mid \mathcal{G}_t$, and secondly that the lines joining $\mathcal{F}_t(p)$ to $\mathcal{G}_t$ have the same values as the Λ_t-corresponding lines joining $\mathcal{F}_t(q)$ to $\mathcal{G}_t$.

These various field operations give us a concise notation with which to build larger fields from smaller fields, to concentrate attention on subsets of given fields, and to compare field quantities in various pertinent ways. In terms of this notation, a field $\mathcal{F}(p)$, $p = \{p_i : i = 1, 2, \ldots, n\}$ is homogeneous at time t if and only if

$$(i) \quad \mathcal{F}_t(p_i) \simeq \mathcal{F}_t(p_j)$$

and

$$(ii) \quad \mathcal{F}_t(p_i) \phi \mathcal{F}_t(p \smallsetminus (p_i)) \simeq \mathcal{F}_t(p_j) \phi \mathcal{F}_t(p \smallsetminus (p_j)),$$

for all i and j. Or, in the "$\simeq$" sense, $\mathcal{F}_t\{p_i\}$ and $\mathcal{F}_t\{p_i\} \phi \mathcal{F}_t(p \smallsetminus \{p_i\})$ are independent of i, which is both a concise and geometrically transparent way to describe the situation.

By letting $T(p_i, q_j)$ be the time required to transmit from p_i to q_j, we can extend the above discussion to temporal interactions in a straightforward way. An inequality like

$$T(\mathcal{P}(\mathcal{F}), \mathcal{P}(\mathcal{G})) > T(\mathcal{P}(\mathcal{H}), \mathcal{P}(\mathcal{J})),$$

or, for brevity,

$$T(\mathcal{F}, \mathcal{G}) > T(\mathcal{H}, \mathcal{J}),$$

means that every transmission from $\mathcal{P}(\mathcal{F})$ to $\mathcal{P}(\mathcal{G})$ takes longer than any transmission from $\mathcal{P}(\mathcal{H})$ to $\mathcal{P}(\mathcal{J})$. T may be viewed as a relation between fields, whence

$$T(\mathcal{P}, \mathcal{Q}) \simeq T(\mathcal{R}, \mathcal{Q})$$

becomes the statement that transmission times from $\mathcal{P}$ to $\mathcal{Q}$ are the same as those from $\mathcal{R}$ to $\mathcal{Q}$. And so on.

70. Pairwise Homogeneous Fields

A weaker notion than field homogeneity, but a very important one, is the following. Let us be given two fields $\mathcal{F}_t(p)$ and $\mathcal{F}_t(q)$ such that

1) $\mathcal{F}_t(p)$ and $\mathcal{F}_t(q)$ are homogeneous,
2) $\mathcal{F}_t(p) \mid \mathcal{F}_t(q_j)$ is independent of j,
3) $\mathcal{F}_t(q) \mid \mathcal{F}_t(p_i)$ is independent of i.

$\mathcal{F}(p)$ and $\mathcal{F}(q)$ are said to be pairwise spatially homogeneous at time t. When conditions (1)-(3) are replaced by conditions (1')-(3'), which differ from (1)-(3) only in that $R(\mathcal{F}, \mathcal{G}) = \mathcal{F} \mid \mathcal{G}$ is replaced by $R(\mathcal{F}, \mathcal{G}) = T(\mathcal{F}, \mathcal{G})$, we say that $\mathcal{F}(p)$ and $\mathcal{F}(q)$ are pairwise temporally homogeneous at time t. $\mathcal{F}(p)$ and $\mathcal{F}(q)$ are pairwise homogeneous at time t whenever both (1)-(3) and (1')-(3') hold. Given a field $\mathcal{F}(p)$, if there exist a nontrivial decomposition $p = p^1 \oplus p^2 \oplus \ldots \oplus p^n$ and a t such that $\mathcal{F}_t(p^i)$ and $\mathcal{F}_t(p^j)$ are pairwise homogeneous, all i and j, we say that $\mathcal{F}(p)$ possesses a partition into homogeneous pairs at time t.

71. Replicated Averages, Ordered Thresholds, and Spectral Analysis

We can now return to the diagram, which we consider at initial

time $t = 0$. Suppose that $\mathcal{T}_0(p)$, $\mathcal{T}_0(q)$, and $\mathcal{T}_0(\tilde{q})$ partition $\mathcal{T}_0(p \oplus q \oplus \tilde{q})$ into homogeneous pairs. (The condition $\mathcal{T}_0(q) \mid \mathcal{T}_0(\tilde{q}_m)$ becomes $\mathcal{T}_0(q_m) \mid \mathcal{T}_0(\tilde{q}_m)$ by the minimality of the field.) $\mathcal{T}(q)$ is no longer to be considered a complete set of antagonists; all interactions are excitatory. $\tau_{\tilde{q}_k \to p_i} > 0$ for all k and i.

In the absence of $\mathcal{T}(\tilde{q}) \to \mathcal{T}(p)$ lines, no conceivable $\pi_i^{(e)}$ can disturb the fact that $\mathcal{T}_t(q)$ and $\mathcal{T}_t(\tilde{q})$ remain pairwise homogeneous for all $t > 0$. In other words, pairwise homogeneity propagates through time for these subfields. Only p among all of the local point fields sustains a non-homogeneous point field. The reason for this is obvious since the $\pi_i^{(e)}$ need not be distributed uniformly. Turned about, this observation means that in a true tabula rasa, or globally homogeneous initial field, no learning is possible. The possibility of inducing nontrivial line residues depends critically on inhomogeneities in the initial field, which include the preparation of nonuniform inputs. Local homogeneities can be extremely useful, however. In the present case, for example, each $S(\tilde{q}_k)$ is responsive to the total $\mathcal{L}(\mathcal{T}(p))$ distribution of excitation, via $\mathcal{T}(q)$, and the $\mathcal{T}(q) \to \mathcal{T}(q)$ interactions help to sustain this excitation within $\mathcal{T}(q \oplus \tilde{q})$. Each $\mathcal{T}_t(\tilde{q}_k)$ is a replica of its $\tilde{q}$ neighbors, so that we have an entire family, conceivably an enormous one, of sources of sustained excitation which measure the total excitation of the field $\mathcal{T}(p)$.

Now allow $\mathcal{T}(\tilde{q}) \to \mathcal{T}(p)$ interactions to occur. Letting $\tau(\tilde{q}_k, p_i)$ be the transmission threshold from $\tilde{q}_k$ to p_i, suppose that $\mathcal{T}(\tilde{q}_i) \mid_\tau \mathcal{T}(p)$ for every i. The various thresholds τ may be indexed so that $0 < \tau_1 < \tau_2 < \ldots < \tau_m$. Suppose that there are ω_k $\tilde{q}_j$'s with τ_k as threshold, forming the set $Q_k \subset q$, with $\Sigma_k \omega_k = n$.

What is the effect of this arrangement on the development of line residues? The lines $\mathcal{L}(\mathcal{T}(Q_1), \mathcal{T}(p))$ will be responsive to the largest number of $\pi_i^{(e)}$ of all the $\mathcal{L}(\mathcal{T}(\tilde{q}), \mathcal{T}(p))$ since τ_1, as the lowest threshold, imposes the weakest condition on $\mathcal{T}(\tilde{q}) \to \mathcal{T}(p)$ transmissions. Even fairly small inputs can induce transmission from Q_1 and thereby

generate new patterns in $C(\mathcal{T}(\Omega_1), \mathcal{T}(p))$ which record the asymmetries in $\mathcal{S}(p)$. $\mathcal{L}(\mathcal{T}(\Omega_2), \mathcal{T}(p))$ will be responsive to a smaller collection of inputs than of $\mathcal{L}(\mathcal{T}(\Omega_1), \mathcal{T}(p))$, while $\mathcal{L}(\mathcal{T}(\Omega_m), \mathcal{T}(p))$ will be the least responsive set of all. A hierarchy of line residues is hereby determined that distinguishes inputs by the total excitation which they generate. Phrased in another way, if we view the possible values of a strength function as a type of excitation or energy spectrum, then we have in this situation a collection of recording devices that are selectively sensitive to various subportions of this spectrum.

Still more is achieved. Consider some $\pi_i^{(e)}$ which is sufficiently intense to generate an $S(\tilde{q}_k)$ function satisfying $\max_{t \in [0,\infty)} S(\tilde{q}_k, t) > \tau_m$. Then all of the $\mathcal{L}(\mathcal{T}(\Omega_k), \mathcal{T}(p))$, $k=1, 2, \ldots, m$, shall eventually transmit and all of the $C(\mathcal{T}(\Omega_k), \mathcal{T}(p))$ shall record $\mathcal{S}(p)$ asymmetries. The functions $S(\tilde{q}_k, t)$ wax and wane in a regular progression, however, as do the $\mathcal{S}(p)$ functions. The purely numerical staggering of τ values is therefore translated into a temporal staggering in the line recording process. The various phases of the joint temporal development of $\mathcal{S}(\tilde{q})$ and $\mathcal{S}(p)$ are recorded in different collections of lines.

72. Nonlocalizability and Stability of Representations

A space-time continuum has hereby been represented in a purely spatial way. If we now imagine momentarily that the $\pi_i^{(e)}$ vector represents some analog of a perceptual event, then we must say that the record of this event is literally spread about in the embedding space. The most intensive study of line residues in a small portion of this space could not tell you that the event occurred. The embedded representation of the event simply cannot be localized. If we turn this remark around, it follows that deleting a small number of $\mathcal{L}(\mathcal{T}(\tilde{q}), \mathcal{T}(p))$ cannot seriously upset this representation, which is another way of saying that the representation is highly stable.

73. Escalation and Self-Improving Discrimination

Notice that if some lines in $\mathcal{L}(\mathcal{T}(\Omega_k), \mathcal{T}(p))$ become large, $k < m$, due to the repetition of a certain $\pi_i^{(e)}$, then transmitted strength from Ω_k to the privileged p_j's will increase, whence the larger $S(p_j)$'s thereby induced will generate higher $\mathcal{L}(\mathcal{T}(q \oplus \tilde{q}))$ values. In this way, the next level, $\mathcal{L}(\mathcal{T}(\Omega_{k+1}), \mathcal{T}(p))$, might be activated and employed thereafter to better distinguish the various phases of the excitation patterns. The net result of this escalation of the spectrum and involvement of new lines is often a self-improving discrimination of the $\mathcal{T}$-input process.

The lines leading from $\tilde{q}$ need not terminate on p. Suppose, in fact, that $\mathcal{L}(\mathcal{T}(\tilde{q}), \mathcal{T}(p)) = \phi$, and construct $\mathcal{L}(\mathcal{T}(\tilde{q}), \mathcal{T}(r))$, where $r \cap (p \oplus q \oplus \tilde{q}) = \phi$. In this situation, pairwise homogeneity of $\mathcal{T}_0(q)$ and $\mathcal{T}_0(\tilde{q})$ strictly propagates in time: $\mathcal{T}(q \oplus \tilde{q})$ remains an "unbiased estimator" of the total $\mathcal{L}(\mathcal{T}(p))$ excitation through time as it simultaneously decomposes the total excitation spectrum within the $\{\mathcal{L}(\mathcal{T}(\Omega_k), \mathcal{T}(r))\}$ sets and correlates this spectrum with the $\mathcal{L}(r)$ spectrum within $\{\mathcal{L}(\mathcal{T}(\Omega_k), \mathcal{T}(r))\}$.

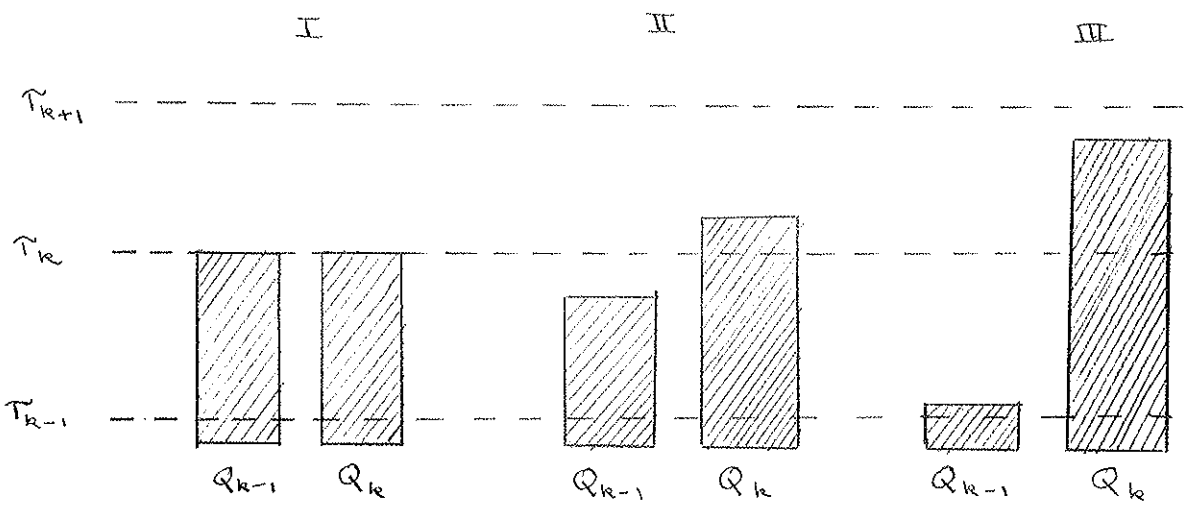
74. Narrow Band Analysers

The sensitivity of $\{\mathcal{L}(\mathcal{T}(\tilde{q}), \mathcal{T}(r))\}$ to special excitatory spectral components can be adjusted by varying the ω_k distribution. In all cases, however, the $\{\mathcal{L}(\mathcal{T}(\Omega_1), \mathcal{T}(r))\}$ are wide-band transmitters compared to $\{\mathcal{L}(\mathcal{T}(\Omega_m), \mathcal{T}(r))\}$. Is it possible to construct a set of lines whose c values record activation within a series of non-overlapping narrow bands of strength excitation? To do this, at least qualitatively, again suppose that we are given ordered sequences $\Omega_1, \ldots, \Omega_m$ and $0 < \tau_1 < \tau_2 < \ldots < \tau_m$. Attach lines $\tilde{L}_{ki}$ from Ω_k to Ω_i, $i \leq k-1$, in a factorizable way, and suppose that these lines carry inhibitory excitation with transmission threshold close to τ_k. One example of such a system, for volume conductors, determines its $\tilde{e}_k$ functions by

$$\frac{d\tilde{e}_k}{dt} = \tilde{\alpha}(\tilde{M}_k - \tilde{e}_k)(\tilde{r}e_k(t-\tilde{t}_{kk})\tilde{p}_{kk}\tilde{c}_{kk}$$

$$- \tilde{\epsilon}\tilde{e}_k(1 + \tilde{\gamma}\Sigma_{n=k+1}^{m} \max\left[\tilde{e}_n(t-\tilde{T}_{ni}) - (\tau_n + \epsilon_n),\ 0\right]\tilde{p}_{nk}),$$

where $|\epsilon_n| < \min\{\tau_{n+1} - \tau_n,\ \tau_n - \tau_{n-1}\}$ and $\tau_k + \epsilon_k < \tau_{k+1} + \epsilon_{k+1}$, for all k. Whenever $s(Q_k)$ exceeds τ_k, the activation of $\{\mathcal{L}(\mathcal{T}(Q_k), \mathcal{T}(r))\}$ will be associated with the tendency for $\{\mathcal{L}(\mathcal{T}(Q_\lambda)), \mathcal{T}(r))\}$ transmission to be suppressed for all $\lambda \leq k-1$. When $\{\mathcal{L}(\mathcal{T}(Q_k)), \mathcal{T}(r))\}$ ceases to transmit, the recovery from inhibition in Q_{k-1} will allow $\{\mathcal{L}(\mathcal{T}(Q_\lambda), \mathcal{T}(r))\}$ to transmit once again. Thus, only $\{\mathcal{L}(\mathcal{T}(Q_k), \mathcal{T}(r))\}$ tends to transmit when the uninhibited $s(\tilde{q})$ function takes its values in $[\tau_k, \tau_{k+1})$, which accomplishes the spectral analysis into non-overlapping excitation intervals. The exact features of the spectral analysis produced depend sensitively on $\{\omega_k\}$, $\{\tau_k\}$, and the $\tilde{p}_{mi}$ and $\tilde{T}_{mi}$ values of the $\tilde{L}_{mi}$. In particular, suppose that $s(Q_k, t_o) = s(Q_{k-1}, t_o) = \tau_k$, and consider the following diagram, in which the height of the shaded regions gives the level of strength excitation.



This diagram shows one possible sequence of effects on $s(Q_{k-1}, t)$ as $s(Q_k, t)$ grows. Here we have assumed that k is the least i such that

$s(Q_{i+1}, t) = s(Q_{i+2}, t) = \ldots = s(Q_m, t)$. Even in (III), the $\mathcal{L}(\mathcal{T}(Q_{k-1}), \mathcal{T}(r))$ are transmitting, but the growing inhibition of $s(Q_{k-1}, t)$ induces a growing split in $s(Q_k, t) - s(Q_{k-1}, t)$ and an increase in $(s(Q_k, t) - \tau_k) / (s(Q_{k-1}, t) - \tau_{k-1})$. If it is desired that $\mathcal{L}(\mathcal{T}(Q_{k-1}), \mathcal{T}(r))$ transmission be suppressed more completely when $\mathcal{L}(\mathcal{T}(Q_k), \mathcal{T}(r))$ begins to transmit than in this diagram, we must require that the recovery rate from inhibition be rapid. Otherwise there will exist relatively long periods after $s(Q_k, t)$ sinks into $[\tau_{k-1}, \tau_k)$ during which $\mathcal{L}(\mathcal{T}(Q_{k-1}), \mathcal{T}(r))$, and possibly $\mathcal{L}(\mathcal{T}(Q_\omega), \mathcal{T}(r))$ for several $\omega < k - 1$, will not transmit. The size of the recovery rate from inhibition must in turn be measured relative to the rates of growth and decline of the $\pi_i^{(e)}$, which determine the natural time scale of the field. The growing suppression of $s(Q_{k-1}, t)$ by $s(Q_k, t)$ will also reduce the suppression which $s(Q_{k-1}, t)$ exerts on $s(Q_\omega, t)$, $\omega \leq k - 2$. This reduction must be compensated by the growing $s(Q_k, t)$ suppression of the $s(Q_\omega, t)$, which is why lines $\tilde{L}_{ki}$ for all $i \leq k - 1$ are required.

In the above example, we have seen that a fairly trivial arrangement of points, lines, and thresholds exhibits interesting synthesizing and analysing properties. Each of the observed effects can be brought to a high degree of sensitivity either by choosing constants carefully or by arranging the field in a more subtle way. A somewhat more realistic field which extends the above example is the following one.

75. Fields of Type M

Suppose we are given a finite collection $\{\mathcal{T}(p^{(i)})\}$ of pairwise disjoint fields, where $p^{(i)} = \{p_j^{(i)}, j = 1, 2, \ldots, n_i\}$. To every pair $\mathcal{T}(p^{(i)})$ and $\mathcal{T}(p^{(j)})$, associate a pair of fields $\mathcal{H}(p^{(i,j)})$ and $\mathcal{H}(p^{(j,i)})$, possibly vacuous, such that:

1) $\mathcal{T}(p^{(i)})$, $\mathcal{T}(p^{(j)})$, $\mathcal{H}(p^{(i,j)})$, and $\mathcal{H}(p^{(j,i)})$ are pairwise disjoint and pairwise homogeneous.

2) the $\mathcal{H}(p^{(i,j)})$ are pairwise disconnected, $j \neq i$,

3) only one $\mathcal{H}(p^{(i,i)})$ exists for every i,

4) no lines $\mathcal{L}(\mathcal{H}(p^{(j,i)}), \mathcal{T}(p^{(j)}))$ and $\mathcal{L}(\mathcal{T}(p^{(j)}), \mathcal{H}(p^{(i,j)}))$ exist, $j \neq i$; that is, excitation flows in the direction $\mathcal{T}(p^{(j)}) \rightarrow \mathcal{H}(p^{(j,i)}) \rightarrow \mathcal{T}(p^{(i)})$.

Let $\mathcal{G}$ be the total field, $\mathcal{G} = \mathcal{T}(\oplus_i p^{(i)}) \oplus \cdots \oplus \mathcal{H}(\oplus_{j,k} p^{(j,k)})$. $\mathcal{G}$ may be viewed as being subdivided into two layers $\mathcal{L}_i = \mathcal{L}_i(\mathcal{G})$, $i = 1, 2$, with the points $\oplus_i p^{(i)}$ in $\mathcal{L}_1$ and $\oplus_{j,k} p^{(j,k)}$ in $\mathcal{L}_2$. $\mathcal{G}$ is called a <u>field of type</u> $(2, 2)$, where the first 2 indicates the number of layers and the second 2 gives the number of fields in the first layer which are connected to a field in the second layer (diagram 12). We now write $\mathcal{L}_{ij} = \mathcal{L}(\mathcal{T}(p^{(i)}), \mathcal{T}(p^{(j)}))$ and $\mathcal{L}_{ij,k} = \mathcal{L}(\mathcal{H}(p^{(i,j)}), \mathcal{T}(p^{(k)}))$ for brevity. An input sequence to just $\mathcal{T}(p^{(i)})$ generates inhomogeneous line residues only in $\mathcal{L}_{ii}$ and $\mathcal{L}_{ii,i}$; a sequence to $\mathcal{T}(p^{(i)} \oplus p^{(j)})$ alone generates such residues only in $\mathcal{L}_{ii}$, $\mathcal{L}_{ij}$, $\mathcal{L}_{ji}$, $\mathcal{L}_{jj}$, $\mathcal{L}_{ji,i}$ and $\mathcal{L}_{ij,j}$; and so on. Fields of type $(2, 2)$ can be made to provide all of the effects of the previous example, but these effects are restricted to proper subfields of the various layers. When a large number of $\mathcal{T}(p^{(i)})$ exist and the input array is broadly distributed among these subfields, we find much more finely discriminating line residues than we do in two layers for which all of $\mathcal{T}(\oplus_i p^{(i)})$ interacts with a single $\mathcal{H}$ in a pairwise homogeneous way.

Fields of <u>type</u> $(2, n)$ may be similarly constructed, with two layers and n fields of $\mathcal{L}_1$ connected to a given field of $\mathcal{L}_2$. Various subclasses of $(2, n)$ are the fields of <u>type</u> $(2, n, k)$, $k = 1, 2, \ldots n-1$. In a field of type $(2, n, k)$, for any $\mathcal{L}_2$ subfield $\mathcal{H}(p^{(i_1, i_2, \ldots, i_n)})$, the fields $\mathcal{T}(p^{(i_j)})$, $j = 1, 2, \ldots, k$, send lines to $\mathcal{H}(p^{(i_1, \ldots, i_n)})$, while $\mathcal{T}(p^{(i_j)})$ $j = k+1, \ldots, n$, receive lines from $\mathcal{H}(p^{(i_1, \ldots, i_n)})$. In particular, all fields of type $(2, 2)$ are fields of type $(2, 2, 1)$. When several n's and k's appear, say in the pairs (n_i, k_i), $i = 1, 2, \ldots, m$, in the second layer of a given field, we call the field $(2, n_1, k_1) \oplus (2, n_2, k_2) \oplus \ldots \oplus (2, n_m, k_m)$, or simply $(2; n_1, \ldots, n_m; k_1, \ldots, k_m)$.

Fields within $\mathcal{L}_2$ are often connected to a third layer, $\mathcal{L}_3$,

$\mathcal{L}_3$ is connected to $\mathcal{L}_4$, and so on. To have a convenient notation for such situations, we define fields of type M. $M = \{m_{ij}\}$ is a k x k matrix whenever k layers $\mathcal{L}_i$, $i = 1, 2, \ldots, k$, exist. $m_{ij} = n$ means that lines from n fields in $\mathcal{L}_i$ pass to a given field in $\mathcal{L}_j$ if any do. When we write $M = \oplus_i w_i M_i$, where w_i are positive real numbers and M_i are k x k matrices, and if $M_i = \{m^{(i)}_{km}\}$, there exist w_i fields in $\mathcal{L}_m$ which receive lines from $m^{(i)}_{km}$ fields in $\mathcal{L}_k$. For example, the general $\oplus_{i=1}^{m}(2, n_i, k_i)$ becomes $\oplus_{i=1}^{m} w_i \begin{pmatrix} r_i & k_i \\ n_i - k_i & m_i \end{pmatrix}$. This notation for classifying fields makes sense even when the subfields are not pairwise homogeneous. When a field of type M does have only pairwise homogeneous subfields, we say that we are given a homogeneous field of type M, or simply a homogeneous M.

Given two partitions $\{\mathcal{T}_i\}$ and $\{\mathcal{Q}_j\}$ of a given field, whenever every point set $\mathcal{P}(\mathcal{T}_i)$ is contained in some $\mathcal{P}(\mathcal{Q}_j)$, we say that $\{\mathcal{T}_i\}$ is a refinement of $\{\mathcal{Q}_j\}$. It is useful whenever we are given an embedding field $\mathcal{Q}$ to seek the maximal refinement of $\mathcal{Q}$ into pairwise homogeneous subfields, and then to consider the field as a homogeneous M with this decomposition. The value of such a decomposition derives from the fact that certain pairwise homogeneous properties propagate in time under particular input paradigms. Whenever subfields in M do not propagate, we must seek a new refinement of M that includes the new asymmetries induced by the input sequence. The introduction of external perturbations may in this way be studied as a step-wise refinement of local geometrical symmetries in the field. This point of view gives useful insights into the capacity of a given field to represent various classes of input paradigms, as we shall see in greater detail in the coming pages. Since one of the major tasks of neural structures is to be able to discriminate large numbers of different input paradigms, the search for propagating geometrical symmetries becomes a central theoretical problem.

76. Space-Time Summation and Multiple Lines

Consider homogeneous fields of type

$$M = \begin{pmatrix} m_{11} & m_{12} & \cdots & m_{1n} \\ m_{21} & & & \\ \vdots & & 0 & \\ m_{n1} & & & \end{pmatrix}$$

where reciprocal lines occur between all connected layers. It is easy to see that such fields, for various choices of the m_{ij}, strikingly resemble verbal fields with multiple line structures. This will especially be true if any of the following types of condition are built into the field:

1) The transmission threshold from points in layers $\mathcal{L}_i$, $i \neq 1$, is a monotone increasing function of m_{ij}, while the lines leading from $\mathcal{L}_1$ to these points have equal p_{kl} values,

2) The transmission threshold for points in layers $\mathcal{L}_i$, $i \neq 1$, is the same, but the p_{kl} values of lines leading from $\mathcal{L}_1$ to these points are monotone decreasing functions of the m_{ij} associated with their terminal point, or

3) some combination of these two effects. These conditions mean that when many subfields contribute to the activation of a single point in a lower layer, temporally summating excitation from a nontrivial fraction of these fields is necessary to excite the point sufficiently to exert a reciprocal influence on $\mathcal{L}_1$ through transmission to $\mathcal{L}_1$. By varying p densities to these points and their transmission thresholds as functions of the number of $\mathcal{L}_1$ points sending lines to each lower layer point, we determine the relative degree of reciprocal influence of sets of varying size in $\mathcal{L}_1$ on $\mathcal{L}_1$ via $\{\mathcal{L}_i : 2 \leq i\}$ transmission. This is essentially the content of the discussion of why ABC is a better generator of D than is C alone, a discussion which naturally generalizes to why certain inputs, after being conditioned by the arrival of earlier inputs, behave differently than when they are presented in isolation. New $\mathcal{L}(\oplus_{i=2} \mathcal{L}_i, \mathcal{L}_1)$ lines are activated by the combination of conditioning and terminal inputs which

are not activated by the terminal input alone. The entire dynamical trajectory is hereby shifted by the conditioning events. This shift in the dynamical trajectory due to conditioning inputs is discontinuous when it is looked at in terms of which points are transmitting at any time. When it is looked at in terms of the successive refinement by external perturbations of locally homogeneous subsets of the field, however, it is seen to take on a much more continuous, and natural, aspect.

The product functional $\prod_{k\in I} s_k(t-t_{ki})p_{Ii}\tilde{c}_{Ii}$ which we needed for the study of multiple lines is now no longer necessary. It is replaced by ordinary dynamics in an extension of the original field, just as our study of the information functional and of the determination of functional input sequences led us to expect in general. The kind of temporal facilitation within M whereby the activation of a single point requires many almost simultaneous transmissions from other points does not introduce any new dynamical ideas. What does become clear is that the proper measurement of temporal facilitations is a relative matter which only assumes really delicate proportions when the ratio of the line values p_{ik} to a given point and the point's transmission threshold is not constant over all of the points of the field. It is quite useful, whenever possible, to decompose every field into pairwise homogeneous subsets with respect to this important ratio. In fact, that is why we have considered this example to be a field of type M with n layers. All of the layers $\mathcal{L}_i$, $i \neq 1$, can easily be lumped into a single large second layer, but this obscures an important dynamical distinction. In the most sensitive fields, the p_{ik} values converging on a single point are not the same, so this ratio in its simplest form has no significance.

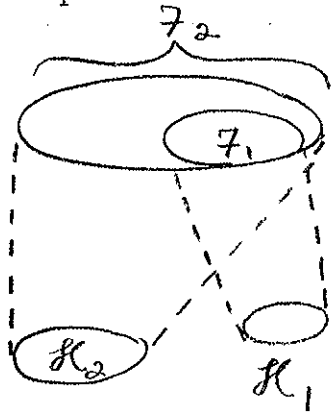
77. Overlapping Projection Domains

Let us now be given a general field $\mathcal{F}$ of type $(2, n_1, k_1)\oplus\cdots\oplus(2, n_m, k_m)$. Let $\mathcal{H}\in\mathcal{L}_2$ mean that $\mathcal{H}$ is one of the subfields of $\mathcal{F}$ which appears in $\mathcal{L}_2$ of the decomposition of $\mathcal{F}$ as $\oplus_{i=1}^{m}(2, n_i, k_i)$. For any

$\mathcal{H} \in \mathcal{L}_2$, denote the set of points in $\mathcal{L}_1$ which send lines to $\mathcal{H}$ by $sp^{-1}(\mathcal{H})$. We say that $sp^{-1}(\mathcal{H})$ is the projection domain of $\mathcal{L}_1$ to $\mathcal{H}$. Identical definitions hold for $\mathcal{H} \in \mathcal{L}_1$. Suppose that every $\mathcal{H} \in \mathcal{L}_2$ acts as a complete set of antagonists when $sp^{-1}(\mathcal{H})$ has a uniform strength distribution. What kind of input paradigms delivered to $\mathcal{L}_1$ generate nontrivial line residues in $\mathcal{L}(\mathcal{L}_2, \mathcal{L}_1)$?

If the paradigm is such that many input onsets occur in a rapid succession and are distributed with almost equal density to every point of some $sp^{-1}(\mathcal{H})$, $\mathcal{H} \in \mathcal{L}_2$, then lateral inhibition within $\mathcal{H}$ will almost completely stifle $\mathcal{L}(\mathcal{H})$ so that $\mathcal{L}(\mathcal{H}, \mathcal{L}_1)$ will not be activated and no line residues will form. An input paradigm $\mathcal{I}$ which excites $\mathcal{H}$ must be distributed in such a way that within any time period of the order of the relaxation time of $\mathcal{L}(\mathcal{H})$, at most a proper subset of $sp^{-1}(\mathcal{H})$ is homogeneously excited. We say that such an $\mathcal{I}$ is temporally partitioned modulo $\mathcal{H}$. We can classify $\mathcal{I}$'s in terms of the $\mathcal{H}$'s by which they are temporally partitioned. Intuitively, those $\mathcal{I}$'s which are partitioned by the largest number of $\mathcal{H}$'s are the input classes which the field can best discriminate.

Suppose that there exists some $\mathcal{T}_1 \in \mathcal{L}_1$ such that $\mathcal{T}_1 = sp^{-1}(\mathcal{H}_1)$ for some $\mathcal{H}_1 \in \mathcal{L}_2$ and $\mathcal{T}_1$ is a proper subset of $sp^{-1}(\mathcal{H}_2)$, for some $\mathcal{H}_2 \in \mathcal{L}_2$. Let a uniformly distributed input array be delivered to all of $\mathcal{T}_1$, and only to $\mathcal{T}_1$. The strength transmission induced by this array will be completely stifled within $\mathcal{H}_1$ but will be effective in generating line residues from $\mathcal{H}_2$ to $\mathcal{L}_1$.



An obvious continuation of this argument shows that by decomposing $\mathcal{L}_1$ into overlapping projection domains of various shapes and sizes, the set of

line loops from $\mathcal{L}_1$ to $\mathcal{L}_2$ and back to $\mathcal{L}_1$ becomes very selective of the kind of strength distribution which can be transmitted over them. The line residues are equally selective. This example resembles our example of the spectral analysis of a given excitation wave by an ordered set of thresholds. Here, ordering the thresholds introduces further refinements, but even with uniform thresholds, a sufficiently rich distribution of overlapping projection domains from $\mathcal{L}_1$ to $\mathcal{L}_2$ suffices to discriminate even minute differences in the global $\mathcal{S}(\mathcal{L}_1)$ distribution, and registers these differences in exquisite detail in disparate $\mathcal{L}(\mathcal{C}, \mathcal{L}_1)$ sets.

In particular, if we embed $\mathcal{L}_1$ into a simple planar region Γ, with $\mathcal{P}(\mathcal{L}_1)$ considered as a set of Euclidean points closely packed in Γ, then $(\mathcal{S}(\mathcal{L}_1))(t)$ almost becomes a surface in $\mathbb{R}^3$ for every t. We can in fact look at $\mathcal{S}(t) \equiv (\mathcal{S}(\mathcal{L}_1))(t)$ as a bona fide surface by smoothly extending $\mathcal{S}(t)$ to points in $\Gamma \sim \mathcal{P}(\mathcal{L}_1)$. The remarks about overlapping projection domains now show that $\{\mathcal{L}(\mathcal{C}, \mathcal{L}_1) : \mathcal{C} \in \mathcal{L}_2\}$ can be made into a delicate instrument for recording the existence not only of different spatial gradients in $\mathcal{S}(t)$ for every t, but also of different curvatures, and in fact practically any geometrical property of a surface that is thinkable. Such a geometrical property need not even be local. It can involve an arbitrary region of Γ. By ordering thresholds in $\mathcal{L}_2$ or building in graded temporal facilitation mechanisms, the time derivative of $\mathcal{S}(t)$ at any point can also be estimated, and so forth. Moreover, all such properties of the surface can be measured simultaneously. These striking differences between the mode of operation of $\{\mathcal{L}(\mathcal{C}, \mathcal{L}_1) : \mathcal{C} \in \mathcal{L}_2\}$ and the usual tests for measuring the properties of surfaces show how strongly we must depart from our accustomed geometrical notions to understand the functional geometry underlying neural processes.

78. Weighted Sets of Antagonists

An important generalization of the notion of complete sets of antagonists is the following: Our present definition hinges on the existence

of an $\mathcal{K}$ and a $sp^{-1}(\mathcal{K})$, such that whenever the points of $sp^{-1}(\mathcal{K})$ are uniformly excited, over a sufficiently long time interval, $\mathcal{L}(\mathcal{K})$ is suppressed below its transmission threshold. The requirement that $\mathcal{L}(sp^{-1}(\mathcal{K}))$ be uniformly excited must be relaxed in many empirical situations. In particular, suppose that an input vector $I = (I_1, I_2, \ldots, I_n)$ is delivered component-wise to $\mathcal{P}(sp^{-1}(\mathcal{K})) = (p_1, p_2, \ldots, p_n)$ so that $\lim_{t\to\infty} I$ exists and $\lim_{t\to\infty} (s_1, s_2, \ldots s_n) = (w_1, w_2, \ldots, w_n)$. For specificity one can imagine that $I_i = \overline{w}_i \chi_{[0,\infty)}$, where

$$\chi_I(t) = \begin{cases} 1 & \text{when } t \in I \\ 0 & \text{otherwise} \end{cases} .$$

If $\mathcal{L}(\mathcal{K})$ is suppressed below its transmission threshold under I, we say that $\mathcal{K}$ is a complete set of antagonists weighted by $(w_1, w_2, \ldots, w_n)$. $\mathcal{T}_1 = sp_w^{-1}(\mathcal{K})$ is used to denote that $\mathcal{K}$ is a weighted set of antagonists for $\mathcal{T}_1$ with weighting $w = (w_1, w_2, \ldots, w_n)$. The notation is used even when $sp^{-1}(\mathcal{K})$ properly contains $\mathcal{T}_1$.

Introducing weighted sets of antagonists into the previous field $\mathcal{T}$, it is no longer homogeneous input arrays which suppress $\mathcal{L}(\mathcal{K}, \mathcal{L}_1)$ transmission, but rather input arrays that generate vectors $(s_1, s_2, \ldots, s_n)$ which do not asymptotically deviate from the weighting $(w_1, w_2, \ldots, w_n)$. Whenever $\mathcal{T}_1 \in \mathcal{L}_1$, $\mathcal{T}_1 = sp_{w(1)}^{-1}(\mathcal{K}_1) = sp_{w(2)}^{-1}(\mathcal{K}_2) = \ldots = sp_{w(m)}^{-1}(\mathcal{K}_m)$, $1<<m$, and the $w^{(k)}$ are distributed over many independent rays in $\mathbb{R}^n$, $\{\mathcal{L}(\mathcal{K}_i, \mathcal{L}_1): i = 1, \ldots, m\}$ provides a sensitive measure of variations in $\mathcal{L}(\mathcal{T}_1)$. The notion of weighted sets of antagonists is useful in the common example of two layers $\mathcal{L}_1$ and $\mathcal{L}_2$ such that $\mathcal{L}_2$ does not decompose into disjoint complete sets of antagonists for $\mathcal{L}_1$. One usually finds, at best, overlapping sets in $\mathcal{P}(\mathcal{L}_2)$ which are weighted sets of antagonists, with possibly different weightings, for overlapping sets in $\mathcal{P}(\mathcal{L}_1)$. When this is true, one often seeks a covering of $\mathcal{P}(\mathcal{L}_1)$ by overlapping sets $\{\mathcal{P}(\mathcal{T}_i)\}$ and a covering of $\mathcal{P}(\mathcal{L}_2)$ by overlapping sets $\{\mathcal{P}(\mathcal{K}_i)\}$ such that $\mathcal{T}_i = sp_{w(i)}^{-1}(\mathcal{K}_i)$ and $\{\mathcal{P}(\mathcal{T}_i)\}$ is a particularly appropriate covering

of $\mathcal{P}(\mathcal{L}_1)$ with which to study a fixed input array. The vectors $f(i) = w^{(i)}$ provide a kind of moving average of this pairwise covering of $\mathcal{P}(\mathcal{L}_1 \oplus \mathcal{L}_2)$. More general coverings may also be sought associating several sets of one layer with a single set of the second layer, but the essential idea remains unchanged. Such considerations arise when studying the interaction of neocortex with subcortical nuclei like the thalamus.

79. A Many-One Field

Let us now be given a (2, 2) field $\mathcal{F}$ with $\mathcal{L}_1$ composed of pairwise disjoint copies $\mathcal{F}(p^{(j)})$, $j = 1, 2, \ldots, k$, where $p^{(j)} = \{p_i^{(j)}, i = 1, 2, \ldots, n\}$, and $\mathcal{L}_2$ consists of a single field $\mathcal{H}(p^{(1, 2, \ldots, k)})$ that receives lines from $\mathcal{L}_1$ but sends no lines to $\mathcal{L}_1$. Let $T_{ij}: \mathcal{F}(p^{(i)}) \longrightarrow \mathcal{F}(p^{(j)})$ be the copying transformation with $T_{ij}(p_k^{(i)}) = p_k^{(j)}$, $T_{ij}(s_k^{(i)}) = s_k^{(j)}$, etc. Augment $\mathcal{F}$ by a field $\mathcal{G} = \mathcal{G}(q)$, disjoint from $\mathcal{F}$, which is connected to $\mathcal{F}$ only by lines received from $\mathcal{L}_2$. Denote the input array to $\mathcal{L}_1$

$$\Pi(t) = \begin{pmatrix} \Pi_{11}(t), \Pi_{12}(t), \Pi_{13}(t), \ldots & \Pi_{1n}(t) \\ \Pi_{21}(t), \Pi_{22}(t), \ldots & \\ \Pi_{31}(t), \ldots & \vdots \\ \vdots & \\ \Pi_{n1}(t), \ldots \quad \ldots, & \Pi_{nn}(t) \\ \Pi_{n+1,1}(t), \ldots & \Pi_{n+1,m}(t) \end{pmatrix}$$

where $\Pi_{ij}(t)$ is the input to $p_j^{(i)}$ at time t, $i = 1, 2, \ldots, n$, and $\Pi_{n+1,k}(t)$ is the input to q_k.

Suppose that we are given two copies of $\mathcal{T} \oplus \mathcal{L}$, denoted by $\mathcal{T}_1 \oplus \mathcal{L}_1$ and $\mathcal{T}_2 \oplus \mathcal{L}_2$. Deliver $\Pi_i(t)$ to $\mathcal{T}_i \oplus \mathcal{L}_i$, $i = 1, 2$. If $\Pi_1(t) = \Pi(t)$ and

$$\Pi_2(t) = \begin{pmatrix} \Pi_{\vartheta(1),1}(t), & \Pi_{\vartheta(1),2}(t), \ldots \\ \Pi_{\vartheta(2),1}(t), & \Pi_{\vartheta(2),2}(t), \ldots \\ \Pi_{\vartheta(3),1}(t), \ldots & \\ \vdots & \\ \Pi_{\vartheta(n),1}(t), \ldots & \\ \Pi_{n+1,1}(t), & \Pi_{n+1,2}(t), \ldots \end{pmatrix}$$

where ϑ is any permutation of $(1, 2, \ldots, n)$, then $\mathcal{IC}_1 \oplus \mathcal{L}_1 \equiv \mathcal{IC}_2 \oplus \mathcal{L}_2$ for all time. For the inputs have only been permuted among the copies of $\{\mathcal{T}_2(p^{(j)})\}$ and each of these is connected to $\mathcal{IC}_2$ in the same way.

Whenever we deliver inputs $\mathcal{J}$ to $\mathcal{T}$ and are concerned only with the line residues that are generated in $\mathcal{T} \oplus \mathcal{L}$, we can restrict attention to the effects of $\mathcal{J}$ on $\mathcal{IC} \oplus \mathcal{L}$, which is the locus of these residues. The insensitivity of $\mathcal{IC} \oplus \mathcal{L}$ to permutations ϑ of inputs delivered to the individual $\mathcal{T}(p^{(i)})$ means that we have more than one choice of inputs at our disposal which $\mathcal{IC} \oplus \mathcal{L}$ does not distinguish. Denoting the $\Pi_2(t)$ above by $\Pi_\vartheta(t)$, we therefore say that the input class $\Pi = \{\Pi_\vartheta(t)$: all $\vartheta\}$ is indistinguishable modulo $\mathcal{IC} \oplus \mathcal{L}$. The multiplicity of $\mathcal{T}(\oplus_i p^{(i)})$ with respect to Π is n!, since there are n! permutations of n objects.

The usefulness of a field structure which cannot distinguish every input becomes clear if to every $\mathcal{T}(p^{(i)})$ we associate another field $\mathcal{J}_i$ for which $\mathcal{L}(\mathcal{J}_i, \mathcal{T}(p^{(i)}))$ is not vacuous. Different excitations arising in disjoint and disconnected $\mathcal{J}_i$ at disparate times can be made to generate

the same $\mathcal{S}(\mathcal{G})$, while the various $\mathcal{S}(\mathcal{J}_i)$ differ among themselves in all other respects. Qualitatively similar situations arise frequently in behavior: A single vowel with fixed pronunciation appears in an enormous number of words. The vowel sound is an identical unit embedded in many otherwise different total units. Since vowels do make a multiple appearance in this way, a mechanism must exist whereby many different sources of excitation can all generate the evokation of the same vowel sound in a multiple crossing. The multiple sources of excitation in this simple example are the various $\mathcal{J}_i$.

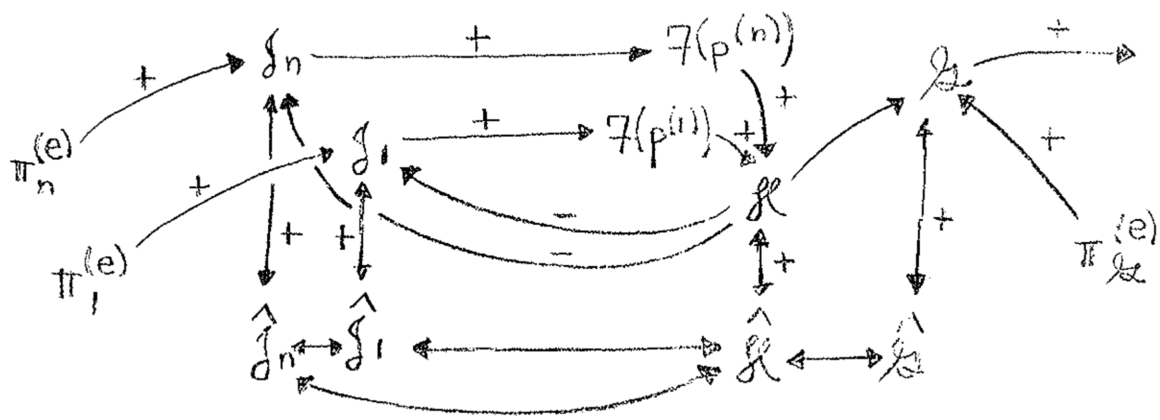
After a person learns to say a vowel sound and can use this sound in several words, it is still possible for him to learn new words in which the same vowel sound appears. The neural structures required to evoke this sound in one word cannot interfere with the growth of new structures which control the evokation of the sound in another word. This property is built into the present example by directing the line connections to flow in $\mathcal{J}_i \rightarrow \mathcal{T}(p^{(i)}) \rightarrow \mathcal{H}(p^{(1,2,\ldots,n)}) \rightarrow \mathcal{L}$ chains. Even after one $\mathcal{J}_i \oplus \mathcal{T}(p^{(i)})$ becomes capable of eliciting the $\mathcal{S}(\mathcal{G})$ corresponding to the evokation of the hypothetical vowel, another $\mathcal{J}_j \oplus \mathcal{T}(p^{(j)})$ can be constructed with the same property without disturbing the $\mathcal{J}_i \rightarrow \mathcal{T}(p^{(i)}) \rightarrow \mathcal{H} \rightarrow \mathcal{L}$ chain, since the fragment $\mathcal{J}_i \rightarrow \mathcal{T}(p^{(i)})$ need not participate in this construction in any way.

Two chains $\mathcal{J}_i \rightarrow \mathcal{T}(p^{(i)}) \rightarrow \mathcal{H} \rightarrow \mathcal{L}$, $i = 1, 2$, do not usually generate the same $\mathcal{S}$ trajectory. The problem is therefore well-posed in the sense of Hadamard. Nonetheless, the $\mathcal{S}$ trajectory of the restrictions of these chains to $\mathcal{H} \oplus \mathcal{L}$ can be made to agree by a simple shift of the time scale. The problem is therefore not locally well-posed. Such failures in locally well-posing a problem are characteristic of dynamical systems which exhibit hierarchically organized interactions.

If an entire string $\mathcal{J}_i \rightarrow \mathcal{T}(p^{(i)}) \rightarrow \mathcal{H} \rightarrow \mathcal{L}$ controls the evolution of a certain trajectory, then while $\mathcal{H}$ and $\mathcal{L}$ are still being excited by some $\mathcal{S}(\mathcal{J}_i)$, it is important that no $\mathcal{S}(\mathcal{J}_k)$, $k \neq i$, excitations simultaneously

arrive. The situation is directly analagous to the chain $\Omega_1 \rightarrow \Omega_2 \rightarrow \ldots \rightarrow \Omega_n$ which illustrated our remarks on the apparent time quantization seen by a macroscopic observer perched on Ω_1. An inhibitory mechanism must operate whenever $\mathcal{H}$ is excited to assure that various $\mathcal{F}(p^{(i)})$ do not induce overlapping excitations within $\mathcal{H} \oplus \mathcal{G}$. The $\mathcal{F}(p^{(i)})$ must not themselves be inhibited by this mechanism, since one of them is still transmitting important excitations to $\mathcal{H}$ when the inhibitory mechanism must operate. $\mathcal{H}$ must suppress some leading subset within each of the $\mathcal{P}(\mathcal{J}_i)$. If no $\mathcal{J}_i$ succeeds in exciting $\mathcal{H}$, this suppression is unnecessary. The excitation of $\mathcal{H}$ must direct inhibitory excitation to all $\mathcal{P}(\mathcal{J}_i)$ to the measure that $\mathcal{H}$ is excited. This can be accomplished by simply drawing inhibitory lines from $\mathcal{H}$ to every $\mathcal{P}(\mathcal{J}_i)$. The system $\{ \mathcal{J}_i \rightarrow \ldots \rightarrow \mathcal{G} : i = 1, 2, \ldots, n \}$ hereby closes itself off to further excitation of the various $\mathcal{P}(\mathcal{J}_i)$ until a complete dynamical cycle has evolved.

Since it is the line residues within $\mathcal{L}(\mathcal{H}, \mathcal{G})$ which are the most central interaction structure of the entire system, these lines must be preserved independently of the activation of any particular $\mathcal{J}_i \rightarrow \ldots \rightarrow \mathcal{G}$ chain. We must therefore augment the field by an $\hat{\mathcal{H}}$ that sends lines directly to $\mathcal{H}$ over which sustaining excitation for $\mathcal{C}(\mathcal{H}, \mathcal{G})$ passes. Each $\mathcal{J}_i$ must also possess a dual field $\hat{\mathcal{J}}_i$, and so must $\mathcal{G}$. The total picture is:



where (+) indicates an excitatory connection and (-) and inhibitory connection. It is a vast oversimplification of any actually occurring case. But it exhibits general features that will reoccur in many of our more complicated

and realistic examples. The most important fact to remember is that well-contracted and distinctive line distributions within $\mathcal{J}_1 \oplus \ldots \oplus \mathcal{J}_n$ and a locally ill-posed $\mathcal{H} \oplus \mathcal{G}$ can coexist in a single field; highly specific and different $\mathcal{L}(\mathcal{J}_i)$ all generate the same commonly shared $\mathcal{L}(\mathcal{H} \oplus \mathcal{G})$ without disrupting $C(\mathcal{H}, \mathcal{G})$.

80. Barriers and a One-Many Field

Consider a field of type

$$M = \begin{pmatrix} m_{11} & m_{12} & & & & \\ & m_{22} & m_{23} & & 0 & \\ & & m_{33} & m_{34} & & \\ & 0 & & \ddots & & \\ & & & & & m_{nn} \end{pmatrix}$$

with every $\mathcal{L}_k$ a homogeneous field. Suppose that a nontrivial homogeneous transmission threshold exists in every $\mathcal{L}_k$, that the transmission time from $\mathcal{L}_k$ to $\mathcal{L}_{k+1}$ is well-defined, and that $\mathcal{L}_k$ is a weighted set of antagonists for $\mathcal{L}_{k+1}$ with weighting vector $w^{(k)}$. The weighting $w^{(k)}$ suppresses all strength distributions from $\mathcal{L}_{k-1}$ which do not deviate from $w^{(k)}$. Consider $w^{(k)}$ as a vector in $\mathbb{R}^n$ and let $w^{(k)\perp}$ be the orthogonal complement of $w^{(k)}$. The strength vector $s^{(k)} = s^{(k)}(t)$ transmitted to $\mathcal{L}_{k+1}$ from $\mathcal{L}_k$ is a curve in $\mathbb{R}^n$ parametrized by t. The inhibition which $\mathcal{L}_k$ exerts on this strength vector during a small time interval $(t, t+\Delta t)$ is qualitatively similar to the action of the projection operator P_k of the strength vector $s^{(k)}(t)$ onto the hyperplane $w^{(k)\perp}$. The strength vector transmitted in $(t, t+\Delta t)$ from $\mathcal{L}_1 \to \mathcal{L}_2 \to \ldots \to \mathcal{L}_k$ to $\mathcal{L}_{k+1}$ is analogous to the vector in the hyperplane $w^{(1)\perp} \cap w^{(2)\perp} \cap \ldots \cap w^{(k)\perp}$ obtained by applying the composed operator $P_k \circ P_{k-1} \circ P_{k-2} \circ \ldots \circ P_1$. The various P_i do not

generally commute when applied to arbitrary strength vectors, and the analogy between projections and weighted sets of antagonists is not a strict one. But it correctly suggests that chains of weighted antagonists select the class of strength vectors which they will transmit by successively restricting this class to vectors that are "orthogonal" to every link in the chain.

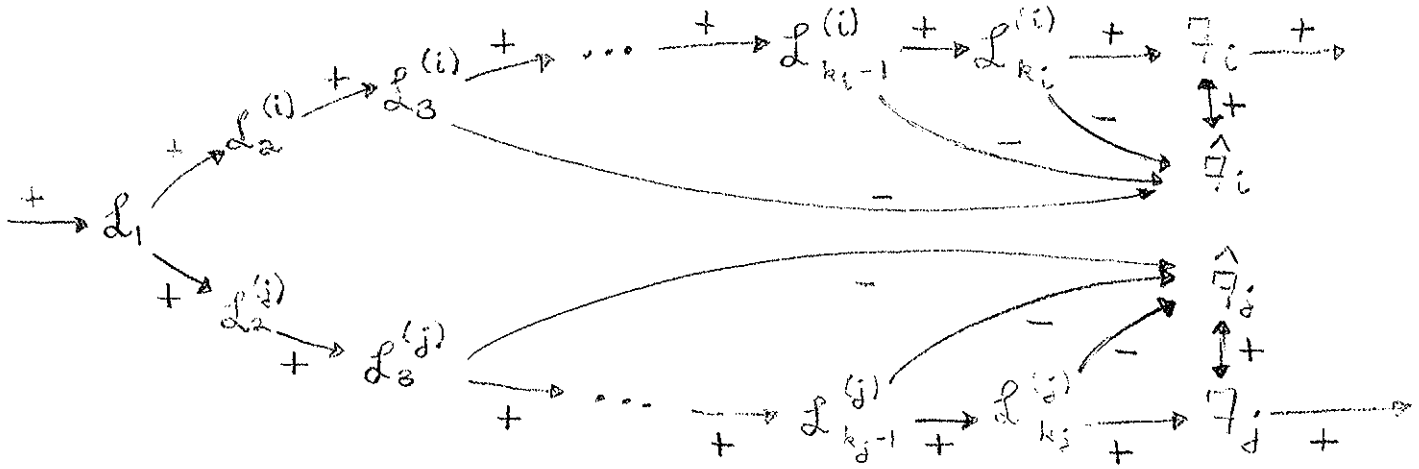
Even short sequences $w^{(1)}, w^{(2)}, \ldots, w^{(m)}$ of pairwise orthogonal $w^{(i)}$, the length m depending on the number of points in each $\mathcal{L}_k$, will transmit practically nothing. When the $w^{(i)}$ are chosen in this way, we say that M forms a barrier or natural boundary for any field to whose periphery it is attached. The use of barriers permits the close juxtaposition of many fields without disrupting the local dynamics of any of them. Barriers become very useful when several fields subserving differentiated and independent processes must be packed into a small region of space, as occurs in mammalian brains. Rows of inhibitory connections can also be used to help to make close packing possible.

Chains of weighted antagonists may also be used as sensitive filters of large sets of strength distributions. The fundamental units for this process are chains $\mathcal{L}_1^{(i)} \rightarrow \mathcal{L}_2^{(i)} \rightarrow \ldots \rightarrow \mathcal{L}_{k_i}^{(i)}$, $i = 1, 2, \ldots, N$, of weighted antagonists such that $\mathcal{L}_1^{(i)} = \mathcal{L}_1$ for all i, and all other components of different chains are disjoint and disconnected. $\mathcal{L}_1$ is the common strength source for this field of chains whose structure may conveniently be summarized by the matrix $W = \left\{ w_j^{(i)} \right\}$, $i = 1, 2, \ldots, N$, $j = 1, 2, \ldots, k_i$, where $w_j^{(i)}$ is the weighting vector for $\mathcal{L}_j^{(i)}$. When a chain for which $\left\{ w_j^{(i)}, j = 1, 2, \ldots, k_i \right\}$ spans a large subspace in $\mathbb{R}^n$ transmits at all, we can infer the general form of the strength vector which induced this transmission at $\mathcal{L}_1$. Qualitatively speaking, the chain maps a small class of strength distributions into 1 and the enormous class of remaining distributions into 0, where 1 designates that the chains's last link does transmit and 0 designates that it does not. The intensity of transmission from the last link gives information about the intensity of the source

distribution. An arbitrary distribution $(s_1(t), s_2(t), \ldots, s_n(t))$, $1 \ll n$, is hereby coded as the simplest distribution $(0, 1)$, with an added intensity parameter, in the end links of a set of weighted chains.

This reduction of a complicated distribution at $\mathcal{P}(\mathcal{L}_1)$ to a set of very simple distributions spread over $\mathcal{P}(\theta_i \mathcal{L}^{(i)}_{k_i})$ provides a convenient way to assure that only a restricted class of strength distributions ever generate a fixed strength distribution at another field locus. The links $\mathcal{L}^{(i)}_{k_i}$ can be attached to fields $\mathcal{7}_i$ whose line structure branches out from $\mathcal{L}^{(i)}_{k_i}$ to generate a complicated strength distribution whenever $\mathcal{L}^{(i)}_{k_i}$ is activated, and the intensity with which this strength distribution is actualized varies directly with the intensity of $\mathcal{L}^{(i)}_{k_i}$ excitation. The previous example illustrated one way in which many distinctive sources can control a single distribution. This example shows a way in which a single source can control many distinctive distributions.

The line structures in the various $\mathcal{7}_i$ must be sustained by dual fields $\hat{\mathcal{7}}_i$. $\hat{\mathcal{7}}_i$ must cease to interact with $\mathcal{7}_i$ whenever the last links of $\mathcal{L}^{(i)}_1 \rightarrow \ldots \rightarrow \mathcal{L}^{(i)}_{k_i}$ are activated, for otherwise $\mathcal{L}(\mathcal{L}^{(i)}_{k_i}, \mathcal{7}_i)$ and $\mathcal{L}(\hat{\mathcal{7}}_i, \mathcal{7}_i)$ transmissions will obscure each other. Inhibitory lines from $\mathcal{L}^{(i)}_1 \rightarrow \ldots \rightarrow \mathcal{L}^{(i)}_{k_i}$ to $\hat{\mathcal{7}}_i$ must therefore be drawn with the last links given the greatest weight since not all activations of an intermediate link excite $\mathcal{L}^{(i)}_{k_i}$. The suppression of $\hat{\mathcal{7}}_i$ activity by $\mathcal{L}^{(i)}_1 \rightarrow \ldots \rightarrow \mathcal{L}^{(i)}_{k_i}$ can be made to occur gradually by using an ordered set of inhibitory lines which are successively excited and which send steadily increasing inhibitory line densities to $\hat{\mathcal{7}}_i$. We have the diagram



which closely resembles the diagram of the last example, but with the "$\downarrow$" arrows reversed. The situation is reminiscent of the classification of diagrams as "projection" and "injective" in algebraic topology.

The previous examples illustrate some important embedding principles in simple settings. They show that delicate analysing and synthesizing properties can be introduced into embedding fields in natural ways. In particular, we have seen that these fields exhibit analogs of projection operators, orthocomplementation, field immersion, excision of subfields, boundaries, functional operators, gradients and curvature measures, transitions between states, interaction densities, and similar concepts derived from such classical theories as the theory of Hilbert space, the theory of manifolds, and the theory of Markov processes. These analogs develop as time-bound processes in the embedding field situation. In all of the examples, some analog of a sustaining dual field $\hat{\mathcal{H}}$ for fields $\mathcal{H}$ has arisen. We now turn to a closer study of some of the properties which these $\hat{\mathcal{H}}$ must possess.

81. Stationary $\mathcal{C}$ Fields and Field Extensions

This study revolves about the following question: Given a field $\mathcal{H}$ and a certain $\mathcal{C}(\mathcal{H})$, can we find a disjoint field $\hat{\mathcal{H}}$ which maximally preserves $\mathcal{C}(\mathcal{H})$ in some natural sense? Before constructing $\hat{\mathcal{H}}$, we must first decide what the inputs from $\hat{\mathcal{H}}$ to $\mathcal{H}$ must look like in order to preserve a given $\mathcal{C}(\mathcal{H})$. We must then search for a $\hat{\mathcal{H}}$ which can induce these inputs. If $\mathcal{C}(\mathcal{H})$ is very simple, the requirements which it poses on $\hat{\mathcal{H}}$ for its sustanence will be small and might not uniquely determine $\hat{\mathcal{H}}$. If we imagine two possible $\mathcal{C}(\mathcal{H})$ for $\mathcal{H}$, say $\mathcal{C}_1(\mathcal{H})$ and $\mathcal{C}_2(\mathcal{H})$, each $\mathcal{C}_i(\mathcal{H})$ will require a certain sustaining input distribution from $\hat{\mathcal{H}}$. It is harder to find a single $\hat{\mathcal{H}}$ which can sustain both line distributions than it is to find an $\mathcal{H}$ which will sustain just one of them. It is harder, still, to find a single $\mathcal{H}$ to sustain each of $\mathcal{C}_i(\mathcal{H})$, $i=1, 2, \ldots, n$, $1<<n$. By considering an enormous class of possible $\mathcal{C}(\mathcal{H})$ we can hope to find a

unique $\hat{\mathcal{H}}$ for $\mathcal{H}$.

The various $C(\mathcal{H})$ which can be induced in $\mathcal{H}$ depend, in turn, not only on the internal structure of $\mathcal{H}$ but also on the distribution of lines to $\mathcal{H}$ and the external inputs which are allowed to reach $\mathcal{H}$ over them. The study of fixed $C(\mathcal{H})$ over $\mathcal{H}$ is seen in this way to be a tool for determining the fields interacting with $\mathcal{H}$ by looking at $\mathcal{H}$ alone: A condition locally definable on $\mathcal{H}$ gives a method for extending $\mathcal{H}$ to the fields with which it interacts, as we naturally expect in any dynamical system that exhibits nonlinear binding on a global level. Each $C(\mathcal{H})$ is viewed as a kind of stationary, or asymptotic, state of $\mathcal{H}$, and the entire collection of fields interacting with $\mathcal{H}$ is studied with the following question in mind: What collection of fields, or set of collections of fields, can generate and sustain a fixed collection of stationary line residues within a given $\mathcal{H}$? Only one such residue actually resides in $\mathcal{H}$ at any time, so that the question has the character: Given a collection of possible line residues C over $\mathcal{H}$, what kind of field connections to and from $\mathcal{H}$ are necessary to produce an $\mathcal{H}$ whose capacity, or maximal collection of $C(\mathcal{H})$, is precisely C ? A complete answer to this question, for arbitrary $\mathcal{H}$, will be a large step in the direction of understanding stable nonlinear systems. Its intuitive meaning is: Although a given $\mathcal{H}$ cannot predict its eventual experience with its environment—the external inputs which it eventually receives—it is important to know those environments to which $\mathcal{H}$ can respond and adapt. Without such asymptotic information, no amount of studying evolutionary development can give full insight into the resultant organism.

We will begin with intuitive observations about several $C(\mathcal{H})$ which arise frequently in simple learning situations. We will also occasionally state general principles in a suggestive language that commends to their truth before actually giving formal interpretations of them. The formal interpretation will alter its specific form in different settings, but the intuitive principle will remain universally valid. To arrive at the first

few general principles of this type, we make the following simple observations.

82. Distribution of Internal Inputs

When an organism learns something new, it is clear that in some sense the organism's goal is to preserve the learned material as well as possible, and that, for higher mammals at least, an extraordinary degree of perfection in the process of retaining learned materials has already been achieved through evolution. In general, such a proposition substitutes mere teleology for insight, since one must first have specific quantities in mind before one can proceed to preserve them. In an embedding field, however, the quantities to be sustained in memory preservation are unambiguous, for the line functions c_{ij} wholly contain the residual record of past experiences and guide the evolution of transient strength fields in revisiting these experiences. The problem of preserving learned materials is translated into the problem of finding suitable conditions that express the preservation of the line structure. This problem may be elaborated into the following General Principle: In the absence of external inputs to an embedding field, the distribution and form of internal inputs will be such as to maximize the preservation of the extent line structure, subject to constraints which are intrinsically related to the field geometry. This principle can be extended to the case for which external inputs are delivered:

General Principle: In the presence of external inputs, the distribution and form of internal inputs maximizes both (1) the accurate embedding of the experiential record represented by these inputs, and (2) the preservation of old embedded materials that are "in harmony" with the new external inputs, again subject to geometrical constraints. One thus imagines that the process of memory development has so evolved that it proceeds according to equilibrium principles that maximally sustain the record of the old and bring this record into accord with the pressures of the new wherever

possible. We first study these principles in a homogeneous field $\mathcal{F}$ of serial type with simple line structure. One possible expression of our principle is that the c_{ij} functions remain constant over certain time intervals in the absence of external inputs. For such a field in the absence of external inputs, the condition that the c_{ij} functions remain constant, $dc_{ij}/dt = 0$, implies

$$\gamma^{+}_{ij} + (A_{ij} - c_{ij})\, s_i(t-t_{ij})\, s_j = \gamma^{-}_{ij}\, c_{ij}\, (M_i - s_i(t-t_{ij}))\, (M_j - s_j),$$

and

$$c_{ij} = \frac{A_{ij}}{1 + \dfrac{\gamma^{-}_{ij}}{\gamma^{+}_{ij}} \left(\dfrac{M_i - s_i(t-t_{ij})}{s_i(t-t_{ij})} \right) \left(\dfrac{M_j - s_j}{s_j} \right)}$$

When $i = j$,

$$c_{ii} = \frac{A_{ii}}{1 + \dfrac{\gamma^{-}_{ii}}{\gamma^{+}_{ii}} \cdot \left(\dfrac{M_i - s_i}{s_i} \right)^2}$$

so that whenever $A_{ii} \neq 0$, $dc_{ii}/dt = 0$ implies that $ds_i/dt = 0$, for all i. $dc_{ii}/dt = 0$ is too strong a requirement in this case unless we are prepared to conclude that every perturbation of the field essentially changes its line structure. That the distributions $\{ c_{ij} : j = 1, 2, \ldots, n \}$ preserve their relative magnitudes even while the individual c_{ij} vary is the next strongest natural possibility: $dc^{*}_{ij}/dt = 0$. A possibility of this type gains intuitive appeal from that fact that $s_i(t) = 0$, $t \in [0, T]$ for all i, implies $dc_{ij}/dt = -\epsilon M^2 c_{ij}$, or $c_{ij}(t) = c_{ij}(0)\, e^{-\epsilon M^2 t}$, whence $c^{*}_{ij}(t) = c^{*}_{ij}(0)$ and $dc^{*}_{ij}(t)/dt = 0$, $t \in [0, T)$, even though $c_{ij}(t) \neq c_{ij}(0)$ so that $\dfrac{dc_{ij}}{dt} \neq 0$.

In the absence of external inputs, the relative magnitudes of $\{c_{ij}: j = 1, 2, \ldots, n\}$ are indeed preserved. Thus, after a period of strength quiescence, an isolated input to p_i in a field for which the $c = c^*$ approximation is valid will generate the same strength transmissions as an isolated input delivered to p_i before the period of strength quiescence began. The strength fields induced in this way before and after the quiescent, input-free period will be much the same. Since all effects of $\mathcal{F}$ on other fields are induced by $\mathcal{S}(\mathcal{F})$, and since the above $\mathcal{S}(\mathcal{F})$ is invariant in first approximation under changes in $\mathcal{C}(\mathcal{F})$ which preserve $dc^*_{ij}/dt = 0$, $\frac{dc^*_{ij}}{dt} = 0$ is a useful measure with which to begin the study of $\mathcal{C}(\mathcal{F})$ preservation. $dc^*_{ij}/dt = 0$ is, however, only a local condition focalized at p_i. If $s_i \equiv 0$ for all $p_i \in \mathcal{P}(\mathcal{F})$ after a certain $\mathcal{C}(\mathcal{F})$ is introduced, then continual activation of other fields $\mathcal{F}'$ can upset the global $\mathcal{C}(\mathcal{F} \oplus \mathcal{F}')$ distribution, since all $\mathcal{C}(\mathcal{F})$ will decay to zero while the $\mathcal{C}(\mathcal{F}')$ will not. It is therefore important to consider the effects of nonzero internal input arrays to $\mathcal{F}$ which help to keep the absolute c_{ij} values from decaying to zero and which also do not strongly violate $dc^*_{ij}/dt = 0$.

Let $dc^*_{ij}/dt = 0$ for all i and j. Then $(dc_{ij}/dt)c_i - (dc_i/dt)c_{ij} = 0$, where $c_i = \Sigma_k c_{ik}$, so that $d(\log c_{ij})/dt = (dc_{ij}/dt)/c_{ij} = (dc_i/dt)/c_i$. In particular, $d(\log c_{ij})/dt$ is independent of j, whence $d(\log c_{ij})/dt = d(\log c_{ik})/dt$, for all i, j, and k, which is the same as $d(\log(c_{ij}/c_{ik}))/dt = 0$ just so long as the c_{ik} are not equal to zero, and this is always true in a minimal field. To fulfill our principle, we should thus at least like to insure that the functions $d(\log(c_{ij}/c_{ik}))/dt$ are as small in absolute value as possible. We express this fact by introducing the functions

$$G_i = \sum_{j<k} (d\log(c_{ij}/c_{ik})/dt)^2$$

and

$$G = \Sigma_i G_i$$

and interpreting the General Principle as:

The distribution and form of internal inputs, in the absence of external inputs to a homogeneous $\mathcal{T}$, minimizes G.

The construction of G by summing over the G_i emphasizes that all points in the field must be treated on an equal footing in a homogeneous $\mathcal{T}$. Any symmetric function of the G_i would have accomplished this, but the summation of individual G_i most simply emphasizes the distinctive role played by the single point p_i to which an isolated input is delivered when questions of field renormalizability are considered. We now apply the principle to particular $C(\mathcal{T})$'s.

Let $\mathcal{P}(\mathcal{T}) = \{p_1, p_2\}$ with

$$\dot{s}_1 = \alpha (M-s_1)(r s_1 c_{11} + I_1) - \beta s_1,$$

$$\dot{s}_2 = \alpha (M-s_2)(r s_2 c_{22} + r s_1(t-v) c_{12} + I_2) - \beta s_2,$$

$$\dot{c}_{12} = u r \hat{r} (A_{12}-c_{12}) s_1(t-v) s_2 - \epsilon \rho \hat{\rho} c_{12} (M-s_1(t-v))(M-s_2),$$

$$\dot{c}_{22} = u r \hat{r} (A_{22}-c_{22}) s_2^2 - \epsilon \rho \hat{\rho} c_{22} (M-s_2)^2,$$

etc. All $p_{ij} = 1$ and p_1 receives inputs only from itself and from internal input sources. Let $s_k(0) > 0$ for all k, and let $c_{ij}(0) > 0$ unless i=2 and j=1. Consider the following local analog of the condition G = 0:

$$d(c_{12}/(c_{12} + c_{22}))/dt = 0,$$

which is equivalent to $(dc_{12}/dt)/c_{12} = (dc_{22}/dt)/c_{22}$ and $(d(\log(c_{12}/c_{22}))/dt)^2 = 0$. An intuitive interpretation of this condition is the following: Both c_{12} and c_{22} modulate the strength function s_2 and live over nodes which impinge upon p_2. We have required that the proportional growth of one of these quantities be the same as that of the other. This demand can only be realized if, in some way, local s_2

activity strongly determines not only the behavior of c_{22} --which is always the case-- but also that of c_{12}, which depends on the state of s_1 as well. The flow of strength over p_1 and p_2 must therefore be so uniform, in some sense, that s_1 and s_2 are indistinguishable to an observer perched on p_2. To give rigor to these intuitive remarks, note that

$$\frac{\dot{c}_{12}}{c_{12}} = \frac{\dot{c}_{22}}{c_{22}}$$

implies, writing $\gamma_{ij}^{+} = \gamma+$ and $\gamma_{ij}^{-} = \gamma-$, that

$$\gamma^{+} \cdot \left[\left(\frac{A_{12}}{c_{12}} - 1\right) s_1(t-v) - \left(\frac{A_{22}}{c_{22}} - 1\right) \cdot s_2\right] \cdot s_2 = \gamma^{-}(M-s_2)(s_2 - s_1(t-v)),$$

$$\gamma^{+} \cdot \left[\frac{A_{12}}{c_{12}} s_1(t-v) - \frac{A_{22}}{c_{22}} s_2\right] \cdot s_2 = \left[s_1(t-v) - s_2\right]\left[\gamma^{+} s_2 - \epsilon(M-s_2)\right].$$

$s_2(0) > 0$ implies $s_2(t) > 0$, for all $t > 0$. Hence if $s_2(t) = s_1(t-v)$, then $c_{12}/A_{12} = c_{22}/A_{22}$. Conversely, if $c_{12}/A_{12} = c_{22}/A_{22}$, then

$$\gamma^{+} \frac{A_{22}}{c_{22}} s_2\left[s_1(t-v) - s_2\right] = \left[s_1(t-v) - s_2\right] \cdot \left[\gamma^{+} s_2 - \epsilon(M-s_2)\right]$$

Now suppose that $s_2(t) \neq s_1(t-v)$. Then

$$\gamma^{+}\left(\frac{A_{22}}{c_{22}}\right) s_2 = \gamma^{+} s_2 - \epsilon(M-s_2),$$

or

$$\gamma^{+} s_2 \left(1 - \frac{A_{22}}{c_{22}}\right) = \epsilon(M-s_2)$$

But $(M-s_2) > 0$ while $1 - (A_{22}/c_{22}) < 0$, so that

$$0 > \gamma^{+} s_2 \left(1 - \frac{A_{22}}{c_{22}}\right) = \epsilon(M - s_2) > 0$$

which is a contradiction. It follows that $c_{12}/A_{12} = c_{22}/A_{22}$ if and only if $s_2(t) = s_1(t-v)$. Since $c_{12}/\theta h_{12} = c_{12}/A_{12} = c_{22}/A_{22} = c_{22}/\theta h_{22}$, $c_{12}/h_{12} = c_{22}/h_{22}$ if and only if $s_2(t) = s_1(t-v)$, where c_{ij}/h_{ij} is the excitation density of N_{ij}. Whenever $s_{i+1}(t) \equiv s_i(t-v)$, we say that the strength flow along $p_i \longrightarrow p_{i+1}$ is translation stable with period v. We have proved the following

Theorem: Given a two point field governed by equations (A) and nontrivial initial conditions, under the stability hypothesis $\frac{d}{dt}\left(\frac{c_{12}}{c_{12}+c_{22}}\right) = 0$, the strength flow from p_1 to p_2 is translation stable with period v if and only if the excitation densities of N_{12} and N_{22} are equal.

Also notice that if the nodal excitation densities are not equal and the flow is still translation stable, then s_1 and s_2 are identically zero.

This theorem makes precise both the sense in which the growth of c_{12} and c_{22} are locally bound (equal excitation densities) and the nature of the uniformity of the flow (translation stability). The existence of equal excitation densities in this situation under a translation stable flow is intuitively appealing, since each excitation of s_2 induced by p_1 will generate an internal input to p_2 which can adjust itself to tightly bind the $s_1(t-v)$ and s_2 functions in the absence of other external sources of excitation.

These simple considerations have led us to a remarkable phenomenon. For we have found a dynamical system in which a wave motion is necessary and sufficient to sustain the homogeneous interaction structure of the system!

83. Brain Rhythms and Internal Inputs

This simple example also provides a glimpse of the neurological solution to the problem of sustaining line distributions on a global scale. Some sustaining excitation of embedded line structures must be provided, or else they will decay to zero in uncontrollable ways. If this excitation is delivered with uniform intensity

to all points, a chaotic disorganization of lines will ensue. Thus, for relatively brief periods of time, the strength fields in certain embedding regions must be allowed to decay. During these periods, the c^{*}_{ij} values in these regions will remain quite stable, and since the periods are brief, total c_{ij} decay will not be great. Also the quiescent cells and their auxiliary structures have an opportunity to refurbish their metabolic stores. While the strength distributions of certain regions are quiescent, other regions are receiving sustaining inputs which, in configuring themselves to help preserve desirable patterns in the lines, also often display interesting rhythmic behavior. The actual determination of the path of this excitation process will depend on familiar renormalization properties, and we shall find winding and sometimes self-intersecting curves and globules of excitation in the space of embedded forms. The sustaining motion will often pass through portions of embedded trajectories, which we here represent as single points or small clusters of points. Each such trajectory can encompass a broadly distributed collection of cells in the universal space at any time. Moreover, a single renormalizing point like a p_w directly interacts with large field structures; cf. , the thalamus. A measuring device of electrical volume potentials, say on the scalp, will usually be too localized to pick up any traces of translation stability in the space of embedded forms or between broadly interacting renormalization structures. Such an instrument does not measure motions in the space of forms, but rather measures excitation changes in certain cell groups that are localized in Euclidean norm. Translation stability in the space of forms often appears to such an instrument as ordinary periodicity. Rhythms induced by sustaining activity will appear in such instruments, and rhythms appear inthe Electroencephalogram.

84. Normalization Sets. Lines and Loops

In the previous example, c_{12} was normalized by $c_{12}+c_{22}$. We call $\{c_{12}, c_{22}\}$ the normalization set of the field. The various normalization sets for any field $\mathcal{F}$ give a local description of the interaction of $\mathcal{F}$ with its dual $\hat{\mathcal{F}}$. Intuitively, if $i(N)$ is the set of indices in a normalization set N for $\mathcal{F}$ and $i(N_1) \oplus i(N_2) \oplus i(N_3) \oplus \dots \oplus i(N_k)$ is a partition of the indices of $\mathcal{P}(\mathcal{F})$, then $\hat{\mathcal{F}}$ may

be decomposed into k subfields $\hat{\mathcal{F}}_j$, $j = 1, 2, \ldots k$, such that each $\hat{\mathcal{F}}_j$ interacts most strongly with $\{p_i: i \in i(N_j)\}$ and the various $\hat{\mathcal{F}}_j$ are not completely coupled to one another.

A useful extension of the above two-point field to an infinite field $\{p_i: i = \ldots, -1, 0, 1, 2, \ldots\}$ with infinitely many normalization sets is given by letting $p_{ij} = \delta_{i+1,j} + \delta_{ij}$, $d(c_{k,k+1}/(c_{k,k+1} + c_{k+1,k+1}))/dt = 0$, $k = 0, \pm 1, \pm 2, \ldots$, and determining the A_{ij}'s and M_k's in a homogeneous way ($A_{ij} = A(1-\delta_{ij}) + B\delta_{ij}$). We assume that the sustaining input regime has begun so long ago that all initial perturbations have completely faded and that equal excitation densities have been achieved at each point separately. What can be said about the internal input I_i to p_i?

By translation symmetry of the M, A, and p functions, and the translation stability of the s_j ~~which is seen directly from the two-point case by an obvious chaining argument~~ it follows that the I_i are translation stable as well: $I_i(t) = I_{i-1}(t-h)$, for some $h > 0$. Also $h = v$, for otherwise a lag or lead between input arrivals and strength transmission would develop, thus destroying translation stability with period v in the s_i's.

The various c_{ij} are also translation stable with period v; for example $c_{i,i-1}(t) = c_{i-1,i-2}(t-v)$. For by translation stability,

$$\dot{s}_i = \alpha(M-s_i)(r\, s_i c_{ii} + r\, s_{i-1}(t-v)\, c_{i-1,i} + I_i) - \beta s_i$$

$$= \alpha(M-s_i)\left[r\, s_i(c_{i-1,i} + c_{ii}) + I_i \right] - \beta s_i$$

so that

$$\dot{s}_{i+1} = \alpha(M-s_{i+1})\left[r\, s_{i+1}(c_{i-1,i}(t-v) + c_{ii}(t-v)) + I_{i+1} \right] - \beta s_{i+1}$$

But we know also that

$$\dot{s}_{i+1} = \alpha(M-s_{i+1})\left[r\, s_{i+1}(c_{i,i+1} + c_{i+1,i+1}) + I_{i+1} \right] - \beta s_{i+1}.$$

Equating the two expressions for $\dot{s}_{i+1}$ and recalling that $0 < s_k < M$ for all k and t, it follows that

$$c_{i-1,i}(t-v) + c_{ii}(t-v) = c_{i,i+1}(t) + c_{i+1,i+1}(t) \qquad (\alpha)$$

By equal excitation densities,

$$\frac{c_{i-1,i}}{A} = \frac{c_{ii}}{B}$$

and since (α) obviously implies

$$\frac{c_{i-1,i}(t-v) + c_{ii}(t-v)}{A} = \frac{c_{i,i+1} + c_{i+1,i+1}}{A},$$

$$\frac{c_{ii}(t-v)}{B} + \frac{c_{ii}(t-v)}{A} = \frac{c_{i+1,i+1}}{B} + \frac{c_{i+1,i+1}}{A},$$

$$c_{ii}(t-v)\left[\frac{1}{B} + \frac{1}{A}\right] = c_{i+1,i+1}\left[\frac{1}{B} + \frac{1}{A}\right]$$

and

$$c_{ii}(t-v) = c_{i+1,i+1}(t) \qquad (\beta)$$

Applying (β) to (α) gives

$$c_{i-1,i}(t-v) = c_{i,i+1}(t) \qquad (\gamma)$$

and (β) and (γ) together are what we set out to prove. The entire dynamical situation is translation stable with period v. Internal input pulses are thus emitted from $p_{-\infty}$ at a regular rate. The $c_{i-1,i}$ and c_{ii} at a fixed p_1 wax and wane with the arrival and passing of these pulses but the normalization condition is always preserved.

A similar situation occurs for $p_{ij} = \delta_{ij(\mathrm{mod}\ n)} + \delta_{i+1,j(\mathrm{mod}\ n)}$; or for

$p_{ij} = \delta_{i+1, j(\text{mod } n)}$; that is, for a loop. Here the internal input pulses run around the loop at a fixed rate. When viewed by an observer perched on a single point, the local dynamics are identical in both the infinite and the looped field. The looped field possesses the same transmission structure as a serial field which has reached the cyclic asymptote that one expects when intratrial and intertrial intervals are identical. When the intertrial interval exceeds the intratrial interval and the linear asymptote has been reached, one source of regular internal input pulses is the dual point of p_1, and these pulses travel at a fixed rate to p_n, where they fade. If the strength field is quiescent, an internal input pulse can be delivered to any p_i and then travels to p_n where it fades. When a pulse is approaching a certain p_i, however, it blocks the emergence of spontaneous pulses to p_i until it has passed over p_i in order to insure that the normalization condition is not violated. These properties are of the same type as the conditions which we used to construct dual fields for the serial field. Renormalizations during learning and during sustaining activity obey the same principles.

85. A Rigidity-Plasticity Continuum. Channels

The loop field and the cyclic asymptote for serial learning with equal intratrial and intertrial intervals do require similar internal inputs but do not require the same dual field. For it is much harder to reach the cyclic asymptote in a homogeneous $\mathcal{F}$ and then to sustain it than it is to simply propagate pulses in a loop. A loop can be attached to almost any dual and still retain the general form of its line distribution, whereas a serial field manifestly cannot.

This difference between loops and cyclic asymptotes is a general one. Certain fields, like a loop $\mathcal{K}_1$, admit a wide variety of dual fields but are capable of sustaining only a limited number of $C(\mathcal{K}_1)$. Other fields, like a serial field $\mathcal{K}_2$, place more stringent demands on candidates for $\hat{\mathcal{K}}_2$, but the realization of these demands is compensated by the fact that $\mathcal{K}_2$ can sustain a much larger variety of $C(\mathcal{K}_2)$ then $\mathcal{K}_1$ can. $\mathcal{K}_1$ is a <u>rigid</u> field in the sense that it can sustain only very

few $C(\mathcal{H}_1)$, while $\mathcal{H}_2$ is a plastic field. The more stringent demands placed on the $\hat{\mathcal{H}}_2$ for a plastic $\mathcal{H}_2$ are aimed at preventing the flexibility of $C(\mathcal{H}_2)$ from degenerating into a mere chaos of transitions from one $C(\mathcal{H}_2)$ to another.

It is not correct to assume that a given $\mathcal{H}$ may generally be characterized by a fixed degree of rigidity. This is because the p_{ij} functions are not always strictly constant in time. Rather, they are functions which vary very slowly compared to the c_{ij} functions. The c_{ij} functions grow large when p_i and p_j are both activated at overlapping times. They provide a kind of nonlinear average of recent simultaneous activity of this kind. The p_{ij} functions provide a similar kind of average. If a given l_{ij} connection is frequently activated over a long period of time, the p_{ij} functions show a gradual net increase, subject to appropriate geometrical constraints. On the other hand, if over a long period l_{ij} remains inactive, p_{ij} will slowly decrease. Although c_{ij} and p_{ij} both provide averages of l_{ij} activity, p_{ij} is influenced by events occurring over a much longer time interval than is c_{ij}.

If a small number of p_{ij}, and only these, are activated over a long period of time, all other p_{ij} will gradually decrease, and the field will slowly become more rigid than it originally was. This increase in rigidity is permitted under the expectation, gleaned from many past events, that the unused p_{ij} will be superfluous in the future experience of the organism. The advantage of removing unnecessary lines is that it will be simpler in the future for the field's dual to preserve the important line residues. By lessening the task of the dual in this way, it becomes possible to pack more line residues into nearby fields which also depend for their preservation on the same dual, without endangering the old and important residues. From this follows the interesting observation that although plastic $\mathcal{H}_2$'s can sustain many more possible kinds of $C(\mathcal{H}_2)$ than rigid $\mathcal{H}_1$'s can, by gradually rigidifying a plastic $\mathcal{H}_2$ on the basis of its past experience, we can increase the capacity of $\mathcal{H}_2$ for sustaining many closely packed $C(\mathcal{H}_2)$ simultaneously. Put in another and more familiar terminology, this rigidification process gradually transforms a homogeneous embedding surface into a large collection of very reliable channels.

86. c* Invariance and "On"-"Off" Regions

One form of translation stability with period v is ordinary periodicity with period v. If $s_k(t) = s_k(t-v)$ for all k in a structural line (or loop) such as $p_{ij} = \delta_{j,i+1}$ (or $p_{ij} = \delta_{j,i+1(\mathrm{mod}\ n)}$), the linear (loop) structure is not quickly destroyed. In a homogeneous field whose c* structure closely approximates a line (or loop) asymptote, however, choosing between a weak type of translation stability such as $s_{i+1}(t) = s_i(t-v)$ and periodicity involves enormous differences in c*-sustanence. To see this, recall that nonzero sustaining internal inputs are necessary to preserve the global distribution of c weights. We wish to find that internal input array that best preserves c* residues and global c weights. We let

$$U = \sum_{m=-\infty}^{\infty} \int_{mv}^{(m+1)v} s_m(t)dt > 0$$

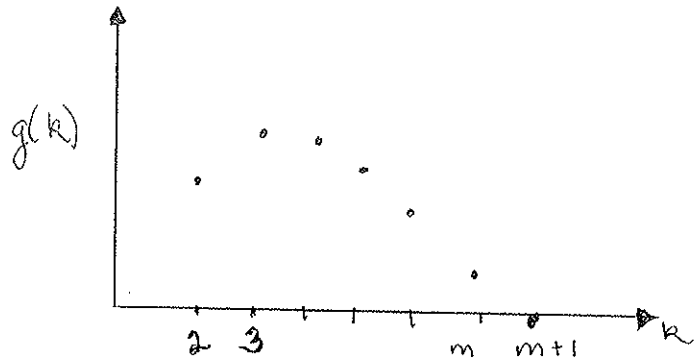
be fixed throughout our search for such a maximally effective sustaining input array, or arrays.

If $s_{i+1}(t) = s_i(t-v) = s_i(t)$ for all i, then there exist relatively long time intervals during which p_i-induced transmissions will arrive at several simultaneously active points, and $c^*_{i,i+1}$ will fluctuate away from the $c^*_{i,i+1} = 1$ asymptote. In order to avoid this difficulty, the strength field must be arranged so that the only point receiving actively transmitted excitation from p_i at any time is p_{i+1}. This condition cannot strictly be realized if the transmission threshold is zero in a purely excitatory field, since once $s_i(t_o) > 0$, $s_i(t) > 0$ for all $t \geq t_o$. Nonetheless, we can still assure that $s_i|_{[t_i+v,\infty)}$ takes very small values, and in fact that most of the mass of $\int_{t_i}^{\infty} s_i(t)dt$ is concentrated in $[t_i, t_i+v)$. Our success in achieving this concentration depends on U. Supposing that most of the mass of s_i is concentrated in an interval I_i of length less than v, it is clear that deviation from $c^*_{i,i+1}$ can be made very small if $I_i \cap I_j = \phi$ whenever $i \neq j$. By the translation symmetry of the $c^*_{i,i+1} = 1$ condition and the requirement that p_i directly excite p_{i+1}, a physically natural solution to the problem of sustaining c* values is thus to regularly space $I_i, I_{i+1}, I_{i+2}, \ldots$ in such a way that

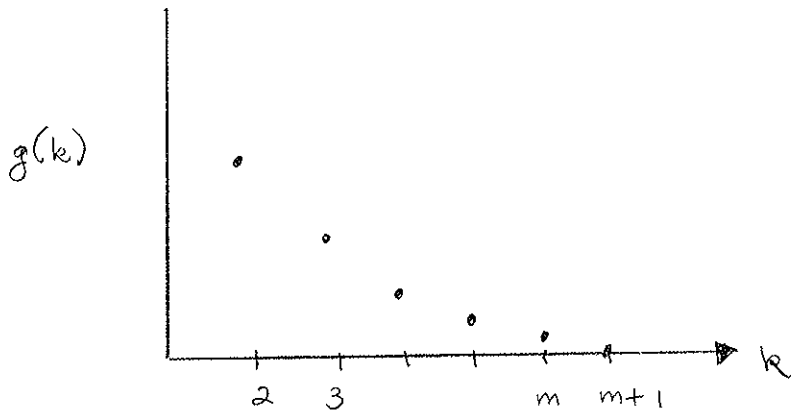
each s_i is generated by an internal input pulse that is translation stable with period v.

One way to improve the line-sustaining property still further is to introduce transmission thresholds $\Upsilon > 0$. If we do this, it is no longer necessary that $s_i\big|_{[t_i+v,\infty)} \equiv 0$ to completely preserve c^* values. All that is required is that $s_i\big|_{[t_i+v,\infty)} < \Upsilon$. By spacing the I_i pairwise disjointly, we can arrange that only one point actively transmits at any time, and once again a translation stable input pulse over chains of points is suggested by symmetry conditions.

A further improvement can be achieved by introducing inhibitory lines from p_i to p_k, where $|k-i| \geq 2$ and the inhibitory p_{ij} line densities leading from p_i take large values only for a few $p_{i+2}, p_{i+3}, \ldots, p_{i+k}$. Indeed, $g(k) = p_{i+k}$ is chosen so that we have a graph either of the form



or of the form



The introduction of such topographically-ordered local inhibitory lines enables p_i to actively suppress the s_{i+k}'s with $g(k) > 0$ after s_i is activated by excitatory internal inputs. This suppression helps to contract the set of all simultaneously transmitting points to the set $\{p_{i-1}, p_i, p_{i+1}\}$. This contraction simplifies the

task of preserving c* values. Moreover, when s_i is large and is likely to generate c* deformations, the lateral inhibitions from p_i to $\{p_{i\pm k}: g(k) > 0\}$ will also increase and will suppress the marginal $p_{i\pm k}$'s more strongly than when s_i is small and unlikely to generate c* deformations. U can thus be increased without seriously enlarging the point concentration sets generated by a translation stable input pulse. The introduction of inhibitory lines maintains a sharply localized excitatory distribution even when the intensity of excitation of individual points is markedly increased.

87. Virtual Self-Inputs

Introducting inhibitory lines also introduces restrictions on the time interval w between onsets of the input pulse at any point p_i and its successor point p_{i+1}. To see this, let the transmission thresholds initially be zero for simplicity, and let $\bar{t}_{ij}$ be the time necessary for p_i to transmit inhibitory strength to p_j. By the homogeneity of the initial field, we let $\bar{T} = \bar{t}_{i,i+1} = \bar{t}_{i+1,i}$. We also let $\bar{t}_{i,i\pm n} = n\bar{T}$ for specificity. We must restrict w so that the input pulse does not arrive at p_{i+2} at the same time that input-induced inhibitory transmission from p_i arrives at p_{i+2}. The jointly arriving excitatory and inhibitory input components would cancel one another in a most inefficient manner. Thus $\bar{t}_{i,i+2} \neq 2w$, and indeed $\bar{t}_{i,i\pm k} \neq kw$, $g(k) > 0$. Equivalently, $\bar{T} \neq w$.

Suppose that $\bar{T} > w$. The excitatory input pulse now passes from p_i to p_{i+2} before p_i-induced inhibitory transmission can. After a brief time interval of length $2(\bar{T}-w)$ of purely excitatory growth and transmission, s_{i+2} is suppressed by p_i-induced inhibition. Similarly, whenever $g(k) > 0$, s_{i+k} is suppressed by p_i. p_i-induced inhibition arrives at p_{i+k} $k(\bar{T}-w)$ time units after input-induced excitation does. Since all inhibitory contributions arrive at least $\bar{T}-w$ time units after input-induced excitation does, there exists a brief period of purely excitatory s_{i+k} growth and transmission before any inhibitory transmissions arrive at p_{i+k}. The total inhibition arriving at p_{i+n} at time t

from the past field $\{ p_{i+m} : m < n \}$ is proportional to

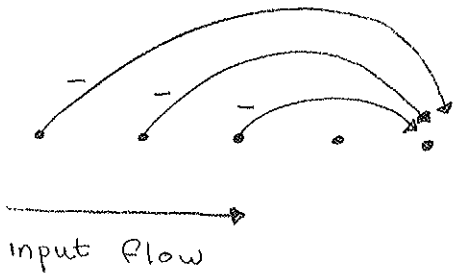
$$\sum_{m<n} \bar{p}_{i+m,\, i+n} s_{i+m}(t - \bar{t}_{i+m,\, i+n}) =$$

$$\sum_{m<n-1} \bar{p}_{i+m,\, i+n} s_{i+m}(t - (n-m)\bar{T}) =$$

$$\sum_{m<n-1} \bar{p}_{i+m,\, i+n} s_{i+n}(t + (n-m)(w - \bar{T})).$$

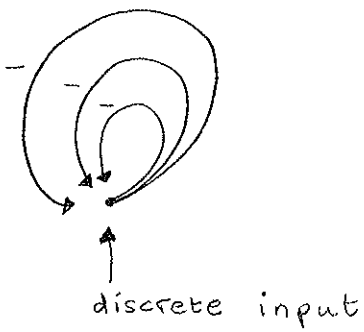
We have here used the translation stability with period w of the strength functions that follows from translation stability of the input. The total inhibitory input from the past field can be collapsed to a sum of inhibitory self-excitations of p_{i+n} at regularly spaced times. The entire past field of p_{i+n} can thus be ignored when we consider a translation stable input flow if only inhibitory contributions are being studied, since all of these contributions are equivalent to self-inhibitions. Indeed, let a measuring device be implanted in p_{i+n} and be sensitive only to p_{i+n} and not to the surrounding points. A theorist studying the measurements recorded from such a device will think that p_{i+n} regularly inhibits itself after an excitatory input is introduced. A single excitatory input to a point with staggered inhibitory self-excitations generates the same inhibitory transmissions as a translation stable input flow does. Moreover, whenever $w < \bar{T}$, these self-inhibitions will enter the equation for s_{i+n} as retarded potentials, and this equation will exhibit retarded inhibitory potentials from the past field if and only if $w < \bar{T}$. We conclude that there exist dynamical systems whose variables are not really governed by self-inhibitory terms but which, when observed locally, appear to exhibit self-inhibitions of a retarded potential type. In order to rectify this local illusion, the experimenter must have the good fortune to be measuring from a point that is perturbed by input flows of varying velocities which distinguish the global properties of the transmission structure. The trend of the discussion shows,

however, that as increasingly effective provisions are made to insure c* stability, the variability of admissible sustaining input flows decreases. In very stable situations, we can therefore expect to find very little mean variation in the form of the sustaining input schemata. The experimentalist interested in such steady state phenomena can therefore be easily deluded into drawing a theoretical picture with localized self-excitatory retarded potentials. The actual diagram



(I)

is contracted to



(II)

Since local descriptions are often preferred to non-local descriptions and since (I) and (II) are indistinguishable with respect to inhibitions induced by the past field, (II) will usually be considered a preferable theoretical picture to (I), but (I) is correct and (II) is not.

We find strictly retarded self-inhibitory potentials from the past field only if $T^{\cdot\cdot} < w$. If $w < T^{-}$, self-inhibitory advanced potentials are introduced. For advanced potentials, inhibitory transmissions from p_i reach p_{i+n} $n(T^{-}-w)$ time units before the excitatory sustaining input wave does. s_{i+n} is thus successively suppressed to increasingly small values as repetitive inhibitory transmission waves reach p_{i+n} from $\{p_{i+k}: k < n\}$. When the excitatory

wave finally does arrive at p_{i+n}, it must compete with the prior inhibitory suppression and is not effective in raising s_{i+n} to a sufficiently high value to generate a satisfactorily large $p_i \longrightarrow p_{i+1}$ excitatory transmission. If a nontrivial transmission threshold is given, it is even conceivable that no transmission whatever occurs when $w < T^-$, although transmission is necessary for the preservation of the global weighting of c values. Thus $T^- < w$, and retarded potentials, is a preferable condition to $w < T^-$ from the viewpoint of c^* invariance.

Summarizing, we see that surrounding a point p_i by a proximal "on" region, to which it sends excitatory lines (here $\{ p_{i-1}, p_{i+1} \}$) and a more distal "off" region, to which it sends inhibitory lines (here $\{ p_{i+k}: g(k) > 0 \}$), facilitates c^* invariance and the global preservation of c weights. When the linear asymptote $c^*_{i,i+1} = 1$ is nearly satisfied, a translation stable sustaining input flow is called for, and this flow may be locally described by a single input onset followed by self-induced transmission terms which are often composed of sums of retarded potentials.

88. Fields and Antifields

The above example of c^* invariance used the existence of a proximal "on" region and a distal "off" region in a critical way. Do there exist situations in which we could have replaced the proximal "on" region by a proximal "off" region, and the distal "off" region by a distal "on" region? If we do decide to make this replacement, we would also have to replace $T^- < w$ by $w < T^-$ and the excitatory input wave by an inhibitory input wave. Without these compensatory changes, excitatory transmissions generated in the distal "on" region arrive at p_i after the excitatory input pulse arrives at p_i, and no excitatory-then-inhibitory sequence of inputs exists. With these compensatory changes, an excitatory transmission generated in the distal "on" region arrives at p_i before the inhibitory input wave arrives, and once again a brief period of purely excitatory growth and transmission is permitted before inhibitory suppression sets in. These compensatory changes require that an inhibitory input generate excitatory transmissions. How can an inhibitory wave generate excitatory transmissions? This

can only be accomplished if we also replace the strength functions s, which we now write as s^+, by strength functions s^- which are excited by precisely those input events which suppress s^+. The purely geometrical exchange of proximal "on" regions and distal "off" regions thus requires changes in both temporal and dynamical functions as well, if c* invariance is not to be violated. We call the field $\mathcal{F}^-$ generated by these changes in $\mathcal{F}$ the antifield of $\mathcal{F}$. We now turn to a closer study of antifields.

How does the backward flow from the future field $\{ p_{i+m} : m > 0 \}$ of p_i look in $\mathcal{F}^-$? The total inhibitory input received at p_i from its future field in $\mathcal{F}$ at time t is, ignoring c functions, proportional to

$$\sum_{0 < m} p^-_{i+n,i} \, s_{i+n}(t-mT^-) = \sum_{1 < m} p^-_{i+n,i} \, s_i(t-m(w+T^-)).$$

Only the sum $w+T^-$ of w and T^- enters the localized self-inhibitory description. The relative sizes of w and T^- are unimportant and we always find retarded potentials. When we pass to $\mathcal{F}^-$, changing $(-)$ signs to $(+)$ signs, we find that the total excitatory input received at p_i from its future field in $\mathcal{F}^-$ at time t is proportional to

$$\sum_{0 < m} p^+_{i+n,i} \, s_{i+n}(t-mT^+) = \sum_{1 < m} p^+_{i+n,i} \, s_i(t-m(w+T^-)).$$

These self-excitations arrive at p_i after the inhibitory input wave does. In order to insure the excitatory-then-inhibitory wave sequence that is so desirable for c* invariance, we must also replace $w+T^-$ by $-(w+T^-)$ to find the advanced potentials

$$\sum_{1 < m} p^+_{i+n,i} \, s_i(t+m(w+T^-)).$$

This replacement is exactly analogous to the reversal of the inequalities $w < T^-$ and $T^- < w$ which occurs when passing from c* invariance in $\mathcal{F}$ to c* invariance in $\mathcal{F}^-$ with respect to transmissions from the past field of p_i.

This reversal amounts to the mapping $w-T^- \longrightarrow -(w-T^-) = T^- - w$. The passage from $\mathcal{T}$ to $\mathcal{T}^-$ is therefore accompanied by the mapping

$$\begin{pmatrix} w-T^- \\ w+T^- \end{pmatrix} \rightarrow - \begin{pmatrix} w-T^- \\ w+T^- \end{pmatrix}$$

which is a time-reversal operator applied to the time coordinates measuring the relative rates of flow of the input pulse and the transmission pulses within the field.

89. Macroscopic and Microscopic Intensive Continua

In order to understand the symmetries which are involved in passing from $\mathcal{T}$ to $\mathcal{T}^-$, we must exploit the hint that there exist s^- functions which behave quite differently from s^+ functions under inhibitory inputs. We first write the dynamical equations in a slightly different way. To the present, we have considered equations of the form

$$ds_i/dt = \alpha(M_i - s_i)I_i^+ - \beta s_i(1+I_i^-),$$

where I_i^+ is an induced excitatory input to p_i, I_i^- is an induced inhibitory input to p_i, and 1 is a spontaneous inhibitory input to p_i. 1 creates an ostensible asymmetry in the excitatory and inhibitory inputs. This asymmetry was introduced to assure that when α and β are properly chosen, $\lim_{t \to \infty} s_i(t) = 0$ in a free embedding field ($I_i^+ \equiv 0$), where zero is the equilibrium potential P_{eq} of the field.

Why did we set $P_{eq} = 0$? We began with macroscopic psychological data for which the natural number scale is plotted from a zero response level to a certain maximum level. With this response scale in mind, we scaled our strength functions to grow from 0 to M_i for convenience, and noticed that responding only occurs when certain members of the strength distribution strictly exceed zero.

Zero itself never entered the discussion as an intrinsic theoretical quantity, but only as a scaling factor. The implication of this insensitivity to zero is that, whatever be the microscopic laws that combine to generate macroscopic behavioral events, they must be insensitive to such scaling matters as the choice of an initial scaling point. All that really matters is how dynamical functions distribute themselves within the chosen scale. This fact is emphasized by the introduction of nontrivial excitatory transmission thresholds $\Upsilon_i > 0$. All that matters to a transmission threshold is how far the intensity of excitation has deviated from P_{eq}. From the perspective of a macroscopic observer, Υ_i is a more natural level at which to begin scaling than P_{eq}, since macroscopic behavioral events are only generated when transmissions induce a motor act of some kind. The existence of intensive levels below Υ_i permits the conditioning of dynamical events before any behavioral event occurs, but the form of the laws governing this conditioning process obviously cannot drastically change by simply exceeding the threshold Υ_i, for the conditioning process itself is determined by transmissions which are generated by laws that are restricted to the levels $[\Upsilon_i, M_i]$. It can easily be seen that a splitting in the dynamical laws governing excitation densities below Υ_i/h_i and densities above Υ_i/h_i would introduce a chaotic behavioral paradigm. The macroscopically visible intensive dynamical continuum must therefore be embedded in the microscopic intensive dynamical continuum in such a way that the form of dynamical laws does not change when they are viewed in $[\Upsilon_i, M_i]$ or in $[P_{eq}, M_i]$. Υ_i can, however, be chosen anywhere in $[P_{eq}, M_i)$, and P_{eq} is itself determined only up to a scaling factor. All dynamical laws therefore depend only on how much a given dynamical level s_i deviates from M_i and from m_i, the smallest potential allowed in our scaling. The intensive dynamical continuum must be a homogeneous series of intensive levels whose effects on dynamical behavior depend only on how s_i lies relative to M_i and to m_i. That is, M_i and m_i enter dynamical growth laws as $M_i - s_i$ and as $s_i - m_i$. When $P_{eq} = m_i = 0$, we find $M_i - s_i$ and s_i as the local densities which are directly influenced by excitatory and inhibitory transmission, respectively. This is the special scaling which we have been using to the present.

Since $P_{eq} = m_i = 0$ is an arbitrary scaling choice, we can rescale P_{eq}

in a more symmetrical way without essentially changing anything, just so long as $P_{eq} \leq \tau$. To find P_{eq}, we must first pick a scale $[m, M]$ on which to estimate dynamical quantities. We then write the equation for s in a symmetrical way, since spontaneous decay is a scaling asymmetry:

$$ds/dt = \alpha^+(M-s)(1+I^+) - \alpha^-(s-m)(1+I^-) .$$

$P_{eq} = P_{eq}(M, m; \alpha^+, \alpha^-)$ is defined as that dynamical value assumed by s when $I^+ \equiv I^- \equiv ds/dt \equiv 0$. Thus,

$$0 = \alpha^+(M-P_{eq}) - \alpha^-(P_{eq}-m),$$

or

$$P_{eq}(M, m; \alpha^+, \alpha^-) = \frac{\alpha^+ M + \alpha^- m}{\alpha^+ + \alpha^-}$$

We choose a special symmetric case for simplicity: $\alpha^+ = \alpha^- = 1$, $m = 0$. Then $P_{eq} = M/2$, and we have the equation

$$ds/dt = (M-s)(1+I^+) - s(1+I^-),$$

which we write in the more suggestive form

$$ds^+/dt = (M-s^+)(1+I^+) - s^+(1+I^-).$$

In this symmetric situation, field symmetries assume a particularly transparent form.

90. Antagonistic Processes

Transform all excitatory inputs into inhibitory inputs and all inhibitory inputs into excitatory inputs:

$$\begin{pmatrix} I^+ \\ I^- \end{pmatrix} \longrightarrow \begin{pmatrix} I^- \\ I^+ \end{pmatrix}$$

Call the variable for the resulting equation s^-:

$$ds^-/dt = (M-s^-)(1+I^-)-s^-(1+I^+).$$

s^- is excited by precisely those inputs that inhibit s^+. Map s^- into $M-s^- = y$ to find

$$-dy/dt = y(1+I^-)-(M-y)(1+I^+),$$

or

$$dy/dt = (M-y)(1+I^+)-y(1+I^-).$$

The equation for y is precisely the same as the equation for s^+, so $y = s^+$. Thus the embedding equation

$$ds^+/dt = (M-s^+)(1+I^+)-s^+(1+I^-)$$

is invariant under the transformation

$$\begin{pmatrix} s^+ \\ I^+ \end{pmatrix} \longrightarrow \begin{pmatrix} M-s^+ \\ I^+ \end{pmatrix},$$

which can also be written

$$\begin{pmatrix} s^+ \\ I^+ \end{pmatrix} \qquad \begin{pmatrix} s^- \\ I^+ \end{pmatrix}$$

Similarly,

$$ds^+/dt = \alpha\left[(M-s^+)(1+I^+)-(s^+-m)(1+I^-)\right]$$

is invariant under the transformation

$$\begin{pmatrix} s^+ \\ I^+_- \end{pmatrix} \longrightarrow \begin{pmatrix} M-m-s^+ \\ I^{\mp} \end{pmatrix} .$$

Thus, if we exchange excitatory and inhibitory inputs at the same time that we exchange the roles of M and m, the physical situation is not changed. From this point of view, M should be viewed as the excitatory asymptote for s^+, and m as the inhibitory asymptote for s^+. Since s^- responds oppositely from s^+ to a given input, its excitatory and inhibitory asymptotes are the reverse of the s^+ asymptotes.

The insensitivity of macroscopic data to microscopic scaling thus implies a beautiful symmetry in microscopic dynamics. This particular symmetry suggests a physical analogy. The operator $s \longrightarrow M-m-s$ is analogous to the physical phase space operator P: $(q, p) \longrightarrow (-q, -p)$, and the operator $I^+_- \longrightarrow I^{\mp}$ is analogous to the physical charge conjugation operator C. In terms of this analogy, we have found a type of CP invariance in local embedding dynamics.

What happens to the c_{ij} functions under these mappings? When $P_{eq} = \Upsilon = 0$,

$$dc_{ij}/dt = \gamma^+_{ij}(A_{ij}-c_{ij})s_i(t-t_{ij})s_j - \gamma^-_{ij}\, c_{ij}(M_i-s_i(t-t_{ij}))(M_j-s_j).$$

It is easy to see that whenever $\gamma^+_{ij} = \gamma^-_{ij}$, the mapping

$$\begin{pmatrix} s_i \\ c_{ij} \end{pmatrix} \longrightarrow \begin{pmatrix} M_i-s_i \\ A_{ij}-c_{ij} \end{pmatrix}$$

leaves this equation invariant. Thus, whenever $\alpha^+ = \alpha^-$, $\gamma^+_{ij} = \gamma^-_{ij}$, and $P_{eq} = \Upsilon = 0$, if we let $c = c^+$,

$$\Omega: \quad \begin{pmatrix} s^+ \\ c^+ \\ I^{\pm} \end{pmatrix} \longrightarrow \begin{pmatrix} M-s^+ \\ A-c^+ \\ I^{\mp} \end{pmatrix} = \begin{pmatrix} s^- \\ c^- \\ I^{\mp} \end{pmatrix}$$

leaves the entire system of embedding equations invariant, just so long as we let $p = p^{+} = p^{-}$.

When I^{+} and I^{-} are not independent of s and c, Ω transforms their s and c dependent terms. For example, suppose that

$$I_i^{+} = \bar{I}_i^{+} + r\sum_k s_k^{+}(t-t_{ki}^{+})\, p_{ki}^{+} c_{ki}^{+},$$

where $\bar{I}_i^{+}$ is not a function of s^{+} and c^{+}. When we apply Ω, $I_i^{+} \longrightarrow I_i^{-}$, $s_i^{+} \longrightarrow s_i^{-}$, and $c_{ij}^{+} \longrightarrow c_{ij}^{-}$, so that if we also let $p_{ij}^{+} \longrightarrow p_{ij}^{-}$ and $t_{ij}^{+} \longrightarrow t_{ij}^{-}$,

$$I_i^{-} = \bar{I}_i^{-} + r\sum_k (M_k - s_k^{+}(t-t_{ki}^{-}))p_{ki}^{-}(A_{ki} - c_{ki}^{+}).$$

Thus

$$I_i^{-} = \bar{I}_i^{-} + r\sum_k s_k^{-}(t-t_{ki}^{-})\, p_{ki}^{-} c_{ki}^{-},$$

and we have the involutory mapping

$$\Omega: \quad \begin{pmatrix} s^{+} \\ c^{+} \\ I^{+} \\ p^{+} \\ t^{+} \end{pmatrix} \longrightarrow \begin{pmatrix} s^{-} \\ c^{-} \\ I^{-} \\ p^{-} \\ t^{-} \end{pmatrix}$$

which leaves the field invariant. We have not, to the present, discussed functions c^{-} as real dynamical entities, but have rather always considered functions $c = c^{+}$ which grow when $s = s^{+}$ excitation is increased. In terms of c^{+} and s^{+}, the usual equation for $c = c^{+}$ becomes

$$dc_{ij}^{+}/dt = \gamma_{ij}^{+} c_{ij}^{-} s_i^{+}(t-t_{ij}^{+}) s_j^{+} - \gamma_{ij}^{-} c_{ij}^{+} s_i^{-}(t-t_{ij}^{-}) s_j^{-}.$$

We find the equation for c^- by changing signs:

$$dc_{ij}^-/dt = \gamma_{ij}^- c_{ij}^+ s_i^- (t-t_{ij}^-) s_j^- - \gamma_{ij}^+ c_{ij}^- s_i^+ (t-t_{ij}^+) s_j^+ .$$

Formally these considerations are very simple. The c_{ij}^- functions which arise so naturally when we complete the field-antifield duality in a symmetric way show that it would not be surprising to actually empirically find c^- functions which are suppressed by s^+ excitation and activated by s^- excitation. This is exactly the opposite of the behavior expected from c^+ functions. By considering the pairs (s^+, s^-) and (c^+, c^-) as carriers of antagonistic processes, rather than merely as various functions associated with a single process, we can see the beautiful symmetry which has been achieved to perfect stable nonlinear binding in the embedding field situation. The vectors (s^+, s^-) and (c^+, c^-) may be coupled in several ways. To determine a given coupling, one must consider all features of the dynamical interactions in a given example. For example, if in two different situations c activation excites s^+ and s^-, we would be inclined to search for two antagonistic transmitters released by c activation, or two antagonistic membrane processes, one which excites s^+ and another which inhibits s^-.

When $\tau > 0$, the functions s_k, $k=i, j$, determining c_{ij} are replaced by $\max(s_k - \tau_k, 0)$, and all of our previous remarks are altered in an obvious way.

91. c* Invariance and Field Symmetries

With these symmetries in mind, we can return to a discussion of c* invariance for a linear asymptote with greater assurance. c* invariance is now interpreted as $(c^+)^*$ invariance. We earlier saw that $(c^+)^*$ invariance is compatible with two different kinds of geometrical situation. In the one, proximal "on" transmissions and distal "off" transmissions are given. In the other, proximal "off" transmissions and distal "on" transmissions are given. Each situation is associated with a set of dynamical equations, and to pass from one set of equations to another, we merely exchange the roles of excitatory and inhibitory asymptotes and the accompanying dynamical functions. We also distinguish these two situations by exchanging the inequalities $w < T^-$ and $T^- < w$, and this exchange is taken care of by the map

$$\begin{pmatrix} w-T^- \\ w+T^- \end{pmatrix} \longrightarrow - \begin{pmatrix} w-T^- \\ w+T^- \end{pmatrix} .$$

Because of the symmetric roles of c^+ and c^-, $(c^+)^*$ invariance is almost equivalent to $(c^-)^*$ invariance. The two kinds of invariance match the field-antifield duality. The choice between one or the other type of invariance is decided by questions of psychological emphasis rather than by crucial theoretical differences, since by turning the entire field situation upside-down excitatory influences look inhibitory and vice versa. When we consider $(c^-)^*$ invariance relative to a fixed notion of $(c^+)^*$ invariance, the order of the $w < T^-$ and $T^- < w$ inequalities is exchanged, since inhibitory waves replace excitatory waves and the inhibitory input replaces the excitatory input. Thus, we want to find an inhibitory-then-excitatory input wave pattern at each p_i under $(c^-)^*$ invariance. $(c^-)^*$ invariance generates the mirror image of $(c^+)^*$ invariance with respect to spatial, temporal, and dynamical functions. Neither the geometry nor the dynamics of either field or antifield is independent of the other. Geometry and dynamics are molded into a single coherent frame. The study of one without the other is inconceivable. Local dynamical laws have evolved expressly to permit stable dynamical interactions on a global scale within a certain geometrical carrier, and conversely. We will see many examples of this close geometrico-dynamical bond in later pages.

The reversal of the two inequalities $w < T^-$ and $\bar{T} < w$ when passing from $(c^+)^*$ invariance to $(c^-)^*$ invariance and the exchange of retarded potentials for advanced potentials in future field self-inhibitions can be realized by the mapping

$$\begin{pmatrix} w-T^- \\ w+T^- \end{pmatrix} \longrightarrow - \begin{pmatrix} w-\bar{T} \\ w+T^- \end{pmatrix}$$

which is equivalent to

$$\begin{pmatrix} w \\ T^- \end{pmatrix} \longrightarrow - \begin{pmatrix} w \\ T^- \end{pmatrix}.$$

We hereby reverse the time coordinates for the relative direction of flow of the input pulse and the transmission pulses within the field. To an observer who can measure time only relative to the motion of one of these systems, this relative time reversal will appear as a time reversal in the direction of the observer's time scale.

Given a translation stable flow, under the mapping

$$\Omega^-: \quad \begin{pmatrix} s^+ \\ c^+ \\ p^+ \\ I^+ \\ w \\ T^+ \end{pmatrix} \longrightarrow \begin{pmatrix} \bar{s}^+ \\ \bar{c}^+ \\ \bar{p}^+ \\ \bar{I}^+ \\ -w \\ -\bar{T}^+ \end{pmatrix}$$

we pass from one physical situation to an equivalent one. If we write $w=w^+$, $\bar{w}=-w$, and $\bar{T}^+ = -T^+$, Ω^- simply changes all (+) signs to (-) signs, and vice versa, and is thus obviously involutory. Ω^- simultaneously reverses the relative direction of time between input and transmission flows, switches all measuring rules relative to the excitatory and inhibitory asymptotes, and exchanges inhibitory and excitatory lines, including the lines over which input pulses arrive. These alterations are reminiscent of the operators of time

reversal T, parity reversal P, and charge conjugation C which one finds in quantized physical fields. Viewed in this way, the invariance of the embedding

equations under Ω^- becomes an analog of a PCT theorem for embedding fields, the content of the theorem in this context being that, by exchanging (+) and (-) signs in a suitable way, one passes from a given field to its antifield. This theorem has arisen in the present context entirely from the desire to assure $(c^{\pm}_{\mp})^*$ invariance, and the principle of $(c^{\pm}_{\mp})^*$ invariance intuitively means simply that the interaction structure is arranged to insure its own stability. In this example, we therefore find a dynamical system for which a stability condition implies important field symmetries.

Notice that if a time reversal $t \longrightarrow -t$ accompanies $(w,T^-) \longrightarrow (-w,-T^-)$, then in the transmission term

$$\sum_{m<n-1} p^-_{i+m,i+n} c^-_{i+m,i+n} s^-_{i+n}(t+(n-m)(w-T^-)),$$

$t+(n-m)(w-T^-)$ is mapped into $-(t+(n-m)(w-T^-))$, so that the $(t,w,T^-) \longrightarrow (-t,-w,-T^-)$ time reversal operator reverses even the sign of the argument of the difference terms. On the other hand, under $t \longrightarrow -t$, the right hand side of the equation for $s^{\pm}_{\mp}$ is multiplied by -1, since we are dealing with first-order differential equations. We hereby find $s_i^{\pm}$ functions which grow fastest when $s_i^{\pm}$ is small, rather than when s_i^+ is small, and whose growth is stimulated by large s_j^+, $j \neq i$, rather than large $s_j^{\pm}$ values. Moreover, retarded (advanced) potentials are mapped into retarded (advanced) potentials. Unless the addition of $t \longrightarrow -t$ to Ω^- is compensated by further changes in the field operators, $t \longrightarrow -t$ induces field behavior which is nonintuitive. Without further changes, $(c^{\pm}_{\mp})^*$ invariance recommends the relative time reversal operator alone, and t is viewed as a formal parameter against which to measure the relative velocities of input and transmission pulses.

92. Excitatory and Inhibitory Transmission Types

Having briefly considered the involutory symmetry of the field and antifield pictures, we now recall that in our initial studies of lateral inhibitory effects, for example in the "thalamus," the total induced excitatory term Σ^+ and the total induced inhibitory term Σ^- were of the form

$$\Sigma^+ = \sum p^+ c^+ s^+, \qquad \Sigma^- = \sum p^- c^- s^+,$$

where we chose $c^- = 1$. Even if we allow c^- to vary, $\Sigma^- = \sum p^- c^- s^+$ is not the antifield dual of $\Sigma^+ = \sum p^+ c^+ s^+$, for the dual of Σ^+ is obviously $\sum p^- c^- s^-$. This failure in $\mathcal{J} - \mathcal{J}^-$ duality is a necessary one, for we definitely wanted the excitation of one point to inhibit the activation of nearby points. The asymmetry in the excitatory and inhibitory contributions is matched by an asymmetry in the transmission thresholds which must be exceeded for lateral inhibition to occur.

Can we preserve these ostensible violations of $\mathcal{J} - \mathcal{J}^-$ duality in a field which nonetheless exhibits this duality? The answer is yes. To do this, we must extend the field once again. First it is necessary to introduce the following classification of points. Suppose that to every point p_i is associated a transmission threshold τ_i and an equilibrium potential P_i^{eq}. Either $\tau_i < P_i^{eq}$ or $\tau_i \geqslant P_i^{eq}$. We denote the class of points for which the former is true by $\Pi^{\downarrow}$ and the class of points for which the latter is true by $\Pi^{\uparrow}$. The points for which $\tau_i = P_i^{eq}$ are placed in $\Pi^{\uparrow}$ arbitrarily, and it is not clear that such points actually occur in neural interactions. When an input I_i to p_i causes s_i to deviate from P_i^{eq} in the direction of τ_i, we say that I_i excites p_i. When a transmission from p_i causes s_i to deviate from P_i^{eq} away from τ_i, we say that I_i inhibits p_i.

We must classify points according to whether exciting them produces transmissions which excite or inhibit recipient points. Pick any $p_i \in \Pi^{\uparrow}$ and imagine that transmission lines are drawn from p_i to all points. Denote the set of points in $\Pi^{\uparrow}$ ($\Pi^{\downarrow}$) which are excited by a transmission from p_i by $\Pi_+^{\uparrow\uparrow}(i)$ ($\Pi^{\uparrow\downarrow}(i)$) and the set of points which are inhibited by transmission from p_i $\Pi_-^{\uparrow\uparrow}(i)$ ($\Pi_-^{\uparrow\downarrow}(i)$). The set of $\Pi^{\uparrow}$ ($\Pi^{\downarrow}$) points which are neither excited nor inhibited by p_i transmission, say because the transmitter liberated by p_i endbulbs

does not excite the recipient membrane, is denoted by $\Pi_o^{\uparrow\uparrow}(i)$ ($\Pi_o^{\uparrow\downarrow}(i)$). p_i thus completely partitions all points as

$$\Pi_+^{\uparrow\uparrow}(i)\oplus\Pi_-^{\uparrow\uparrow}(i)\oplus\Pi_o^{\uparrow\uparrow}(i)\oplus\Pi_+^{\uparrow\downarrow}(i)\oplus\Pi_-^{\uparrow\downarrow}(i)\oplus\Pi_o^{\uparrow\downarrow}(i).$$

For $p_i \in \Pi^{\downarrow}$, the points in $\Pi^{\uparrow}$ ($\Pi^{\downarrow}$) excited by transmission from p_i are denoted by $\Pi_+^{\downarrow\uparrow}(i)$ ($\Pi_+^{\downarrow\downarrow}(i)$) and the inhibited points by $\Pi_-^{\downarrow\uparrow}(i)$ ($\Pi_-^{\downarrow\downarrow}(i)$). When we pass from a field to its antifield, $\Pi_u^{\uparrow\uparrow}(i)$ ($\Pi_u^{\uparrow\downarrow}(i)$) becomes $\Pi_u^{\downarrow\downarrow}(i)$ ($\Pi_u^{\downarrow\uparrow}(i)$), u=+,-,0.

It is natural to ask how the various partitions overlap when i is allowed to vary. Given any two partitions of a set W, say $W=W_1\oplus W_2=W^1\oplus W^2$ for simplicity, the partition

$$W=(W_1\cap W^1)\oplus(W_1\cap W^2)\oplus(W_2\cap W^1)\oplus(W_2\cap W^2)$$

derived by taking all intersections of $\{W_1,W_2\}$ with $\{W^1,W^2\}$ is called the refinement of W induced by $\{W_1,W_2\}$ and $\{W^1,W^2\}$. To discuss the overlap of the p_i-induced partitions, we pass to the refinement induced by $\{\Pi_u^{\uparrow\uparrow}(i),\Pi_u^{\uparrow\downarrow}(i): u=+,-,0\}$ for every $p_i \in \Pi^{\uparrow}$. Every set in the refined partition is called a $\Pi^{\uparrow}$ transmission type. If we assume that $\mathcal{T}$-$\bar{\mathcal{T}}$ duality strictly holds, then to every $\Pi^{\uparrow}$ transmission type Π is associated another transmission type $\widetilde{\Pi}$ which is its antifield analogue. Each $\widetilde{\Pi}$ is a $\Pi^{\downarrow}$ transmission type, in a natural sense. Given $\mathcal{T}$-$\bar{\mathcal{T}}$ duality, every $\Pi^{\downarrow}$ transmission type is a $\widehat{\Pi}$. In this circumstance, it is not impossible that Π, $\widetilde{\Pi}$ interactions exist, just so long as they are also dual. Instead of writing $\Pi(\widetilde{\Pi})$ for a $\Pi^{\uparrow}(\Pi^{\downarrow})$ transmission type, we can now write $\Pi^{\uparrow}(\Pi^{\downarrow})$ without danger of confusion.

Various $\Pi^{\uparrow}$, $\Pi^{\downarrow}$ interaction possibilities exist. When the transmissions of a given $\Pi^{\uparrow}$ type excites a given $\Pi^{\downarrow}$ type, we write

$$\Pi^{\uparrow} \xrightarrow[+]{} \Pi^{\downarrow}.$$

Similarly,

$$\Pi^{\uparrow} \xrightarrow[-]{} \Pi^{\downarrow}.$$

means that $\Pi^{\uparrow}$ transmission inhibits $\Pi^{\downarrow}$, and

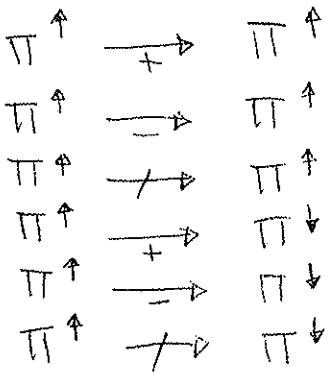
$$\Pi^{\uparrow} \not\longrightarrow \Pi^{\downarrow}$$

means that $\Pi^{\uparrow}$ transmission has no effect on $\Pi^{\downarrow}$.

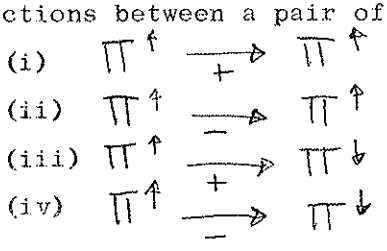
The decomposition of all points into transmission types groups together all points which have similar dynamical effects on all other points of a given type. These effects can be represented with a kxk transmission type matrix $\Delta(\Pi) = \{\Delta_{ij}(\Pi)\}$ by listing all transmission types in some given order $\Pi_1, \Pi_2, \ldots, \Pi_k$, and letting

$$\Delta_{ij}(\Pi) = \begin{cases} +1 & \text{if } \Pi_i \xrightarrow{+} \Pi_j \\ -1 & \text{if } \Pi_i \xrightarrow{-} \Pi_j \\ 0 & \text{if } \Pi_i \not\longrightarrow \Pi_j \end{cases} .$$

We can easily enumerate the possible interactions between a $\Pi^{\uparrow}$ type and a $\Pi^{\downarrow}$ type. They are



and the six interactions found by dualizing ($\Pi^{\uparrow} \longrightarrow \Pi^{\downarrow}$, $\Pi^{\downarrow} \longrightarrow \Pi^{\uparrow}$). Omitting non-interacting types and dualizations, we find just four possible nontrivial interactions between a pair of types:



Each of these four types, rather than merely the two coupled types (i)-(iii) and (ii)-(iv) is a distinguishable form of interaction because "$\uparrow$" and "$\downarrow$" introduce specific orientations into local dynamics. All nontrivial interactions are constructed from the model of (i) through (iv), but each of

(i),...,(iv) may appear in sets of types which either do not interact with one another at all, or interact with one another in patterns that change when the entire set of types is changed.

We use the notion of transmission types to introduce 7-7 duality into a field whose inhibitory transmissions ostensibly violate this duality. To do this, we must split p_i into two parts, $p_i = p_i^1 \oplus p_i^2$, and we must associate a set of field functions F_i^k with each p_i^k, $i=1,2$. F_i^1 replaces the original set of field functions of p_i and F_i^2 is introduced to induce lateral inhibitions. Duality is violated only if we try to produce all inhibitory interactions using either F_i^1 or F_i^2 alone, but not if we use the joint set $F_i^1 \oplus F_i^2$. F_i^1, for example, is now inhibited by $\{F_j^2 : j \neq i\}$ in the way

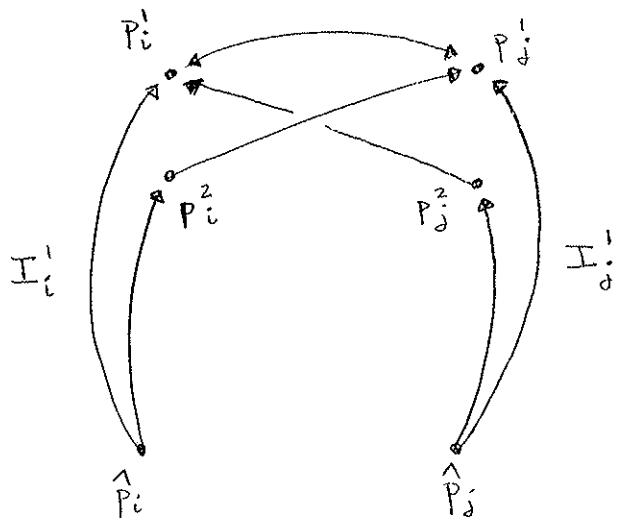
$$\Sigma_i^- = \gamma \Sigma_{k \neq i} p_{ki}^- c_{ki}^- s_k^+ (t - t_{ki}^-)$$

used to inhibit s_i. Σ_i^- is now replaced by

$$\Sigma_i^2 = \gamma \Sigma_{k \neq i} p_{ki}^{(2)} c_{ki}^{(2)} s_k^{(2)} (t - t_{ki}^{(2)}),$$

where $\{p_{ki}^{(2)}, c_{ki}^{(2)}, s_k^{(2)}\} \subset \cup_j F_j^2$. The exact production of lateral inhibition using two sets of field functions per point p_i instead of one can be accomplished in at least two ways.

A)

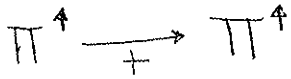


In this situation, $\hat{p}_i$ sends out two kinds of lines. The I_i^k input excites p_i^k for each $k=1,2$. However, p_i^1 transmission excites p_j^1, while p_i^2 transmission inhibits p_j^1. Since we assume, by symmetry, that all p_j^1 are of the same type,

and that all p_j^2 are of the same type,

$$p_i^1 \longrightarrow p_j^1$$

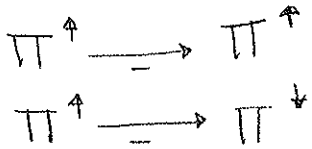
is an example either of



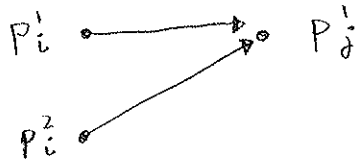
or its dual, while

$$p_i^2 \longrightarrow p_j^1$$

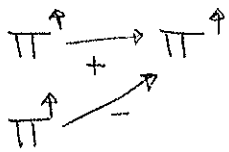
is an example of



or their duals. Thus

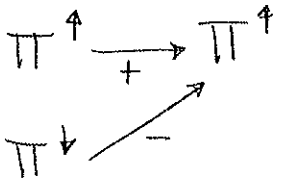


is



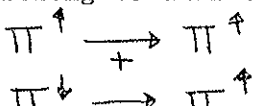
(α)

or



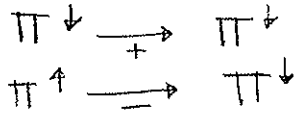
(β)

or a dual of these. If (α) holds, p_i^1 and p_i^2 are obviously of different transmission types, since they have different effects on a fixed p_j^2. If (β) holds, p_i^1 and p_i^2 can belong to dual types, for one way of dualizing



is (γ).

When we can dualize in this way, we also know that



and that .

(γ) means that switching the orientation of the transmission-inducing type also switches the effect of the transmission from excitation to inhibition, and vice versa. Whenever a pair of types (Π_1, Π_2) is given for which this is true, we call

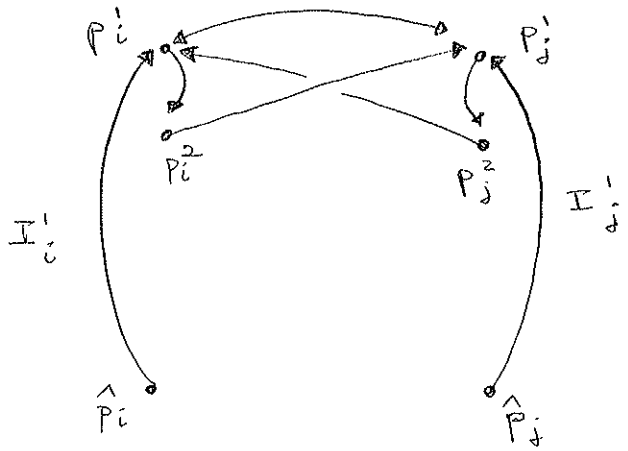
(Π_1, Π_2) a completely dual pair of types. Given any completely dual pair (Π_1, Π_2) and a transmission diagram, this diagram is equivalent to every diagram achieved by making precisely two changes of the form $+ \longrightarrow -, - \longrightarrow +$, $\uparrow \longrightarrow \downarrow$, or $\downarrow \longrightarrow \uparrow$.

(B) shows that the splitting $p_i = p_i^1 \oplus p_i^2$ need only introduce one completely dual pair of types into 7. Whether p_i inhibits or excites p_j depends entirely on the distribution of the lines $\mathcal{L}(p_i^k, p_j^m)$, k,m=1,2. Strong lateral inhibition effects can be achieved by deleting $\mathcal{L}(p_i^1, p_j^1)$ and leaving large $\mathcal{L}(p_i^2, p_j^1)$ densities. These effects can be made quite subtle by varying the four possible transmission latencies and the four possible line densities associated with any ordered pair (p_i, p_j).

One fact that distinguishes this way of producing inhibitions from the next way to be presented is that the input array itself is split into two components. If the input array accomplishes this splitting in the same way that the points $p_i = p_i^1 \oplus p_i^2$ do, then we expect that $\hat{p}_i = \hat{p}_i^1 \oplus \hat{p}_i^2$, and we can continue the pairwise decomposition of points, by a step-wise extension, to find that this method of splitting one point into completely dual components can be used to generate inhibitory transmissions without restriction. At every stage of this step-wise extension process, we must choose the transmission times and line densities in such a way that the global interaction of the various steps is organized to produce stable line asymptotes under a predetermined class of repetitive inputs.

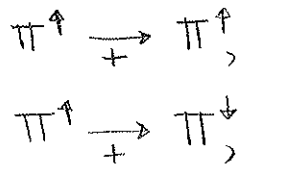
93. Inhibitory Interneurones and Dale's Principle

B) Another way to preserve $7 - 7''$ duality is to use a diagram like



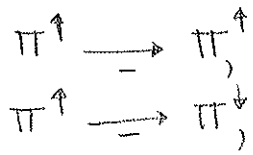
Here $\hat{p}_i$ excites p_i^1 but has no direct influence on p_i^2. Exciting p_i^1 by I_i^1 induces transmission which excites p_i^2. p_i^2 generates a transmission that inhibits p_j^1. Assuming by symmetry that each of $\{ p_k^1 \}$ and $\{ p_k^2 \}$ again belong to individual types, we have the following possibilities.

$p_i^1 \longrightarrow p_i^2$:

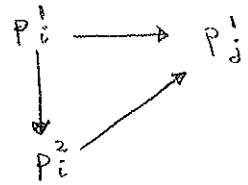


and their duals;

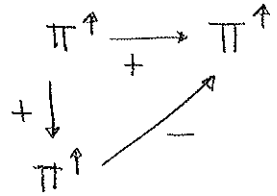
$p_i^2 \longrightarrow p_j^1$:



and their duals.

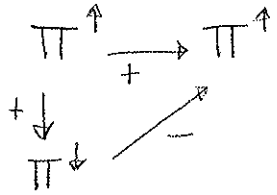


can therefore be realized by



(α')

or by



(β')

or their duals. In (α'), p_i^1 and p_i^2 are of different $\Pi^{\uparrow}$ types, and in (β'), p_i^1 and p_i^2 are not completely dual, as they were in (β). This way of introducing $p_i = p_i^1 \oplus p_i^2$ splitting thus requires at least two distinct transmission types. p_i^2 here serves as an inhibitory interneuron or inhibitory interneuronal pool which mediates inhibitions from a p_i^1 to a p_j^1 of the same transmission type as p_i^1.

An inspection of the diagrams for examples (A) and (B) shows that they preserve $7-\tilde{7}$ duality in the presence of lateral inhibitory interactions. In

particular, we see that the existence of inhibitory interneurones is a natural consequence of this duality and of intrinsic renormalizability, taken together, when only one type of input line perturbs the field. Dale's Principle is another such consequence.

94. Euclidean Packing Does Not Imply Interaction Packing. Multiple Synapses

The transmission type diagrams do not describe particular types. Rather each $\Pi^{\uparrow}$ or $\Pi^{\downarrow}$ symbol describes a whole class of equivalent types, When a particular type, indexed by i, is considered, we distinguish this fact by replacing $\Pi^{\updownarrow}$ by $\Pi_i^{\updownarrow}$. In particular, the pair of symbols $\Pi_i^{\uparrow}$ and $\Pi_i^{\downarrow}$ denotes a pair of completely dual types. It is not a priori impossible that a single point in some type excites one type and inhibits another, non-dual, type. Such a possibility does not necessarily violate Dale's Principle, for the same transmitter can generate different effects on receptive membranes whose electro-chemical fine structures differ. Our analysis shows, however, that even if many types exist, their interaction patterns are built up from a small number of alternatives. Examples (A) and (B) illustrate not only that we can use these alternatives to preserve $\mathcal{T}-\mathcal{T}^{-}$ duality in the face of ostensible violations of duality in Σ^{+} and Σ^{-} by splitting each type into components that subserve antagonistic processes. They also show that this duality can be preserved by using variable numbers of types. In example (A), only one completely dual pair of types was needed, and we showed that the splitting $p_i=p_i^1 \oplus p_i^2$ can be extended to $\hat{p}_i=\hat{p}_i^1 \oplus \hat{p}_i^2$, and so on, without ever using more than one such pair. In example (B), we needed two types to achieve a similar effect. Although we were able to remove the $\mathcal{L}(p_i^2,p_i^1)$ lines, we were forced to introduce new $\mathcal{L}(p_i^1,p_i^2)$ interneuronal lines and a new type. An advantage of (B) is that each p_i^2 can be the recipient of many p_j^k lines, k=1,2, which are ordered in definite ways that are determined locally without passing out of $\mathcal{T}(\{p_k^1\} \oplus \{p_k^2\})$. Moreover, the inhibitory process takes place in two stages $p_i^1 \xrightarrow{+} p_i^2 \xrightarrow{-} p_j^1$ instead of one, as in (A). It is, in fact, easy to see that by introducing increasingly large numbers of types, one can construct multi-stage inhibitory processes which are able to distinguish many more inputs than are distinguished by the single-stage process of (A). A major advantage of multi-staging is the introduction of multi-stage conditioning processes which are sensitive to simultaneous excitation from segments of the field of variable size. We earlier saw in fields of type M that

such conditioning processes are possible even when only one transmission type is available. When several types are available, much greater sensitivity is possible. In particular, several cell transmission types can be interspersed in small Euclidean regions without interacting with one another, or they may interact in well-ordered ways. Different types which are closely packed in Euclidean norm can be made to lie at large distances from one another when they are considered in terms of an interaction norm; for example, in interaction chains that are embedded in Euclidean space in ostensibly chaotic ways. Non-interacting types can independently prepare field averages, or can prepare averages which depend on one another in well-determined and non-chaotic ways. These same types that are so far away from one another in terms of the limited direct interactions which they share can simultaneously project to common interneurons and dual points. The greater sensitivity envisaged for multiple types immediately follows from this sharing of otherwise isolated averaging processes in common recipient points. By making special choices of structural densities between different types, we can construct dynamical systems that occupy minute regions of Euclidean space but nonetheless exhibit interaction patterns of a highly organized kind. When one considers the possibility of multiple synapses, with synaptic endings interpolated on more synaptic endings, and so on, the dilation introduced by replacing the Euclidean norm by an interaction norm becomes especially great when we admit the possibility of multiple types. All such constructions and extensions can be guided by principles like those which so naturally led to examples (A) and (B). Many of these we must leave to the reader confronted with particular experimental problems, for lack of space.

An inspection of the diagrams of these examples has shown that they do not violate field-antifield duality, even though they produce ostensibly non-dual interactions. These examples thus show that the appropriate field extension does not only remove non-intrinsic field functionals; it also removes ostensible field asymmetries. For completeness, we emphasize that each $p_i^{\pm}$ that arises in this extension is characteristically not a single cell. Rather it is a cell collection, and the macroscopic equations hold either if the microscopic densities of every $p_i^{\pm}$ are independent of the microscopic densities of every $p_i^{\mp}$; $p_j^{\pm}, j \neq i$; $q^{\pm}, r^{\pm}, \ldots$, or if these densities are dynamically indistinguishable modulo the inputs which perturb them.

95. Acts. Binary Alternatives

Although c* invariance for a linear asymptote recommends that the input pulse be at least locally translation stable and suggests an alternative between the inequalities $w < \bar{T}$ and $\bar{T} < w$, it does not, in its weakest form, completely determine the w required to preserve the linear asymptote. It is very useful to the organism that a certain freedom in choosing admissible w's exists. To see this most clearly, recall that the dynamics generating sustaining inputs and the dynamics controlling inputs directly associated with overt behavior share many common features. In the control of behavior, we are familiar with the fact that a single complicated sequence of behavioral motions can often be performed at various rates of speed; for example, walking. Yet we do not feel intuitively that these motions are essentially different when they are performed at different velocities. This feeling of the communality between certain sequences of motions performed at different velocities is a prerequisite to our belief that all such sequences are manifestations of a single behavioral "act". Underlying this perception of communality between these behavioral motions is the fact that all of the dynamical trajectories realizing these motions include many of the same points and lines, in the same order, and the dynamical functions of each trajectory are related to those of the other trajectories in a simple way. One embedded structure controls a single "act," and the control of this "act" in all of its forms involves the control of only one embedded structure. Were this not the case, no organism could hope to develop a flexible behavioral repertoire, for even trivial variations in behavioral sequences representing a fixed act would necessitate the embedding of entirely new trajectories. The "transfer" from one situation to another would be negligible and the organism could not adapt sensitively to its environment. The problem of allowing the w of translation stable sustaining flows to vary is thus closely tied to the problem of constructing an organism which can perform acts with adaptive variations in them.

Trying to construct such an organism from purely excitatory fields is a hopeless task. For example, let $\{p_i\}$ be embedded as a point set in $\mathbb{R}^2$ such that the Euclidean distance between p_i and p_{i+1} is λ, for all i. Also suppose that the input pulse flows in straight lines between p_i and p_{i+1} with velocity V. Then $w = \lambda/V$. Let us begin with the inequality $w < \bar{T}$. This inequality is preserved as $V \to \infty$. Suppose that the input I_i delivered to each p_i, measured from its onset, is invariant under changes in V. Let V increase, and let $\mathcal{T}^V$ be

the field generated for a fixed value of V. As V is increased, the relaxation times of the field functions remain constant. Thus as V grows, increasingly large numbers of points and lines of $\mathcal{F}^V$ will simultaneously have large values, and in the limit $V = \infty$, the field behaves as if it received a globally homogeneous input. When V is small, we find a local, ordered strength flow from each point in $\mathcal{F}^V$ to its successor, while when V is enormous, practically all points of $\mathcal{F}^V$ are simultaneously excited and all dynamical ordering vanishes. The only response point which cannot distinguish such enormous changes in $\mathcal{F}$ is one which only registers whether or not some point in $\mathcal{F}$ is activated at all. A collection of such response points generates an organism whose response paradigm is isomorphic to a direct sum of binary alternatives. If we want a more subtle organism, we must search for something more complicated than a purely excitatory field. Our consideration of intrinsic renormalizability, c* invariance, etc., suggested that we consider mixed fields whose inhibitory and excitatory lines are distributed in ordered geometrical arrays. For (c^+)* invariance, the proximal "on" field and distal "off" field is a natural choice when the lines of $\mathcal{F}$ are asymptotic to an infinite chain, or a loop.

96. Mixed Fields and Variable Input Wave Velocities

In order to study mixed field from this point of view, we consider a still simpler, but very important, field. Again let $\{ p_i \}$ be embedded in $\mathbb{R}^2$ in such a way that the distance between p_i and p_{i+1} is λ. Also let the input pulse flow in a line between p_i and p_{i+1} with velocity V, and order the points so that they lie in an infinite straight line. Suppose that the lines l_{ij} passing from p_i to p_j are straight and let $t_{ij}=T$ whenever $i \neq j$. The velocity of transmission within l_{ij} is thus proportional to the length of l_{ij}. Again $g(k) = \bar{p}_{i+k,i}$ is chosen to have a compact support and at most one maximum.

The choice $t_{ij}=T, i \neq j$, means that all events at p_i are felt by the rest of the field at the same time. All staggering of field transmissions to a fixed p_j reflect nonuniformities in the input sequence itself. Indeed, when the input is a globally homogeneous function of time: $I_i(t)=I_j(t)$, all transmission arriving at p_j can be locally expressed as self-inputs of the form $p_j^{\pm} s_j^{\pm}(t-T)$, $p_j^{\pm}$ being constants. The entire field is contracted to a minimal field with only one point, and a single self-input term for each of the total inhibitory and the

total excitatory transmission terms. Homogeneous fields with $t_{ij}=T$, $i \neq j$, are uniquely privileged in their capacity to reflect input asymmetries in their minimal fields in the simplest possible way.

Under a translation-stable input flow, suppose that the total inhibitory input to p_i from $\{ p_{i-k} : k \geq 1 \}$ is proportional to

$$\sum_{0<k} \bar{p}_{i-k,i} \bar{c}_{i-k,i} s_{i-k}(t-t_{i-k,i}) =$$

$$\sum_{0<k} \bar{p}_{i-k,i} \bar{c}_{i-k,i} s_{i-k}(t-T) = \sum_{k_0 \leq k} \bar{p}_{i-k,i} \bar{c}_{i-k,i} s_i\left(t-T+\frac{k\lambda}{V}\right).$$

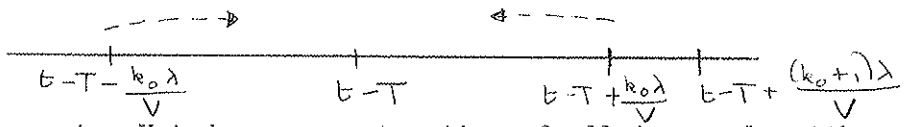
This computation is not essentially changed when we replace s_{i-k} by $\max(s_{i-k}-\bar{\tau}_{i-k},0)$, a nontrivial threshold, just so long as $\bar{\tau}_{i-k}=\bar{\tau}$ for all $i-k$. In such a field, $T-k_0\lambda/V$ time units after p_i is activated by an input, it receives its first self-inhibitory input. Even as $V \to \infty$, $T-k_0\lambda/V$ remains bounded. No matter how quickly the input pulse flows, it will be inhibited at every point within a finite time interval, and the length of all such intervals is bounded above. Moreover, if I_i is taken to be so large that s_i's initial input-induced growth is rapid, the individual inhibitory terms will also increase, so that s_i cannot maintain its initially large size indefinitely. The exact determination of the rate with which s_i is inhibited depends critically on the choice of the $\{ \bar{p}^{\pm}_{i+k,i} \}$ distribution. The fact that staggered s_i terms enter in the self-inhibitory sum insures that the individual inhibitory transmissions are of the proper order of magnitude to suppress s_i, but the slope of the s_i function as it is being suppressed, and in particular the question of whether or not s_i approaches P_{eq} in a smooth and monotone way, depends on $\{ \bar{p}^{\pm}_{i+k,i} \}$. Questions such as the choice of $\{ \bar{p}^{\pm}_{i+k,i} \}$ require a study of the asymptotic stability of nonlinear difference-differential equations. In these introductory qualitative remarks, we simply assume that the line densities have been properly chosen to assure the desired asymptotic behavior of s_i.

The interval between successive self-inhibitions from the past field is λ/V, and decreases as the velocity of the input wave front increases. Increasing V is equivalent to dilating the interval between input onset and the first self-inhibition like $T+k_0\lambda/V$ and to contracting the intervals between successive self-inhibitions like λ/V. The total inhibitory term is proportional to

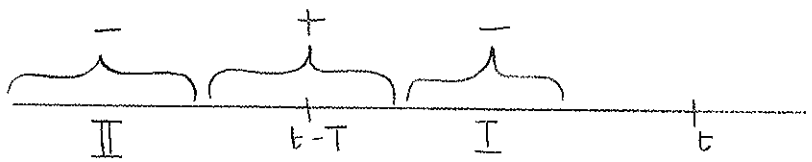
$$\Sigma^{-}_{past} + \Sigma^{-}_{future} =$$

$$\sum_{k>0} p^{-}_{i-k,i} c^{-}_{i-k,i} \max(s_i(t-T+k_o\lambda/V) - \tau^{-}, 0) + \sum_{k>0} p^{-}_{i+k,i} c^{-}_{i+k,i} \max(s_i(t-T-k_o\lambda/V) - \tau^{-}, 0).$$

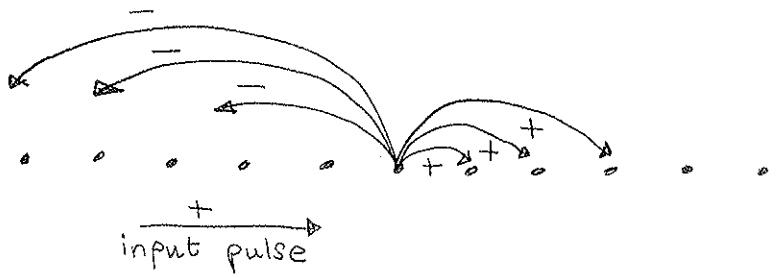
When $\tau^{-}=0$ in the Σ^{-}_{future} component, successive self-inhibitions once again contract like λ/V with increasing V, but the interval between input onset and the first self-inhibition now decreases like $T + k_o\lambda/V$. Graphing the self-inhibitory onset times



we see that increasing V induces a contraction of all intervals with respect to t-T. Considering t-T as the zero point of the graph, the entire time scale is contracted by a factor varying like 1/V. We have, moreover, a diagram of the form



The (+) bracket denotes the region of times which contribute self-excitations to each p_i, while the (-) brackets denote the regions of times which contribute self-inhibitions to p_i. In general, an excitatory input pulse if followed by a sequence: self-inhibitory wave ⟶ self-excitatory wave ⟶ self-inhibitory wave. The first inhibitory wave (I) is derived from the future field of any point. We are however considering a field which has almost realized the linear asymptote $(c^{+}_{i,i+1})^* = 1$. In such a field, it is expected that the $(c^{-}_{i,i+1})^*$ values behave in a mirror-image way to the $(c^{+}_{i,i+1})^*$ values, for s^{+} stimulates $c^{+}_{i,i+1}$ and suppresses $c^{-}_{i,i+1}$ growth. The marked forward bias $(c^{+}_{i,i+1})^* \approx 1$ in the c^{+} field must therefore be matched by a marked backward bias in the c^{-} field. We have, therefore, an asymptotic diagram of the form



The total self-inhibitory term is thus proportional to

$$\Sigma^{-}_{past} + \Sigma^{-}_{future} \approx \Sigma^{-}_{future}.$$

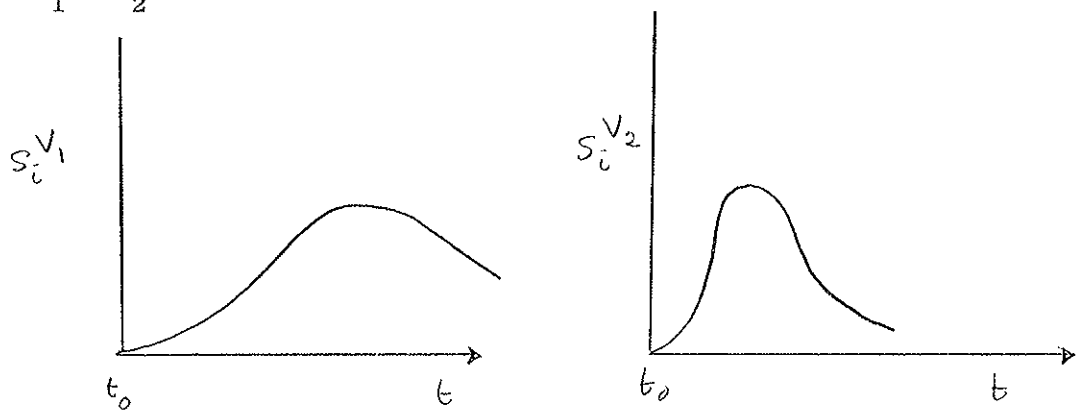
This asymptotic splitting of past and future fields into excitatory and inhibitory components has the desirable effect that the input pulse induces precisely one excitatory wave at each point, and this wave is then followed by precisely one inhibitory wave. This sequence of pulses is exactly what one desires for $(c^{+})^{*}$ invariance. The effective total inhibitory component, Σ^{-}_{future}, also contracts the time scale of successive input onsets in a manner not exhibited when the term Σ^{-}_{past} is dynamically significant.

97. Relativistic Contractions

Does varying V change the effectiveness of the input pulse at sustaining c values? We can admit pulses with varying velocities whose effects on c sustanence are not identical only if we also require that the fraction of pulses with a given velocity which travel through all fields that interact with one another in an identical way is the same. We must also require that the number of admissible velocities decreases as the differences in c sustaining power of inputs at different velocities increases. It is nonetheless clearly of great advantage to avoid such historically bound averaging effects--which are difficult to realize in an actual construction of a dual field--by providing dual fields that automatically produce input pulses all of which have equivalent effects on the sustanence of c values. Such fields thus impose a condition on each input flow rather than a statistical condition on the entire ensemble of admissible flows. We call this condition the local invariance of input velocities. Locality is here understood with respect to an entire input flow, considered as a single point in the space of all such possible flows. The condition of locality imposes immediate restrictions on the possible inputs.

To study these restrictions, we introduce the following notation. Suppose that $I=I(\{p_i\},t)$ is a sustaining input for $\mathcal{T}=\mathcal{T}(\{p_i\})$ with velocity V. Denote I by I^V. Suppose that the velocity of V is varied within an admissible range, say the interval $[V_o,V_1]$. Let I^W be the input corresponding to I^V in this variation whose wave velocity is $W\in[V_o,V_1]$. How does I^V vary with V? The two facts which guide us in determining this are: (1) all I^V, $V\in[V_o,V_1]$ are equally good at sustaining $C(\mathcal{T})$, and (2) increasing V contracts the time scale of self-inputs like $1/V$ in a field that is asymptotic to a chain or a loop. Consider the set $\{\mathcal{T}^V: V\in[V_o,V_1]\}$ of copies of $\mathcal{T}$ such that I^V is delivered to $\mathcal{T}^V$. Fix attention on a single p_i, denoted by p_i^V, in $\mathcal{T}^V$, and suppose that the time is scaled so that all I^V have their p_i^V onset times at t_o. We suppose for specificity that each I_i^V is chosen so that s_i^V is monotone increasing in the interval $[t_o,t_o+T+k_o\lambda/V]$.

If $V_1<V_2$ and $s_i^{V_1}\big|_{[t_o,t_o+T+k_o\lambda/V_2]}\equiv s_i^{V_2}\big|_{[t_o,t_o+T+k_o\lambda/V_2]}$, then $s_i^{V_2}$ cannot sustain c values as well as $s_i^{V_1}$ can. For after time $t=t_o+T+k_o\lambda/V_2$, $s_i^{V_2}$ will immediately receive a succession of self-inhibitory inputs that will rapidly diminish $s_i^{V_2}$. $p_i^{V_1}$ on the other hand has a further time interval of length $k_o\lambda(V_2-V_1)/V_1V_2$ of purely excitatory inputs, and its self-inhibitory inputs will arrive at a slower rate than those of $p_i^{V_2}$. In order to correct this difficulty, $s_i^{V_2}$ must grow under $I_i^{V_2}$ more quickly than $s_i^{V_1}$ grows under $I_i^{V_1}$. This more rapid growth of $s_i^{V_2}$ is counterbalanced by a more rapid succession of self-inhibitory inputs, each of which also grows more rapidly. The rapid rise of $s_i^{V_2}$ is thus followed by a rapid fall. The total effect is a peaking of the s_i^V function as V increases. We therefore have the qualitative diagrams for $V_1<V_2$:



Such an effect induced by increasing V is called a relativistic contraction of s_i^V, since it depends on the relative dimensions of the time scales of $\mathcal{F}$ and of the input wave. Relativistic contractions, just like CPT embedding field symmetries, are a direct consequence of c* invariance. These contractions have been derived from dynamical, and not purely kinematical, considerations, as one customarily does. Indeed, c* invariance itself is but a statement that our field extensions have been chosen in a coherent way relative to the choice of local dynamical densities.

98. Temporal Self-Similarity

The contraction of successive self-inputs like 1/V as V increases suggests that the entire time scale of dynamical interactions contracts when we let V increase. If we let s^V be the s function generated by I^V, this suggestion assumes the form of a condition like

$$f(V)s^V(g(V)t)=f(W)s^W(g(W)t)$$

where V and W are admissible velocities and f and g must be determined. As V varies, the transmission thresholds remain fixed, and all of the dynamical effects which $\mathcal{F}$ has on other fields is determined by transmission from $\mathcal{F}$. Moreover, increasing V contracts successive self-inputs like 1/V. These two facts suggest the choice g(V)=1/V. Whenever

$$f(V)s^V(t/V)=f(W)s^W(t/W)$$

holds for all admissible V and W, we say that the system $\oplus_V\mathcal{F}^V$ is temporally self-similar.

The introduction of temporal self-similarity into our considerations is accompanied by an immediate difficulty, for the embedding equations possess a locally determined time scale. The transformation $t \longrightarrow t/V$ induces the transformation

$$ds^V(t)/dt \longrightarrow V\cdot ds^V(t/V)/dt.$$

The premultiplying V in $Vds^V(t/V)/dt$ is not, however, compensated by a V on the right-hand side of the equation for s^V. Consequently, all rate factors are multiplied by 1/V under $t \longrightarrow t/V$, although they are supposed to represent the rates

of local processes which are independent of the rate of flow of the input pulse. This holds true, in particular , for the spontaneous growth and decay processes that are needed to satisfy $\lim_{t \to \infty} s(t) = P_{eq}$ in a free embedding field. $t \longrightarrow t/V$ thus does not leave the embedding equations invariant. Nonetheless, our intuitive remarks about variations in the rate of performing acts suggest strongly that some analog of temporal self-similarity does hold in many situations. Since temporal self-similarity breaks down when we consider the local equations for individual points, self-similarity, or some analog thereof, must be achieved by the intervention of compensatory effects in the behavior of ensembles of points. In particular, the existence of invariant transmission time lags, which cause immediate difficulty in considering any temporally self-similar system, must be overcome by a suitable choice of transmission threshold distribution, distribution of transmission lags, and other geometrical parameters. Since our experience with ensembles of points has thus far been quite limited, we will first turn to less complicated gometrical questions which will later aid in constructing a field that exhibits useful relativistic contractions. Although such constructions are themselves not completely obvious, it should be clear that the intuitive motivation to seek them is almost immediately evident. Notice, finally, that c* invariance suggested the local invariance of input velocities, which is a transition from a consideration of input ensembles to input singletons. Yet this local invariance, together with c* invariance, suggested temporal self-similarity, which itself can be realized only by passing from cellular singetons to cellular ensembles. Some form of ensemble behavior is therefore needed to preserve desirable properties in field situations of this type, and our principles quite effortlessly direct our attention to which ensembles are best suited for the realization of a given intuitively compelling property.

99. Ergodicity and Sustaining Inputs

Before turning to such geometrical questions, we now sketch more completely an earlier train of thought. We earlier saw in our consideration of sustaining inputs that a linear or cyclic line asymptote in a homogeneous field requires an input pulse that follows the direction of the asymptote in a translation stable fashion. Similarly, it is easy to see that in a homogeneous field with

points $\{ p_i \}$, any set of input functions $\{ I_i \}$ such that $I_j(t)=I_k(t)$ for all j and k maximally sustains the field's homogeneity. In particular, translation stable flows are no longer called for and even a quiescent strength field does not violate c* invariance, for all local line distributions are identical. The existence of globally homogeneous fields is perhaps best realized in the brain of an infant, and indeed rhythms in such brains are not so well developed as in the brains of adults. Some rhythms are, however, present, as are those structural inhomogeneities without which a higher neural life could never even begin. As the child's experience increases, homogeneous fields become increasingly asymmetrical and brain rhythms become more pronounced.

Another simple example of sustaining inputs arises when the point set $\{ p_i \}$ is partitioned into subsets P_i such that all field functions are factored by $\oplus_i P_i$. c* invariance is here best served by an input paradigm that itself factors $\oplus_i P_i$. If we now piece together various point sets whose lines approach homogeneous, or linear, or cyclic, or factorable asymptotes, and perhaps intermediate structures, we will expect to find a sustaining input array which at various times is homogeneous, or translation stable, or factorable, or some mixture of these effects. It is easy to imagine that the waves and eddies of point strength produced in this fashion can become extremely complex. Nonetheless, the underlying principle of c* invariance, realized by local walks guided by interactions between a field and its dual fields, brings to the student of these effects a harmonious perspective.

The above discussion shows that a kind of ergodic behavior is found under a sustaining input paradigm. One sense in which ergodicity here holds is that the line densities, which contain the structural content of the field, determine the average strength distributions, which represent the more rapidly fluctuating temporal field factors. In particular, a point on which a large number of lines with high line densities end will be expected to possess a larger average strength function through time than a point that receives only a small number of lines with low line densities, if renormalization properties are the same for both points. The successive input onsets, in turn, may be viewed as a generalized transition operator that operates on the "states" represented by the field points. The distribution of input onsets depends critically on the distribution of line densities. In particular, two subsets of points which are practically disconnected and share dual fields that project to them with only low intensity will exhibit input transitions that are pairwise

more independent than those found for two subsets which share strong direct connections, each subset interacting strongly with common dual fields.

Since the sustaining input situation is the natural analog of the steady- or stationary-state of an embedding field, we see that an ergodic hypothesis holds for the stationary behavior of these fields. This hypothesis takes on a more classical aspect in fields for which an availability diagram, and thus a bona fide transition structure, is valid. Viewed in the reverse order, the sustaining input situation provides a generalization of the notion of ergodicity, from those systems that possess an availability diagram and an underlying dynamics whose densities can be represented by a form of embedding equation, to systems for which the input paradigm is too complicated to permit the use of availability diagrams. Moreover, the same local dynamical equations which hold in the stationary situation are equally valid in the non-stationary situation, for which new ensemble interactions are introduced.

100. Asymptotic Translation Stability

These discussions of sustaining inputs, in particular of translation stable inputs, have covertly assumed an important property that follows by properly choosing the field constants. This property becomes apparent if we consider a large homogeneous embedding field with weak initial line structure such that an indefinitely long sequence of identical inputs is delivered to different points at a linear rate W starting at time zero. Order the points according to the number of the input they receive; thus p_n is the point receiving the n^{th} input. For $t \leq (n-1)W$, all functions of the same type that are associated with pairs of points p_k, $k \geqslant n$, will agree by symmetry; for example, $s_n(t)=s_{n+1}(t)$, $t \leq (n-1)W$. If n is taken sufficiently large, a situation will be approximated for which all functions associated with p_n and evaluated at times $t \geqslant (n-1)W$, will have almost the same values as the functions associated with p_{n+1} evaluated at time t+W; for example, as n becomes large, $s_n(t)-s_{n+1}(t+W) \longrightarrow 0$, $t \geqslant (n-1)W$. This condition simply means that the initial asymmetries which perforce arise when the input sequence begins will have almost completely faded out by the time $t \geqslant (n-1)W$ and large n are reached. Dynamically speaking, the input sequence begins effectively at $t = -\infty$ for points p_n with such n. We call this property asymptotic translation stability.

Asymptotic translation stability holds also for multiple lines. Let $\Gamma_i(I,t) = \prod_{k\in I} s_k(t-t_{ki})$, $t_{ki} < W$, where I is any finite set of point indices. Then as $\min(k:k\in I)$ becomes large, $\Gamma_i(I,t) - \Gamma_i(I+1,t) \longrightarrow 0$ for $t \geqslant (\min(k:k\in I))W$. Here $I+1=(p_{i_1},\ldots,p_{i_k})+1=(p_{i_1+1},\ldots,p_{i_k+1})$. Let $\bar{n}$ be the index of the last point to which an input has been delivered and let $m(I,\bar{n})=\min(\bar{n}-k:k\in I)$ if $k \leq \bar{n}$, $k\in I$, and $+\infty$ otherwise. Whenever reference is made to $\Gamma_i(I,t)$, it is understood that the discussion applies only to values of t for which $|t-\bar{n}W|$ is small, for example, $|t-\bar{n}W| \leq qW$, where q is a small integer depending on the field constants. We shall watch only the local behavior of the system as it evolves in time.

Cause $\bar{n}$ to become large. For those I such that $m(I,\bar{n})$ is large, $\Gamma_i(I,t)$ will be small as a result of both extensive local point strength decays at points p_k for which $m(p_k,\bar{n})$ is large and the poor transmission that results from the weak line structure. Thus the dynamical contributions of these 1_{Im} may be ignored without great error. Indeed a finite k can be chosen such that all $\Gamma_i(I,t)$ with $m(I,\bar{n}) \geqslant k$ may be safely discarded. For the $\Gamma_i(I,t)$ which are retained, a uniform asymptotic translation stability is achieved.

Corresponding to this translation stability, a comparable translation stability is induced in the functions c_{Ik}. Under these stability conditions, it is clear that within the class of sets I of fixed size, the "connected" sets $I=(p_i,p_{i+1},\ldots,p_{i+k})$ will have the largest c_{Iw} values for fixed w in a homogeneous field. In serial verbal learning, for example, such a situation is approximated for the items in the middle of a very long serial list.

Asymptotic translation stability, which is the closest one can come to translation stability in a purely excitatory field, follows directly from the requirement that the field functions in the free embedding field converge to their equilibrium positions. We can only conceptually deliver sustaining translation stable input pulses originating at $t=-\infty$. Under asymptotic translation stability, such pulses can, however, be rapidly approximated in finite time.

101. Free Associations and Spontaneous Emission

A situation which is a supraliminal analog of a sustaining input paradigm is realized in the situation of free association. Here a subject is

asked to emit strings of words which he considers to be natural associates of a given word, or he merely emits strings of verbal forms chosen from a fixed psychological set; for example, individual letters or numbers, in a spontaneous way. In the case of free associates, the presentation of a word is realized as an experimental input to the appropriate collection of field points. Transmission from these points renormalizes the field until the response criterion is realized. An associate is emitted and a feedback input is delivered by the vector input function that represents this associate. Transmission occurs from these newly excited points, the field is again renormalized until the response criterion is again satisfied, another associate is emitted and a new feedback vector input function is applied. The process continues in this fashion. Whether or not the subject constantly has in view the original input makes an important difference, which is closely tied to the difference between correction vs. non-correction asymptotes. When the input is constantly in view, the subject must produce new associates to the original input and must reject old associates. When the original input is not in view, the subject more properly produces associates to the last (few) associates which he has offered, and the situation of a local walk is much more closely approximated.

In both the sustaining input situation and the case of free associations in strings, the renormalization pattern is strongly guided by the localline distribution. In the free associate situation, a new consideration is introduced since the presentation of frequent feedback inputs helps to strongly contract field dynamics to those associates directly connected with the last associate that was emitted. In the sustaining input situation, by contrast, the dynamical flows between individual points can much more easily decompose into local strength eddies after passing through several stages of points. Feedback inputs are quite necessary to prevent the dynamical trajectories associated with macroscopic behavior from decomposing into local sub-trajectories which have no macroscopic significance. The importance of feedback inputs to the stabilization of embedded forms is familiar from clinical studies of individuals who have suffered partial transections of specialized sensory or motor relay fibers.

In the case of spontaneous emission of units from a fixed psychological set, the response paradigm is guided by the same principles governing free associations, but sometimes presents a slightly different appearance in detail. Indeed, spontaneous emission from a psychological set is just an expression of the

conditioning of one's free associations to a subset of the total field. Suppose for simplicity that the psychological set is a homogeneous field. Fix attention on some p_i. Let $P_{ij}^{(k)}$ be the point which is emitted j units after p_i is emitted for the k^{th} time. Let $w_{ij}^{(k)}(u)$ be the average number of times for which $p_u \in \{ P_{ij}^{(r)} : r \leq k \}$. That is,

$$w_{ij}^{(k)}(u) = \frac{\sum_{r=1}^{k} X_u(P_{ij}^{(r)})}{k}$$

and

$$X_u(p_m) = \begin{cases} 1 & \text{if } m=u \\ 0 & \text{if } m \neq u \end{cases} .$$

By homogeneity, we expect that as k becomes very large,

(i) $w_{ij}^{(k)}(u) \sim w_{ij}^{(k)}(v), \qquad u,v \neq i,$

(ii) $w_{ij}^{(k)}(u) \sim w_{mj}^{(k)}(u), \qquad i,m \neq u.$

These conclusions are derived from the following familiar facts about homogeneous fields:

a) When an isolated input is delivered to p_i in a homogeneous field $\mathcal{T}(p)$, all points $p \setminus p_i$ to which the input was not delivered share an equivalent dynamical role, and the role of $p \setminus p_i$ is the same whatever p_i is chosen. The p_i to which the input is delivered is distinguished from $p \setminus p_i$ not only by direct differences in its strength functions. In addition, the production of p_i as a response to an input to p_i requires an asymmetrical consideration of multiple lines and, hence, also the possibility of asymmetrical subject strategies.

b) By asymptotic translation stability, we know that the effects of several successive inputs are short-lived. It does not follow from this that if a point has very recently received a feedback input it will be just as available for a new feedback input as a point which has never been emitted. We may, however, conclude the following. Suppose, for example, that $\mathcal{T}(p)$ is much larger than the set of points $\mathcal{G}_t(p)$ which at any time t are distinguished from $\mathcal{T}(p) \setminus \mathcal{G}_t(p)$ by the fact that the effects of recent feedback inputs have not yet worn off in $\mathcal{G}_t(p)$. Asymptotic translation stability and the homogeneity of $\mathcal{T}(p) \setminus \mathcal{G}_t(p)$ relative to the determination of new feedback inputs imply that we can asymptotically look at $\mathcal{G}_t(p)$ just as we look at $\mathcal{T}(p_i)$ when an isolated input is delivered to p_i. In particular, the number of points in $\mathcal{G}_t(p)$ at a fixed time interval after the last feedback input is delivered will stabilize as $t \to \infty$,

and each p_i will occur in some $\mathcal{Z}_t(p)$, $t \in [0, \infty)$, with equal frequency. Since input dependencies only survive over finite time intervals, say of order Q, the set $\mathcal{Z}_t(p)$ will seem to wander randomly within $\mathcal{T}(p)$ over time intervals that greatly exceed Q. All input dependencies will therefore average out but those that depend on which point received the last input, and (i)-(ii) follow.

102. Response Clustering

The net effect of this wandering of $\mathcal{Z}_t(p)$ is that verbal units seem to be emitted in clusters of finite length when considered from the purview of which points receive inputs independent of other points. Only the points in such a cluster seem to receive new inputs in a manner determined by the past input paradigm. The length of these clusters depends directly on the rates of decay that produce asymptotic translation stability. All units that are not in such a cluster at a given time receive the next feedback input with equal probability, while delivering an input to a point within a cluster can depend on the order in which the last few inputs arrived. In particular, the last point to receive an input is in a privileged position. We can easily imagine subjects whose strategy is simply to repeat one unit over and over again. When such a strategy is forbidden, we can just as well imagine that a subject will covertly reject all $\mathcal{Z}_t(p)$ points in favor of $\mathcal{T}(p) \setminus \mathcal{Z}_t(p)$ points. In spite of these covert individual differences, we can generally expect the existence of shifting finite clusters of dependent units and conditions (i)-(ii) whenever $\mathcal{T}(p)$ is homogeneous.

Another type of clustering effect occurs if the subject tries to emit units very rapidly. In this case, the number of points with high strength values will rapidly increase until the response criterion can no longer be satisfied. A period of silence will then follow until these strength values subside and response-inducing inputs can effectively be resumed. The net effect is one of temporal bursts of short clusters of units followed by silent pauses that precede new bursts, and so on.

In an inhomogeneous field, the remarks on free associates and on spontaneous emission from homogeneous psychological sets may be combined to create a picture of clusters of responses which pattern themselves according to those local line distributions that are activated by recent inputs.

103. Spontaneous Rhythms

A question which must not be overlooked even in these brief sketches of input-output dependencies is the following one. If we require that all field functions converge to their equilibrium values in a free embedding field, how can sustaining inputs be realized over periods of many hours without serious decrement, say during sleep? We have only two kinds of alternatives in answering this question. On the one hand, these sustaining inputs might be induced by proprioceptive inputs from the various muscle groups. Such "externally" induced inputs cannot sustain the great bulk of embedded forms, however, for these depend on the global coordination of many muscle groups. If this alternative is not wholly satisfactory, we must look to the other hand, on which lies the alternative that certain cells have the capacity to spontaneously generate potential changes and transmissions. These transmissions would provide the necessary background excitations from which would be molded sustaining input patterns. Such a possibility violates the requirement of asymptotic equilibrium in free embedding fields in the sense that no embedding field could ever be truly free. The imposition of this requirement was directed at preventing infinitely sustained high strength values both from (i) homogenizing line asymptotes, and from (ii) interfering with the unbiased reception of new inputs. Can we generalize the requirement of asymptotical equilibrium without violating (i) and (ii)?

The answer is yes. To preserve (i), we must require that the points which generate spontaneous transmissions are (a) incapable of generating broad excitatory strength distributions, at the same time that (b) they project to so many points that the spontaneous background excitation is useful for the generation of sustaining inputs on a global scale. The simplest way to achieve (b) is to locate these points in dual fields that project to many cortical points. (a) is achieved by endowing these points with rich lateral inhibitory structures that tend to suppress the activity of nearby points. A situation in which both of these conditions are satisfied is found in the localized complete sets of antagonists which we postulated for the renormalizing "thalamus" with its inhibitory interneurons. Suppose for simplicity that the spontaneously transmitting points are subjected to a locally determined process governed by random excitatory increments, and that the parameters of this process are homogeneous across points. Suppose, moreover, that the lateral inhibitory structure is also homogeneous across points.

In this situation, whenever the phase of the self-excitation of one point gets ahead of the excitation of the other points, the inhibitory transmission induced by this point will suppress the excitatory increments of all points to which it sends inhibitory lines. The inhibited points will then begin to accumulate new excitatory increments at a uniform rate. The existence of broad lateral inhibitory transmission and a homogeneous rate of random excitation thus implies that subsets of points will begin to act in unison. Since points have the alternative of either being excited or inhibited, the effect of this joint action is often the emergence of collective periods of excitation followed by collective periods of inhibition, followed by collective periods of excitation, and so on. Spontaneous rhythms hereby emerge among points that are mutually connected. These rhythms are obviously well-suited for interaction with the cortex to produce effective sustaining inputs.

If these spontaneous rhythems are truly determined by local processes, then we must add a random input term to the strength functions generating these rhythms whose source must eventually be sought in a local metabolic process. To the extent that points in dual fields share a common local dynamical process with other types of points, these latter points must also exhibit spontaneous fluctuations from equilibrium and appropriate provisions for the satisfaction of (i) and (ii). Moreover, if these spontaneous rhythms are indeed a major source of sustaining input excitation in adults, then the relatively less extensive rhythmic activity in young children suggests that the lateral inhibitory structure of the thalamus develops in parallel fashion with cortical development. When the cortex itself displays local lateral inhibitory lines, say of the "on"-"off" variety, such a parallel development of cortex and thalamus is exactly what we would expect from our earlier observation that $7 \oplus \hat{7}$ behaves both geometrically and dynamically like a single unit when questions of sustanence are entertained. We will later study this fact from a deeper point of view, indeed from the perspective of local cellular control processes, once we have introduced enough structure to be able to distinguish the roles of the various parts of the thalamus.

104. EEG Suppression

If spontaneous rhythms are produced by dual fields, how can (ii) be satisfied? The only way is to postulate the existence of inhibitory lines which are activated at an early, pre-cortical stage of stimulus processing and which project to the source of these rhythms. The presentation of new stimuli to a field which displays typical EEG rhythms should therefore momentarily suppress the EEG, or at least mask the EEG with low intensity inputs that are out of phase with one another. By the time the stimulus-induced inputs reach those cortical points which are directly involved in producing line residues that record the inputs, the biases introduced by the spontaneous rhythms will have been suppressed or at least uniformized. This suppression process is a delicate one, since we do not want the dual points to be so heavily suppressed that they cannot participate in the renormalization of the inputs after they reach the cortex. One simple way to avoid this difficulty is to require that both inhibitory and excitatory lines project to the dual points from early sensory-processing nuclei. The inhibitory contribution arrives first but is rapidly followed by a compensatory excitatory input. More subtle ways of removing the difficulty can easily be imagined if the dual field is made more complicated. For example, suppose that the dual field is split into two parts, $\hat{\mathcal{F}}_1$ and $\hat{\mathcal{F}}_2$, such that the $\hat{\mathcal{F}}_1$ projects to both $\mathcal{F}$ and $\hat{\mathcal{F}}_2$, $\hat{\mathcal{F}}_2$ plays the role of the old $\hat{\mathcal{F}}$ by exhibiting reciprocal interactions with $\mathcal{F}$ that renormalize $\mathcal{F}$, and $\hat{\mathcal{F}}_1$ is the primary source of spontaneous excitations. For this situation, we need only send inhibitory lines from early, pre-cortical sensory processing stations to $\hat{\mathcal{F}}_1$. The spontaneous excitations in $\hat{\mathcal{F}}_2$ and $\mathcal{F}$ will rapidly subside when $\hat{\mathcal{F}}_1$ is inhibited, but $\hat{\mathcal{F}}_2$ will remain free to renormalize the inputs to $\mathcal{F}$ without danger of prolonged suppression. For such a dual field $\hat{\mathcal{F}}_1 \oplus \hat{\mathcal{F}}_2$, $\hat{\mathcal{F}}_1$ will be most active when $\mathcal{F}$ is least perturbed by external inputs, while the active perturbation of $\mathcal{F}$ by external inputs will tend to suppress the activity of $\hat{\mathcal{F}}_1$. The fields $\mathcal{F}$ and $\hat{\mathcal{F}}_1$ will thus exhibit periods of reciprocal activity. The exhibition of more realistic dual fields will bring us close to neuroanatomy proper. Conversely, a thorough study of neuranatomy reveals the particular choice of dual fields which evolution has wrought.

105. Equal Input Densities ⟶ Equal Output Densities

We now turn to an important problem concerning the conditioning of ensembles of points. When a large set of points is collectively responsible for the performance of a given act, it is often important that the conditioning inputs to each of these points be so arranged that all of the points reach threshold and begin contributing to the performance of the act, through their transmissions, at the same time. One can think of the control of large collections of muscle groups for heuristic motivation. This condition imposes an immediate restriction on the possible distribution of thresholds of the points considered.

Let $I_i(t)$ be the conditioning input delivered to p_i. For simplicity, suppose that

$$I_i(t) = \begin{cases} I_i & t \in [0, \infty) \\ 0 & \text{otherwise} \end{cases}$$

Since none of the p_i transmit unless all of them do, we find, for example, in a simple purely excitatory field that

$$ds_i/dt = \alpha^+(M_i - s_i)I_i - \alpha^- s_i$$

before transmission occurs, whence if $s_i(0)=0$ for all i,

$$s_i(t) = \frac{\alpha^+ M_i I_i}{\alpha^+ I_i + \alpha^-}\left(1 - \exp\left[-(\alpha^+ I_i + \alpha^-)t\right]\right).$$

We want all s_i to reach their transmission threshold τ_i at the same time T. Thus,

$$\tau_i = \frac{\alpha^+ M_i I_i}{\alpha^+ I_i + \alpha^-}\left(1 - \exp\left[-(\alpha^+ I_i + \alpha^-)T\right]\right).$$

If we let $I_i = I$, for all i, be admissible inputs, then

$$\tau_i/\tau_j = M_i/M_j,$$

or

$$\tau_i = \mu M_i, \quad \mu \in (0,1), \text{ for all } i.$$

Since each p_i is a volume conductor, the condition $I_i = I$ may be interpreted to mean that every p_i receives an equal conditioning excitation density. Similarly, $\tau_i/M_i = \mu$ means that the excitation density required for transmission to occur is the same for all p_i. The content of the requirement of unbiased onset of transmissions across points is thus that equal input excitation densities induce equal output excitation densities. This fact, which arises from a very simple

computation, is nonetheless important, for it demonstrates that nonuniformities in the onset of transmissive events closely parallel nonuniformities in the distribution of excitation densities, given the proper choice of transmission thresholds.

106. Equal Exchange

Let us now turn to a special determination of the p_{ij} functions. In particular, suppose that p_{ij} depends only on M_i: $p_{ij}=p(M_i)$. Suppose also that the distribution of lines is such that every point receives an equal average input from the other points over time and that the inputs to the system are randomly distributed. We say that a system exhibits equal exchange when this is true. If the total number of points with every M_i value is the same, then the fact that the time average excitiation of each s_i and c_{ij} is proportional to M_i and A_{ij} shows that $\sum_{j\neq i} M_j p_{ji} A_{ji}$ is independent of i. A simple realization of this condition is

$$p_{ji} = p/M_j A_{ji}, \qquad p > 0.$$

Let us now further suppose that the size of each A_{ji} is completely determined by the size of M_i. In particular, suppose that $A_{ji}=AM_i$. Then

$$p_{ji}=(p/A)1/M_jM_i = \gamma/M_jM_i, \qquad \gamma=p/A.$$

107. Local Densities and Global Constraints

What happens when we substitute the values $\tau_i = \mu M_i$ and $p_{ji} = \gamma/M_jM_i$ into the embedding equations? In the simplest purely excitatory fields, for example, we find, dividing the equation for s_i by M_i and the equation for c_{ij} by A_{ij} that

$$\frac{d\left(\frac{s_i}{M_i}\right)}{dt} = \alpha^+\left(1-\frac{s_i}{M_i}\right)\left(\tau p \sum_j \max\left[\frac{s_j}{M_j}(t-t_{ji})-\mu, 0\right]\left(\frac{c_{ji}}{A_{ji}}\right)+I_i\right)-\alpha^-\left(\frac{s_i}{M_i}\right),$$

$$d\left(\frac{c_{ij}}{A_{ij}}\right)/dt = \beta^{+}\left(1-\frac{c_{ij}}{A_{ij}}\right)\max\left[\left(\frac{s_i}{M_i}\right)(t-t_{ij})-\mu, 0\right]\left(\frac{s_j}{M_j}\right)$$

$$-\beta^{-}\left(\frac{c_{ij}}{A_{ij}}\right)\left(1-\max\left[\left(\frac{s_i}{M_i}\right)(t-t_{ij})-\mu, 0\right]\right)\left(1-\frac{s_j}{M_j}\right),$$

where $\beta^{+} = \text{urr}\hat{\gamma}$ and $\beta^{-} = \epsilon\rho\hat{\rho}\gamma$. Here we have assumed that the threshold for the process coupling s_j values to the $\bar{N}_{ij}$ complex is zero for simplicity. Letting $\mathcal{A}_i = s_i/M_i$ and $\mathcal{C}_{ij} = c_{ij}/A_{ij}$, these equations become

$$d\mathcal{A}_i/dt = \alpha^{+}(1-\mathcal{A}_i)\left(n_p \Sigma_j \max\left[\mathcal{A}_j(t-t_{ji})-\mu, 0\right]\mathcal{C}_{ji} + I_i\right) - \beta\mathcal{A}_i,$$

$$d\mathcal{C}_{ij}/dt = \beta^{+}(1-\mathcal{C}_{ij})\max\left[\mathcal{A}_i(t-t_{ij})-\mu, 0\right]\mathcal{A}_j$$

$$-\beta^{-}\mathcal{C}_{ij}\left(1-\max\left[\mathcal{A}_i(t-t_{ij})-\mu, 0\right]\right)\left(1-\mathcal{A}_j\right),$$

which are equations for local excitation densities. A special form of (i) equal exchange, and (ii) the transformation of equal input densities into equal output densities thus imply that (iii) the dynamical equations are local. Moreover, whenever (iii) holds, we can choose our geometrical constants in such a way that (i) and (ii) also hold. (i) and (ii) are global conditions on the system of points and express the fact that the global interactions exhibit a kind of unbiased stability in their average behavior. The fact that (i)-(ii) $\Longleftrightarrow$ (iii) may thus be interpreted in the following way. Given a system of local dynamical densities, if we want to be able to extend this system geometrically in a homogeneous way (namely, as volume conductors), then we must impose global conditions like (i)-(ii) on the interactions of all points which we so extend. Put in a heuristic way: if we want to be able to completely describe these systems by looking only at their smallest parts, we must bind together even the most far-flung interactions into a tight geometrical unit.

108. Effective Surface Area

In order to better understand this situation, we now consider each p_i separately. M_i is a measure of the effective surface area of p_i. Effective surface area is the area of that region of the cell surface which actively receives transmissions from arborizing endbulbs, etc. Since all recipient regions do not necessarily receive equal excitation densities from their impinging endulbs, the computation of effective surface area must weigh each recipient region according to its relative sensitivity to the excitation received after a standard transmission burst. This area can then be conceptually contracted to a set of receptive regions all of which do respond with equal sensitivity to transmission pulses of equal excitation density, using the spatial homogeneity of the local laws governing the interneuronal transmission process. The theoretical situation is particularly simple when effective surface area and cellular volume are proportional, as Bok's work suggests.

Suppose that the length of the lines which p_i sends to other points are proportional to M_i. Thus, when $M_i = M_j$ for all i and j, $p(M) = \mu/M^2$ varies as the inverse square of the distance between two points. Also suppose that the diameter of these lines is proportional to M_i and that the velocity of transmission over any line is proportional to its diameter. The transmission time over any line is thus independent of M_i, and these points generate the smallest possible minimal fields. By (iii) and the setting of the diameters, the transmission threshold $\tau_i = \mu M_i$ means that the excitation density along the initial segment of the diameter of each line must reach μ before transmission spikes are generated over the line. Moreover, a line of diameter M_i generates a transmission whose total effect on its recipient point would be proportional to M_j if p_{ji} were itself independent of M_j. The choice of p_{ji}, however, just cancels the size of M_j, so that every point contributes transmission densities independent of its size to the other points.

To see more clearly how this happens, we notice also that when the nodes N_{ij} reach p_j, they are encouraged to grow until their size is proportional to M_j. The growth of N_{ij} is determined by the functional contact which N_{ij} makes with the p_j membrane. If A_{ij} stabilizes at a value proportional to the effective surface area of N_{ij} --that part of the surface of N_{ij} that actually participates in generating transmissions to p_j-- then the fact that p_{ij} is proportional to $1/M_j$ means simply that the excitation transmitted to N_{ij} from p_i is uniformly distributed along the effective surface area of N_{ij} after it reaches N_{ij}. If we continue to

argue in this way, the fact that p_{ij} is proportional to $1/M_i$ ought to mean that the effective area of N_{ij} is also proportional to the diameter of the l_{ij} line which leads to it. The fact that the total transmission from p_i is proportional to M_i is therefore compensated exactly by the fact that this transmission must be distributed over an effective N_{ij} area that is also proportional to M_i. This distribution of p_i-induced excitation in N_{ij} is completely compatible with our earlier observations that N_{ij} transmissions are induced by a random process with two intensity parameters, one giving the concentration of effective "transmitter" distributed along the effective N_{ij} area and the other giving the concentration of p_i-induced excitation along the effective surface N_{ij} area.

The choice of p_{ij} thus follows from the existence of two geometrically locally determined line growth processes. On the one hand, the diameter of the initial segment of a p_i line, proportional to M_i, propagates itself to determine the diameter of the entire line at every stage of its growth away from p_i when the cells send out new lines to other cells. On the other hand, when a growing l_{ij} finally approaches p_j and a mature node N_{ij} begins to form, the joint dimensions of the tip of l_{ij} and of the effective surface of p_j determine the effective area of A_{ij}.

We see from these remarks that we made an interpretive error in our original remarks on equal exchange, for $A_{ij} = AM_iM_j$ and $\neq AM_j$. The source of this error was that we erroneously supposed that the size of M_i directly influences the size of the densities in an N_{ij} transmission. We now see that, in our present system, the diameter of l_{ij} is exactly balanced by the M_i component of effective area of N_{ij}. Since the diameter of l_{ij} is proportional to the circumference of l_{ij}, and since a spike is imagined to be a travelling depolarizing wave along unit strips of the circumference of l_{ij}, we see that the circumference of l_{ij} is proportional to its effective area. Thus the effective area of l_{ij} is proportional to the p_i component of the effective area of N_{ij}, and the effective area of p_i is proportional to the effective area of l_{ij}, while the effective area of the p_j component of N_{ij} is proportional to the effective area of p_j. The effective surface area is therefore, up to scaling constants, a geometrical invariant in this system when we consider isolated points, and it is from the viewpoint of this invariance that we should have considered the effects of transmissions when positing equal exchange. Equal exchange still holds when $A_{ij} = AM_iM_j$, but we see that the effective area of p_i alone is not the critical variable in determining

the intensity of interactions; the M_i term corresponding to this influence may be deleted. What is important is how the effective area varies in successive stages of the transmission process. By the self-similarity of the system, this variation is wholly determined by A_{ij}.

Notice that many properties of the system survive if the lengths of the various l_{ij} are not all proportional to M_i. Indeed, the growth law of the lines is unchanged, for it is locally determined and leaves the line diameter invariant. Equal exchange is not violated, since the phases of individual transmissions are unimportant in defining the concept. ALthough the line lengths themselves may be flexibly determined, the fact that all growth processes determining these lengths are local is very important, for without local determination, we could easily be forced into teleological discussions of how nerves know where to grow.

109. Spatial Self-Similarity

The system before us provides an example of a stationary system in at least the sense that all geometrical relations are determined by local laws which mesh together in such a way that a stable global dynamical interaction pattern is achieved without distorting the geometry. This particular kind of stationary system is of the greatest importance, since all of the geometrical and dynamical relations associated with one point p_i depend on a single parameter M_i. This means that we can be given a standard cell p and can realize all possible cells of the system by simply varying p's M value. This statement must be modified by the observation that the lines emitted by points p_i with a common M_i value can be of different lengths if they happen to fall in the region of attraction of p_j's which lie at different distances from p_i. Nonetheless, all processes that depend only upon isolated p_i's are distinguished entirely by M_i.

The significance of this fact is clearly revealed when we compare the present system with a system from celestial mechanics. In celestial mechanics, one commonly ignores the enormous numbers of variables which are necessary to give an incisive description of a single celestial body. Instead, one simply imagines that the various celestial bodies are so far away from one another that these many variables become irrelevant in a discussion of the relative motions of the bodies. One conceptually contracts each body to a single point and geometrically distinguishes one point from the next by attaching to each point a

single parameter, its mass. Similarly, in the present system, we do not suggest that we do not need a large number of variables to describe the manner in which a single cell is constructed. What we do say is that the cells in the present example can be distinguished by a single parameter, M_i, when questions about the ensemble interactions of these cells are considered. M_i here plays the geometrical role which mass plays in celestial mechanics, and we have already used the artifice of contracting each cellular volume conductor to a point. The analogy of M_i with a mass is a close one even dynamically. In fact, given equal exchange, if we let

$$\mathcal{M}_i(\tau_i) = \int_0^\infty \max(s_i(t)-\tau_i, 0)\,dt$$

be the τ_i-energy of P_i, then $\mathcal{M}_i(\tau_i)$ is proportional to M_i. $\mathcal{M}_i(\tau_i)$ is the energy of p_i which is felt during the time interval $[0,\infty)$ by a measuring instrument that detects all excitations of p_i that exceed τ_i. Such an "instrument" is another point p_j to which p_i projects, with $rp_{ij}c_{ij}=1$. Another useful energy measure is

$$\overline{\mathcal{M}}_i(\tau_i) = \lim_{t\to\infty} \frac{1}{t} \int_0^t \max(s_i(w)-\tau_i, 0)\,dw.$$

This is also proportional to M_i under equal exchange. Other definitions of energy can easily be imagined. The definition depends on the particular kind of measuring instrument that is used.

When the individual points p_i of a system are, as above, distinguished by a single parameter M_i, we say that the points of the system are spatially self-similar. When, moreover, the points interact in such a way that all ensemble characteristics of the system are distinguished entirely by the set of M_i displayed by the field points, we say that the entire system is spatially self-similar. In particular, if the lengths of the l_{ij} lines are indeed chosen to vary with M_i alone in some prescribed way, say that they are proportional to M_i as before, then the above system is spatially self-similar. When the system is spatially self-similar, two further desirable properties can be realized: (i) the transmission time varies with M_i in a simple way; in particular, it can be made independent of M_i. Minimal fields are therefore possible in which the temporal ordering of virtual self-inputs directly reflects the structure of the source points. (ii) The field can easily be made homogeneous in a natural sense, even though $M_i \neq M_j$ for all i and j. This can be done, for example, by imagining that clusters of points are distributed in columns such that every column

contains an equal number of points with each M_i value and the various M_i values are randomly distributed among the various point positions within each column. Stack these columns side by side on a Euclidean plane with uniform density. Connect the lines l_{ij} of length kM_i with points in columns that are kM_i units away. Let the lines of a given length be uniformly distributed in random directions with every line terminating on some point. This field is homogeneous in the sense that each column is identical with all other columns in every essential respect. Here, therefore, is an example in which homogeneity cannot be achieved for individual points but can be achieved for packets of points.

110. Quantization

Letting $M_1 < M_2 < \ldots < M_{n-1} < M_n < \ldots$, suppose that the points are arranged in columnar packets as above. Notice that if (a) the mean distance between columns is h, $0 < h$, and is a stable mean with little fluctuation, (b) the length of each l_{ij} is proportional to M_i, (c) every line terminates on a point, then

$$M_i - M_{i-1} \approx k_i h, \quad i = 1,2,\ldots$$

where k_i is a positive integer, so that the series of M_i values is quantized with parameter h. It is not difficult to imagine a hypothetical physiological system for which all points are separated by a stable mean positive distance. One need only embed the points in a collection of tightly packed objects of a fixed dimension, say a system of vacuoles of a rigidly fixed size that is determined by surface tension. The particular mechanism whereby such a separation might be realized is not so important for our purposes as is the observation that in a spatially self-similar system, a quantization of the separation between points, which is a global property, automatically quantizes M_i, which is a local variable. If you quantize anything in such a system, you must quantize everything, and with the same h. In particular, you must quantize such average dynamical estimates as the various energies $\mathcal{M}_i(\tau_i)$, $\overline{\mathcal{M}}_i(\ _i)$, etc. We see, moreover, that the appearance of h, and not powers or roots of h, is determined by the fact that all geometrical self-similarities varied with h and not with powers or roots of h. Thus, the appearance of a single h depends on a self-similarity property that envelopes the structure of volume conductors, the additivity of component inputs, the random release of transmitter at endbulbs, and so on, which permits us to imagine that such a system can be constructed from its smallest parts out of fundamental geometrical units, in spite of the existence of natural membranous

boundaries. Once one is given a self-similar system, therefore, quantization of this system becomes a rather simple matter because the number of the units in the system is finite, while the self-similarity of the system itself is a stability property that unites geometrical and dynamical variables into a single coherent frame.

111. Reciprocity Between Local Control and Global Interactions

The compatibility of equal-exchange with spatial self-similarity has an interesting interpretation in terms of control processes within a given cell, or within maximal collections of dynamically equivalent cells. Suppose that every p_i possesses a control mechanism that helps to determine its stable M_i values. Only one such mechanism is needed in each cell of a self-similar system, although this mechanism must control the total output of all processes that vary with M_i. This mechanism can be in various states $S(w)$, where w varies in a predetermined parameter set Ω, and p_i has M value $M_i(w)$ if and only if $p_i \in S(w)$. Suppose further that the stable M_i value depends on the distribution of the total transmissions which p_i receives over long periods of time. Under equal exchange, every cell receives equal total average transmission over long time intervals. Thus the determination of which $S(w)$ shall be realized in p_i proceeds in the same way for all i. In fact, suppose that the average total transmission received by any point in a field with n points is Λ and that only minor fluctuations from this average exist. Letting $N_n(M(w), \xi)$ be the number of points with M value $M(w)$ when $\Lambda = \xi$,

$$\Pr(p_i \in S(w) \mid \Lambda = \xi) = \lim_{n \to \infty} \frac{N_n(M(w), \xi)}{n} .$$

In such fields, we can hereby, in principle, acquire information about microscopic control processes in single cells by making macroscopic measurements on many cells.

A more important fact emerges by reversing the order of these considerations. In any realistic situation exhibiting equal exchange, the excitation densities do not remain fixed at any stable mean value. Rather they fluctuate over a broad spectrum of intensities. Similarly, $S(w)$ cannot depend for its determination in such situations on a particular choice of $\Lambda = \xi$. Nonetheless, given the macroscopic requirement that all admissible M_i values appear

in equal numbers, we can conclude that all S(w) which are admissible under the given spectrum of excitation densities must occur with equal probability. We therefore come to the very appealing conclusion that the random occurrence of each admissible state with equal probability produces a spatially self-similar system with equal exchange. In this sense, requiring that the microscopic control process by equally distributed produces a macroscopic system which is itself equally distributed, both geometrically and dynamically, in a natural sense. Conversely, given an equally distributed macroscopic system of the present type, with randomly fluctuating excitation densities, we expect that this system helps to sustain the microscopic equi-distribution law within the admissible range. The macroscopic and microscopic systems thus stand in a reciprocal relation to each other, the distribution of the one helping to stabilze the distribution of the other in parallel fashion.

112. Introduction to a Potential Theory

From these remarks, we see that the following properties are all simultaneously realizable in a single field:

1) equal exchange (uniform dynamical stationarity in the mean)
2) spatial self-similarity
3) the mapping of equidistributed inputs into equidistributed outputs
4) global field quantization
5) the superposition of inputs
6) the existence of local dynamical laws which obey a CPT theorem
7) reciprocity between microscopic control distributions and macroscopic geometrico-dynamical distributions
8) global field homogeneity of packets of points
9) pointwise geometrical invariance of effective surface area
10) the existence of minimal fields that directly reflect geometrical asymmetries in the temporal ordering of virtual self-inputs, and exhibit relativistic contractions, dynamically described
11) the emergence of appropriate wave motions for the preservation of a stable system of interactions, these wave motions exhibiting ergodic properties,

and so on.

The appearance of all of these interesting properties with so little computation cannot be considered an accident. It is due to the fact that an embedding field theory seeks to explicitly represent those aspects of a situation which determine its stability. Our above remarks have the character: if you can see how a given system keeps itself from flying apart, then you also immediately know why, or why not, such properties as (1)-(11) hold. Given properties (1)-(11), we can search for special fields which exhibit all, or a proper subset, of them, using different choices of field functions. Each such choice will give us simultaneous insight into global interactions, local densities, microscopic control processes, and how these various levels of experience interact. From our example and other remarks, we see that one can now begin to give quantitative sense to the claim that, in order to produce a system in which these various levels of experience do interact in a coherent way, we must immediately throw away most conceivable systems. In fact, we must study these systems from the viewpoint of whether or not they can be made to exhibit such properties as (1)-(11), intrinsic renormalizability, field-antifield duality, and so on. A study of the fields in which these various properties contribute to sustain the given field structure in a stable fashion provides us with the set of stationary fields to which all fields eventually must converge. This study thus introduces a type of potential theory into the classification of embedding fields, and limits the class of genetic mechanisms which can control the growth of neural systems.

113. "Name a Fruit That You Like". Habit-Family Hierarchies

From our discussion of the threshold choice $T_i = \mu M_i$, it is clear that variations in the c_{ij} functions of p_i through time have absolutely no effect on the delivery of inputs to p_i until the transmission threshold of p_i is reached, at least if p_i is a fixed point in $\mathcal{P}(\mathcal{I}^*)$. The determination of whether or not p_i begins to transmit under a new input is completely independent of the line residues which have been induced by prior p_i transmission. Whether or not p_i begins to transmit is a wholly geometrical consideration. When $T_i = \mu M_i$ for all i, for example, the onset of every p_i's transmissions displays equal sensitivity to a given input irrespective of the individual differences between prior inputs to the p_i. Once a point begins to transmit, on the other hand, previous discussions amply show that prior experience strongly determines the distribution of the

transmission.

That the prerequisite excitation density for a point to transmit is time-invariant (or slowly varying) enables us to sketch how complex commands are automatically carried out. This sketch will be heuristic and slightly fanciful, but the underlying ideas remain essentially true in realistic fields and are useful to keep in mind during forthcoming constructions of such fields. All points represent collapsed control forms throughout.

How does it happen, for example, when we are commanded: "Name a fruit that you like," that a fruit is indeed often named? To describe even sketchily the process whereby we actually come to use language intelligently in understanding and replying to such a request, or even the process whereby we gain conscious control over complicated muscular acts and predispositions to act, is of course a difficult and lengthy task. Assume that this task has already been accomplished and that we are given a finite fields of points $\{p_i\}$ whose structure is capable of performing the task, say under auditory presentation.

When "name"alone is said, the command is still possibly somewhat ambiguous to the subject, although prior experience shows that "name" does not usually precede all of the other words in a sentence unless it is being used as a command. Thus an input is delivered to the point p_1 representing "name" in its role as a command word. p_1, in turn, transmit subliminal excitation to those points which subserve the general function of speaking; for example, the points controlling jaw, tongue, lips, and similar speech motions. If the subject is ill-experienced in English, "name" alone might be an ambiguous input, so that points representing "name " in other roles will also be nontrivially excited by the vector input function; for example, "name" in its role as a noun. By the time "a" is also said and induces an input to the "a" point p_2, however, the point complex $\{p_1,p_2\}$ will activate new lines that uniquely determine which "name" point is intended, whence the subliminal speech excitations will again be induced. The subliminal excitation of the speech points makes it easier for immediately future inputs to cause muscular motions of the speech type.

The input "fruit" activates p_3. p_3, by past experience in concept formation, subliminally excites a collection of points $\{p_{31},p_{32},\ldots,p_{3n}\}$, each of which represents a particular fruit name. The input to p_3 thus temporarily forms a psychological set which includes both the supraliminally excited "fruit" concept point p_3 and the subliminally excited individual "fruit" naming points $\{p_{3i},i=1,\ldots,n\}$.

"that," or more properly "fruit that" assures that sequential inputs continue to arrive without pause after p_3 is excited and thereby, in particular, suppress those internal mechanisms that deliver spontaneous inputs to the field points when no external inputs are actively arriving. Delivering an input to some p_4, here the point representing "that," thus suppresses all internal mechanisms which would wash away the previously delivered external and subliminal inputs before all of the important information in the command is presented. The input to p_4 acts as a "fill-in" in the sense that (i) p_4 does not itself subliminally excite a special set of points that possess a particular denotative similarity and motore control forms, as did p_3, and (ii) the interpolation of p_4 allows p_3 to induce its renormalization of the p_{3i} more gradually and more completely than would have been possible if p_3 was immediately followed by a p_4 which itself conrolled an extensive psychological set. Such fill-ins also permit smooth variations in attached emotive fields. Delivering an input to a "fill-in" like "that" thus preserves the field normalization induced by preceding inputs, which includes, in particular, keeping the organism oriented to the reception of auditory inputs, and prevents competing renormalizations due to new inputs from occurring until prior renormalizations are carried out.

The self-referential "you" is, of course, a fairly special point, p_5, from the viewpoint of development, since it is embedded only after a complicated series of self-inputs has occurred. Here, we simply assume that an input to p_5 excites a broad distribution of lines leading from p_5. By the time the input for "like" begins to be processed cortically, the previous points have already conditioned the line structure in such a way that the vector input arrives primarily at p_6, which is the emotive "like" point, and only secondarily, if at all, at the "like" point that is used for making comparisons. p_6 projects to the emotive points: $p_7,\ldots,p_r$, and excites those points associated with positive affects. These points reciprocally inhibit the points associated with negative affects. p_5 also projects to $p_7,\ldots,p_r$ in a similar fashion. Indeed, with growing experience, $\{p_5,p_6\}$ projects increasingly as a multiple complex. The excited $p_7,\ldots,p_r$ points, in turn, transmit broadly to the cognitive fields, in particular, to $p_{31},\ldots,p_{3n}$. Those p_{3i} receive the greatest input from $p_7,\ldots,p_r$ which have been most strongly associated in the past with positive affects.

The complex $\{p_{3i}, i=1,\ldots,n; p_5, p_6, p_7, \ldots, p_r\}$ may indulge in more complicated cyclic interactions than we now choose to describe. Yet the qualitative result is clear. Within a few moments, the sequence of inputs: "name a fruit that you like" has automatically excited, from among many thousands of points, a small collection of points representing fruit names and among these fruit names has excited still more those fruit names which stand for fruits that are well liked. Moreover, the fruits which are most common in the experience of the organism will be most highly excited by p_3 transmission, while the fruits which are best liked by the organism will be most highly excited by $p_7, \ldots, p_r$ transmission. Thus two hierarchies of excitation subliminally summate to single out a very small number of points, and the muscular mechanisms involved in expressing these points behaviorally are subliminally prepared to act. Under more complicated input sequences, many more hierarchies, both excitatory and inhibitory (the "habit-family hierarchies"!), can be activated to localize subliminal field excitability in a very small set of points--due to the existence of a geometrically determined transmission threshold. If a single control form is a decided favorite in all hierarchies, it is quite possible that the transmission threshold for this form will immediately be reached. Due to the subliminal preparation of the motor speech centers, this form will immediately induce the dynamical walk leading to the evokation of the name of the well-liked fruit. If not, the p_{3i} points will enter into a phase of active competition governed by lateral inhibitory processes until the response criterion is reached.

An important fact to observe is that once the various subliminal preparations have automatically been made, only two kinds of tasks need to be provided for within the organism, leading to response: (1) Sustain the inputs induced from p_3, p_5, p_6 until a response is made, (2) deliver small random inputs to the cognitive fields in order to help bring the double subliminal hierarchy into the supraliminal response range. The subliminally most highly activated points will of course be most benefited by such a random input. (1) can be achieved by a moderately simple reverberation process, just so long as not so many inputs are rapidly delivered that the renormalizing dual-field excitation is dragged along in pursuit of the most recent inputs. (2) can be achieved by any broadly distributed excitatory mechanism.

The crucial point common to both (1) and (2) is that the organism need not work to distribute inputs itself. This is automatically accomplished by the

interaction of embedded material with the incoming vector input functions. The most that the organism needs to do it to keep the processed inputs alive until the response criterion is reached. Once a response occurs and a feedback input is delivered, the field is slightly renormalized and the organism might change its mind!

This discussion is, of course, a considerable oversimplification of the actual flow of neural events. Nonetheless, it shows how concepts of multiple lines, broadly distributed projection domains, summating subliminal excitations, renormalizing lateral inhibitions, control subforms, vector input functions, and the like, can be applied together to give immediate insight into the qualitative features of how organisms come quite rapidly, and with little conscious effort, to the production of complicated behavioral acts. A rather enormous number of behavioral examples can be based on the model of this example. One discouraging characteristic of such examples is that one supposes that the embedded material is already available and that the input is obediently delivered to the proper embedded point, after which all interactions look quite simple. We will therefore avoid the propagation of such examples until we perform some more realistic field constructions to illustrate the manner in which old inputs become embedded and how these embeddings come to interact automatically and harmoniously with later inputs. These constructions are largely based upon principles which we already know, but they provide useful crystallizations of these principles.

114. Failure of Energy Principles

Before proceeding to such constructions, notice that the previous example shows in several places why any reasonable "energy principle" must fail. "Energy principles" as classically understood, say in psychophysics, refer to the supposition that neural processes, themselves not directly measurable by psychophysical techniques, behave in a simple incremental fashion under the application of increments of physical stimuli that vary along a given intensive or geometrical continuum. Such principles break down violently during lateral inhibitory processes, for even the most intense equally distributed inputs to homogeneous complete sets of antagonists generate asymptotically non-transmitting

strength functions and thus no response.

An important special case of this breakdown occurs as a result of the process of subliminally exciting several hierarchies H_i, i=1,2,...,n, of points during closely overlapping time periods, as in the previous example. If the subliminal excitation to all hierarchies has the same distribution, the energy principles are perhaps not too strongly violated, since even after lateral inhibition the point which receives the largest input in any one hierarchy will possess the largest strength asymptote. When these hierarchies are excited by more nearly orthogonal distributions, however, all energy principles fall to pieces. In particular, suppose that H_i excites $\{p_1,p_2\}$ by delivering a subliminal input of unit intensity to p_i, i=1,2, and of zero internsity to the other point. Also suppose that $\{p_1,p_2\} = sp^{-1}(\mathcal{H})$, where $\mathcal{H}$ is a homogeneous complete set of antagonists. Under either H_i alone, s_i has an excellent chance to satisfy the response criterion, especially when $\mathcal{F}(p_1 \oplus p_2)$ receives small spontaneous random inputs. Under $H_1 \oplus H_2$, on the other hand, $s_1 = s_2$, so that s_1+s_2 will be asymptotically very small due to reverberation with $\mathcal{H}$. Doubling the total input thus reduces the total s_1+s_2 output by an enormous factor.

This example also shows where an energy principle might best seem to hold. Suppose an input to a mixed field is so localized that it does not actively stimulate any points that lie in the "off" region of the excited points. The difficulty with lateral inhibition is here removed, if one is willing to restrict attention to the one-step transmissions of the excited points. If the excited points also lead directly to response points, without the interpolation of pre-biased renormalizing fields, intensifying the input will indeed increase the probability of the expected responses by familiar mechanisms. Yet even this apparent verification of an energy principle is a local illusion. For, if one considers the "total (instantaneous) energy" $\Sigma_i s_i$, where Σ_i varies over all points connected with the excited points P, even as the s values of the P points grow, the s values of the other points will be more strongly suppressed. These considerations hold for all mixed fields, including eye retinas, where they have often been misunderstood in psychophysical theorizing about the neural transformation of light energy. The fact that local point excitations carry with them suppression of their peripheral "off" fields may be used to explain such phenomena as synchronizations in the lateral geniculate.

115. Local Membrane Systems and Potential Averages

It is also interesting to note that even in an isolated volume conductor, no additive energy principle strictly holds if the volume conductor is endowed with a bounded homogeneous set of intensive levels, as ours are. For example, subliminally excite p_i by I_1 and follow this excitation immediately, at time t_1, by an I_2 that is sufficiently large to bring s_i to supraliminal values. If I_1 brings s_i to an asymptote of λ, I_2 will begin its excitation with an effective embedding space of $\alpha(M_i - \lambda)$ rather than one of $\alpha(M_i - m_i)$. But if $\mathcal{A}_i(I_2, \sigma)$ is the asymptote of s_i under I_2 when $s_i(t_1) = \sigma$, then $\mathcal{A}_i(I_2, \sigma) - \sigma$ is a monotone decreasing function of σ. Since the relative asymptote $\mathcal{A}_i(I_2, \sigma) - \sigma$ that I_2 induces depends on the initial s_i value σ, our conclusion immediately follows. In particular, if we distribute subliminal excitation nonuniformly to a set $\{p_i\}$ and follow this subliminal excitation by a uniform input to all points, the ordering of the new asymptotes will be preserved, but the absolute differences between the new asymptotes will be smaller than the differences between the old asymptotes. Such a contraction in the distribution of asymptotic differences occurs also when a uniform random input brings the values of a subliminal hierarchy into the supraliminal range. Observe, however, once the subliminally excited points begin to transmit, that if they transmit to quiescent points, the contraction in their relative asymptotes will be compensated by the large effective embedding spaces of the recipient points, so that the contracted asymptotes of the transmissions will induce a relative dilation in the asymptotes of the recipient points. In any one cycle: subliminal excitation, uniform random input, transmission to quiescent points, the contraction in relative asymptotes is partially compensated by a dilation.

One is interested in the additivity of strength increments under additive inputs partly because there seems to be evidence that the simultaneous presentation of two inputs produces an EPSP asymptote that is the sum of the EPSP asymptotes produced by the two inputs separately. (See Eccles' The Physiology of Nerve Cells.) On general grounds, one is interested in this problem because it is always useful to see how linearity is introduced into an essentially nonlinear system. The above discussion shows that to preserve such additivity in s_i, we must pass to more complicated cells than volume conductors. We must pass to cells for which short bursts of simultaneous N_{ij} and N_{kj} transmission induce potential changes in p_j such that the N_{ij} (N_{kj}) induced potentials do not substantially influence the operation of the p_j membrane adjoining the N_{kj} (N_{ij}) node. The cell membrane

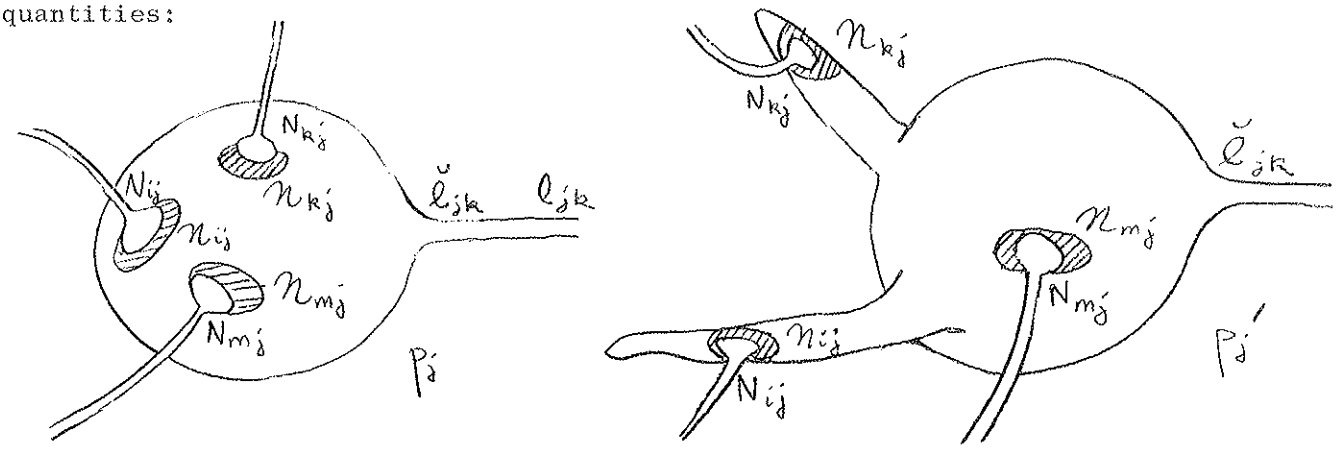
must therefore be conceptually decomposed into a collection of local systems clustered along the effective surface area of the membrane. These various local systems are coupled to the particular N_{ij} to which they are closely juxtaposed. Call the local system associated with N_{ij} $\mathcal{N}_{ij}$. If we do not want the excitatory asymptotes which N_{ij} can induce to be completely independent of the initial local cell potential at $\mathcal{N}_{ij}$, the decomposition $\oplus_i \mathcal{N}_{ij}$ must depend on some combination of two types of processes: (i) the propagation of local $\mathcal{N}_{ij}$-induced potential changes is slow compared to the time scale of unit events in any $\mathcal{N}_{kj}$ process, $k \neq i$, and (ii) an ongoing $\mathcal{N}_{ij}$ process proceeds with sufficiently great inertia that it is (at most) weakly affected by simultaneous volumetrically propagating potential changes induced by $\oplus_{k \neq i} \mathcal{N}_{kj}$. (i) and (ii) express functional conditions for the splitting of the cellular volume into partially independent subsystems. (i) accomplishes this by postulating a restricted propagation mechanism of global potential exchange in the cell, while (ii) allows the already actived local membrane systems to partially disregard globally propagating influences. (i) and (ii) thus represent complementary ways to achieve the same result.

If (ii) holds, it is difficult to avoid the conclusion that the $\mathcal{N}$ processes involve higher energy exchanges than the volumetrically propagating potential changes. In fact, unless (i) alone is a sufficient mechanism to ensure s_j additivity, N_{kj} transmission must induce a temporary asymmetry in the $\mathcal{N}_{kj}$ process: events at the exterior of the effective surface area of $\mathcal{N}_{kj}$ must dominate the entire $\mathcal{N}_{kj}$ process. Otherwise, potential changes induced at $\mathcal{N}_{ij}$ by simultaneous N_{ij} and N_{kj} unit transmissions will reach N_{kj} before the $\mathcal{N}_{kj}$ process is spent. Once these potential changes propagate to $\mathcal{N}_{kj}$, the interior $\mathcal{N}_{kj}$ process parameters will be immediately reset. If the exterior $\mathcal{N}_{kj}$ parameters do not take precedence over the interior parameters, the exterior parameters will be forced to follow suit. The $\mathcal{N}_{kj}$ process rate will thus be immediately modified, and s_j additivity is lost.

When (i) and/or (ii) hold, the local cell volume potentials do not undergo an instantaneous averaging process. The initial segment $\breve{l}_{jk}$ of the l_{jk} circumference therefore no longer responds to a globally uniform volume potential. Rather, the $\mathcal{N}$-induced local potential changes must propagate through the cellular volume to $\breve{l}_{jk}$. As these local potentials propagate, they must average with one another, and the resultant excitation densities of this averaging process which impinge on $\breve{l}_{jk}$ trigger $\breve{l}_{jk}$ transmission. This resultant is the local analog of a globally uniform excitation density.

116. Dendrites and Cell Deformations

The cellular membrane is thus decomposed into a large set of local systems that help to control a resultant $\overset{\cup}{l}_{jk}$ potential which triggers the onset of transmissions. An important new feature results from this conception of the transmission process. For example, if the locally propagating $\oplus_i \mathcal{N}_{ij}$-induced potentials diffuse in a classical manner through the cellular volume, then the resultant $\overset{\cup}{l}_{jk}$ potential depends on the shape of the cell. M values alone are no longer sufficient to characterize even the ensemble behavior of cells. The cells must be viewed as bodies embedded in $\mathbb{R}^3$ to describe the three dimensional diffusion of local potentials. In particular, let us be given a spherical cell p_j with fixed effective surface area, fixed volume, and fixed transmission lines. Let p_j be deformed to p_j' as in the diagram without changing the fixed quantities:



p_j and p_j' are indistinguishable in terms of M_j. But they are not dynamically indistinguishable mod $\mathcal{T}(\overset{\cup}{l}_{jk})$, since equal N_{ij} and N_{kj} inputs to p_j and p_j' induce potentials that take longer to be propagated to $\overset{\cup}{l}_{jk}$ in p_j' than in p_j, and execute wholly different diffusions on the way. Moreover, in p_j', it is obviously much easier to accomplish s_j additivity with respect to N_{ij} and N_{kj} than it is in p_j.

It might be extremely difficult for Nature to create chemical systems, suggested by (ii), which succeed in almost completely favoring exterior membraneous factors in the $\oplus_i \mathcal{N}_{ij}$ process. The deformation of portions of $\oplus_i \mathcal{N}_{ij}$ into cellular elongations might be much simpler to accomplish. (i) becomes an increasingly important factor as such deformations are performed, and the realization of (ii) becomes less vital. We may therefore view the existence of many cellular elongations as an expression of facts like the following: (1) it is sometimes useful that additive inputs to different $\mathcal{N}_{ij}$ systems generate additive

inputs to the $\overset{\cup}{1}_{jk}$ resultant excitation density, but this cannot be accomplished by volume conductors, (2) some combination of (i) and (ii) are needed to do this, (3) if (ii) is predominant, a strong asymmetry in local chemical dynamics along active membrane sites is required. Such an asymmetry might be difficult to achieve naturally, especially since proper cellular metabolism requires that many different kinds of chemical transport occur across the cellular membrane in both directions, (4) (i) is much easier to realize, for it merely requires that the cellular shape be deformed from a spherical shape to a spheroid with elongated projections, (5) when (i) is primarily relied upon, inputs to the elongations will induce almost additive contributions to the $\overset{\cup}{1}_{jk}$ resultant, while inputs to the spheroidal body will behave more like inputs to a volume conductor. This distinction between inputs to the elongations and the spheroid will be largely destroyed if (ii)'s asymmetry is well accomplished. Thus by failing to rely on (ii) and on the production of a highly asymmetrical $\oplus_i \mathcal{N}_{ij}$ process, the cell succeeds in being able to realize subtle changes in the input-determined transmission process by simply altering its Euclidean shape without changing either its global $\mathbb{M}$ values or its local exchange processes. In effect, by relying primarily upon (i) rather than (ii), the cell embeds itself into $\mathbb{M} \oplus \mathbb{R}^3$-space rather than in $\mathbb{M}$-space alone; and (6) by relying upon (i) and cellular elongations, the cell vastly increases its effective surface area, per unit volume, and thereby becomes a much more sensitive averaging device of the neural potentials carried by fibers in its vicinity.

The cellular elongations in p'_j are, of course, dendrites. We have thus arrived at a partial appreciation of the uses of dendrites, and any other nonspherical deformations of cellular shape, from a simple line of reasoning that directly follows from psychological data.

117. Localized Sources with Long-Range Potentials. The Saltatory Hypothesis

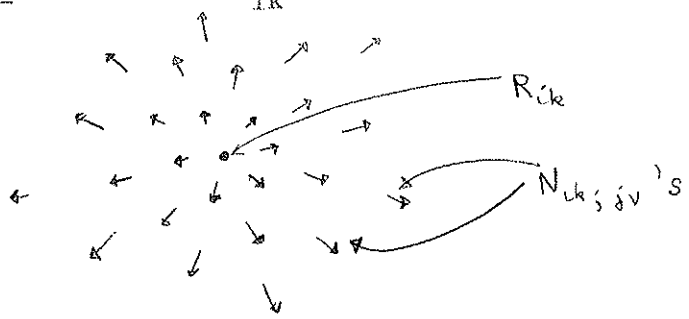
Two general types of conduction process emerge from the above discussion. (1) The first type--(i)--shows that certain cellular processes must be locally propagated throughout the medium of the intracellular volume. Such processes are often of a diffusive character, and depend on global features of the cellular geometry only as boundary conditions. The fact that these processes are governed by local, decremental laws allows us to vary the degree of independence of disjoint

effective surface areas of a single cell by merely deforming the shape of the cellular volume joining them, say into dendrites. (2) The other type of propagation process is not governed by local events in the cellular volume. Rather, it restricts itself to the surface of the cell and the immediately adjoining regions, that together form a unified biochemical system. The best example of this type of propagation is seen by considering a spherical conducting axon whose diameter is invariant over its entire length. In our discussion of $R_{ik} \longrightarrow N_{ik;jv}$ invariance, this type of propagation was realized by considering a thin ring of excitation, surrounding the circumference of the axon, which propagated nondecrementally at a constant velocity from the initial segment of the axon over the total axonal length to $N_{ik;jv}$, where its total excitation was distributed to the effective surfaces of the $N_{ik;jv}$ membrane as an excitation density. This form of propagation is essentially non-decremental over the range of axonal lengths that naturally arise. It is therefore representable by a wave equation, which describes the propagating ring of excitation as it travels down the axonal membrane to $N_{ik;jv}$. (1) is thus a local, volumetrical, and decremental propagation process--a diffusion--, while (2) is a boundary-bound and non-decremental process--a propagating wave. (1) is the propagation process suitable to tying cellular dynamics to the Euclidean topology in which the cell lies, while (2) enables cellular interactions to ignore the Euclidean topology and to reduce the problems of cellular interactions to a very efficient transport of excitation for which even the size of temporal transport lags can be manipulated at will by a proper choice of the reaction constants of the cellular membrane. Although interacting mixtures of (1) and (2) are possible, it is useful to treat the two processes independently at first to emphasize their striking differences.

Let us consider excitation transport along a myelinated axon in the light of (1) and (2). In particular, what does the saltatory hypothesis of transmission down such fibers mean with respect to (1) and (2)? This hypothesis is intended to distinguish the transport of excitation in myelinated axons from the transport of excitation in unmyelinated axons. In long unmyelinated axons, (1) would produce a severe decrement of excitation transport between R_{ik} and $N_{ik;jv}$'s effective membrane surfaces. (2) is the only method whereby long unmyelinated axons can propagate excitation efficiently. If (2) is also applied to long myelinated axons, then unmyelinated and myelinated axons enjoy essentially the same transmission structure, and the myelin sheath no longer can be viewed as an arrangement that functionally distinguishes the transmission dynamics of

myelinated and unmyelinated cells.

If this is so, what does a myelin sheath do? One important use of a sheath is the following. By covering the axon $l_{ik;jv}$ with a sheath, one eliminates the possibility that profuse, strongly coupled $N_{mr;(ik;jv)} \longrightarrow l_{ik;jv}$ connections can occur. The myelin sheath reduces the interneuronal effective surface area of the axon almost to zero. The dynamical counterpart of this geometrical contraction in effective surface area is that R_{ik} and the distant $N_{ik;jv}$ are joined together by a very stable and efficient excitation transport mechanism like (2). The geometrico-dynamical combination of myelin sheath and (2) allows us to think of a parallel transplantation of strength along the $l_{ik;jv}$ line, just as we did when we introduced the macroscopic embedding equations. A very important consequence of this transplantation idea is that the range over which an R_{ik} exerts its influence as an output mechanism can be made very large without risking that inputs to the axonal transmission mechanism bias the outputs from the cell body. That is, by myelinating axons, it becomes possible to talk about a localized source of interactions (the cell body R_{ik}) which can induce long range interactions (to broadly dispersed $N_{ik;jv}$ terminals). In particular, when R_{ik} acts as a volume conductor, we can contract the cell body to a single point. The distribution of $N_{ik;jv}$ effective surface areas now becomes the geometrical carrier of an outgoing potential field around this points, while the $N_{mr;ik}$ areas are the geometrical carrier of an incoming potential to the point R_{ik}.



Then analogy to classicial physical field theories is obvious. Such a local description of source points with prescribed radiating interaction "potentials" is no longer generally possible in the absence of a myelin sheath.

Without a myelin sheath, every millimeter of the $l_{ik;jv}$ axonal membrane is, in prospectus, a region to which new inputs can be delivered, and is therefore a possible source of new transmissions to $N_{ik;jv}$. When the unmyelinated axon $l_{ik;jv}$ is covered with $N_{mr;(ik;jv)}$ terminals, transmissions received by R_{jv} over

$l_{ik;jv}$ are no longer an indication of the resultant $l_{ik;jv}$ excitation, but are rather the resultant of a set of excitation sources that is broadly distributed from $l_{ik;jv}$ to $N_{ik;jv}$. The source of excitation can therefore no longer be contracted to a single point. It is nonlocally distributed throughout a space-time region $\Omega_{ik;jv}$ whose spatial projection is distributed along $l_{ik;jv}$. If we tried to describe such a source in classical field theoretical terms, we would always be forced to use distribution theoretical integrals of the form

$$\int_{\Omega} \psi \ldots du$$

where $\Omega_{ik;jv} \subset \Omega$ and the support of the test functions ψ contains the region $\Omega_{ik;jv}$. A myelinated fiber thus creates a contracted form of the possible source distributions in a nerve fiber. Such a contracted form represents a very stable interaction structure. The price paid for this stability is the rigidity of such structures. They are not capable of the widespread geometrical changes which unmyelinated fibers can sustain in adaptive response to substantial changes in the input distribution. The response of a myelinated fiber $l_{ik;jv}$ is largely restricted to its limited effective surface area, and the $N_{ik;jv}$ structures which interact with this area. The process whereby unmyelinated fibers become myelinated can thus be viewed as another way in which the nervous system forms rigid channels from plastic fields in order to produce asymptotic field structures which respond to their expected input arrays with great stability. Such non-physiological changes as cutting a myelinated fiber, with the subsequent chromatolysis, do not, of course, fall under these remarks.

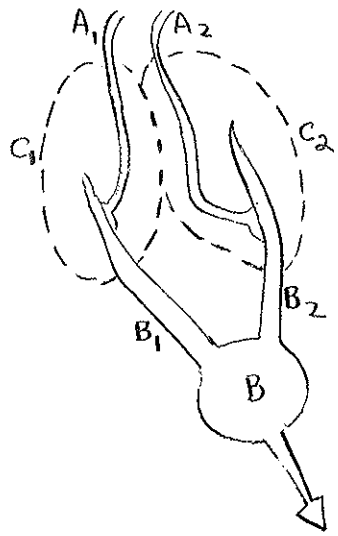
Notwithstanding these considerations, it has been suggested that myelinating a fiber permits a profound increase in the efficiency of local axonal transmission. This increase must improve the efficiency of a mechanism of type (2), for a mechanism of type (1) is hopelessly inefficient for long transmissions no matter how you adjust it. It has, nonetheless, been suggested that the nodal areas serve as sources for actively replenishing transmitted excitation, which thereupon propagates electrotonically down the axon until the next node is reached, whence the process is repeated. Consider such a propagation process. If γ is the number of nodes, regularly spaced, between R_{ik} and R_{jv}, it is easy to see that transmissions along $l_{ik;jv}$, which enter $l_{ik;jv}$ with intensity u, reach R_{jv} with intensity less than uv^m, where v is a positive constant less than 1, $m > 0$. For between every pair of nodes, u decays decrementally in the usual way, and the

resuscitation of the input at a node receiving a transmitted intensity less than u cannot restore the excitation intensity to its original value without making $v > 1$. In this latter case, transmission between cells will be explosive and unstable; the cells separated by the longest distances will exert the most violent effects upon one another. Of course, one can logically search for a resuscitating nodal mechanism for which $v=1$, but such a mechanism will be very hard to realize in vivo, and it is highly unstable because the slightest fluctuations in v will change the transmission process from a decremental process to an explosive process, or vice versa. We therefore reject this possibility, and conclude that even when a myelinated fiber permits (1)-interactions only in the internodal regions, the transmission process is severely decremental over long axons.

The electrotonic hypothesis for internodal transmission in myelinated axons has also been advocated to explain why certain myelinated axons transmit faster than unmyelinated axons. But (1)-transmission depends on a local diffusion whose rate is determined by structural properties of the cell body material. Such properties are more uniform across nerve cells than are axonal transmission rates. Moreover, the local determination of transmission velocities is incompatible with data showing that, in many axons, transmission velocity varies directly with such global factors as axonal fiber diameter, and these global factors can be more easily accounted for by varying the (2)-structure of the membrane. Further, one sees intuitively that if the decision has to be made between a diffusion and a wave when the problem is to transmit quickly and efficiently across long distances, a wave is usually the more rational transport mechanism. For all of these reasons, a (2) mechanism seems preferable to a (1) mechanism for producing fast transmissions. But a (2) mechanism does not require a myelin sheath. Thus, either the myelin sheath, at best, improves the efficiency of a (2) mechanism, or Nature has chosen a very inefficient (1) mechanism to govern Her marvelously delicate neural inventions and has tried to patch up this gross error by wrapping the axons in myelin sheaths. Since these sheaths serve the useful purpose of localizing field sources and beautifully stabilizing cellular interactions--a function independent of the saltatory hypothesis--and since Nature never seems to be sloppy about such important details, we conclude with the suggestion that the saltatory hypothesis, in its classical form, must be modified. Several deeper reasons for the existence of sheaths shall be presented in the following sections.

118. Schwann Cells, Neuroglial Cells, and Functional Boundaries

The use for Schwann cells which we have envisaged above is not, of course, restricted to axons. Our consideration of axonal sheaths showed how the cell body can be made the total source of axonal output. We can just as easily envisage the localization of cellular inputs along a dendritic-cell body system. Wherever Schwann cells form a configuration among neural cells, this configuration can be viewed as a natural functional boundary which separates various cell groups into (partially) independent subsystems with respect to their capacity to form strongly coupled cellular connections. For example, if a strongly coupled terminal axon A and a small dendritic region B are enclosed by a closed surface of Schwann tissue, it follows from our above considerations that the region forms a localized connection whose strong coupling mechanism is functionally independent of even closely justaposed axonal-dendritic terminals. In particular, consider the schematic diagram



Here two closely juxtaposed dendritic branches B_1 and B_2 pass to a single cell body B. The axon A_i forms a strong coupling with B_i, and C_i surrounds $A_i \oplus B_i$ in such a way that $A_1 \oplus B_1$ and $A_2 \oplus B_2$ are separated by the total functional boundary $C_1 \oplus C_2$. Inputs carried over A_i influence only B_i because of this separation. When the dendrites obey alternative (i) of sections 115-116, this separation allows the dendrites to contribute almost independent potential contributions to B. The C_i thus render more efficient a functional tendency that is already implicit in the geometrical configuration of the cell, a tendency which is here realized in the existence of long, separate dendrites. On the other hand, if a cell body B is itself a perfect volume conductor, and if all axons $\{D_i\}$ terminating on B always fire in unison, it is functionally pointless to form a sheath around every $D_i \longrightarrow B$ connection, for all of the D_i transmit together,

whence B, as a volume conductor, cannot dynamically distinguish one D_i from another. This remains true no matter how broadly scattered the cells with D_i axons lie in Euclidean space. The natural transmission unit for such a coupling is manifestly $\oplus_i D_i \longrightarrow B$, whence we will expect any sheath that develops to enclose this entire system as a single unit.

These examples of axonal-dendritic and axonal-volume conductor connections illustrate a general situation. When we want to draw functional boundaries, or sheaths, around cell groups, we do so to separate, or to distinguish, only those transmission systems that are locally dynamically distinguishable. The drawing of sheaths around dynamic ally distinguishable transmission systems is one of Nature's ways of assuring that geometrical distinguishability and dynamical distinguishability go hand in hand, at least asymptotically.

As another example of this general principle, suppose that a collection $\{A_i\}$ of closely juxtaposed cells acts in unison to excite just one output cell B, and that $\{A_i\}$ receives a collection $\{C_i\}$ of fibers, from a single common source D, which are randomly interspersed among the A_i. In this situation, a sheath should enclose the entire system $\mathcal{B}(\oplus_i C_i \oplus \oplus_i A_i)$, where $\mathcal{B}(G)$ is the effective surface area of G. Thus, the sheath system encloses the entire region over which $\oplus_i C_i \longrightarrow \oplus_i A_i$ strong couplings occur. As a final example, suppose that a set of dynamically independent cells $\{A_i\}$ all send lines to the cell body of the volume conductor B. Here, the sheath separates each $\mathcal{B}(A_i \oplus B)$, even though B spreads excitation derived from a single A_i to its entire surface membrane. The vital point is that the individual A_i do not transmit in unison, whence localized $A_i \oplus B$ regions will become strongly coupled independent of one another. Nonetheless, the volumetric conduction within B might stimulate a secondary sheath to form about $\mathcal{B}(\oplus_i A_i \oplus B)$ in addition to the primary sheaths separating the individual $\mathcal{B}(A_i \oplus B)$. This example contrasts the above example for which a pool of neurons $\{D_i\}$ transmits in unison to a volume conductor B. Here $\mathcal{B}(\oplus_i D_i \oplus B)$ alone is the maximal dynamically indistinguishable sheath partition.

Schwann cells need not be the only cells capable of forming sheaths. In the higher central nervous centers, for example, various glial cells seem to be the natural candidates for the task. If the formation of sheaths proceeds significantly more slowly than s fluctuations, then--just as in the case of slowly changing p distributions--these sheaths help to rigidify and therefore to

stabilize the c lines residues, both by helping to channel the chemicals released by new s fluctuations and by actively interacting with nerve cells on a metabolic level to sustain the coupled c values. We will discuss these factors in greater detail in the following pages.

119. Neural-Glial Duality

How do sheaths know where to form? They asymptotically enclose maximal dynamically indistinguishable clusters of cellular coupling sites. Yet cell groups which are dynamically indistinguishable at one time may develop dynamically distinguishable subsets at a later time, and (possibly) vice versa. Indeed, as maturation proceeds and newly learned embeddings are continually formed, the partition of $\mathcal{F}^*$ into maximal dynamically indistinguishable sets shows a distinct drift in the direction of continual refinement. We thus envisage a Schwann cell, or glial cell, entourage for nerve cells which is sensitive to subtle variations in the distribution of dynamically distinguishable cell groups. This sensitivity must obviously be controlled, in first approximation, by either of the two major cellular traces of dynamical distinguishability.

The first trace is closely tied to the values of the c functions at any time, for these functions are the residual repository of past cellular interactions, and thus the stored record of dynamically distinguishable excitation distributions. This trace, even in the absence of excitation for short periods, can redirect the growth of sheaths if the cells from which the sheaths are formed share a mutual metabolic system with the nerve cell. The component of this system in the nerve cell must depend on the value of the nerve cell's c function at any time. The component of this system in the glial cell will therefore respond to variations in c. If increasing c renders the nerve cell a better donor to the glial cell of its requisite metabolic needs, then--and only then--will the glial cells functionally wrap themselves about differentially excited, local strongly coupled regions. And they will distribute themselves within these regions most densely where the local c values are the highest. The other way to envisage the distribution of glial cells is to suppose that they are directly responsive to fluctuations in neural electrical potentials. This hypothesis means that the glial metabolism is benefited by the electric potentials which spread (electrotonically and membrane-coupled) from the neural cell body to the glial cell body proper. For such a hypothesis, the nerve cells and the glia can behave

like completely independent metabolic systems, for the glia satisfy their neurally derived metabolic needs through direct electrical excitation and without chemical intervention via the nerve cell's metabolic machinery. Nonetheless, if the nerve cell metabolism is coupled to its c state, the glia and nerve cells will show parallel metabolic changes, for both respond to highly similar electrical potentials.

A mixture of shared chemical bonds and purely electrical excitation is, of course, conceptually possible, since both methods help to create functional boundaries around dynamically indistinguishable cell groups and between dynamically distinguishable cell groups. It must be observed, however, that such boundaries will be formed more efficiently if the glia are sensitive to the kind of effect--electrical or metabolic--which is most highly localized in the proper cellular region. In particular, if electrical potentials induced by a single axon can spread over a region that encloses the entire excited cell body and all of its terminal axons and dendrites, then "attracting" the glia by electrical fluctuations alone will never enable them to distinguish local axon--terminal-cell interactions. Since a c-determined metabolic neural state which interacts monotonically with the glial metabolism is highly localized, we see that such a mechanism is more efficient at forming sensitive functional boundaries. Similarly, joint neural-glial electrical potentials must be thought of as being carried by the chemical ionic fluxes coupled to them. We earlier tentatively identified c with the state of a neural cell control process underlying the production of transmitter, where "transmitter" is to be thought of in a generalized sense. Using this interpretation of c, and assuming the existence of a joint neural-glial metabolic system as above, it follows that certain alterations in the molecules which control neural cell metabolism will be matched by dual changes in the glial control molecules, and that these neural molecular changes will be induced by learning experiences via the strong coupling process. Control molecules intimately include the various RNA's. Thus, as a result of the easily motivated desire to draw functional boundaries around the natural functional cellular units in neural interactions, we have come to expect dual variations in the RNA ratios of nerve cells and their juxtaposed glia as a result of learning experiences. Such variations have indeed been reported, and appear in the beautiful studies of Hyden.

As a corollary, we must also expect some glia to move about rather freely, or at least to be able to grow or to reproduce themselves more rapidly when juxtaposed c values are high. For otherwise, we would have insufficient glial material available from which to construct new functional boundaries. The

important point is that the glial reservoire be sufficiently rich--at least asymptotically--to match the c-coupled metabolic changes in adjacent nerve cells in a reciprocal way.

Another consequence of neural-glial duality is the following. Let us be given a strongly coupled cellular system A with a closed glial boundary B, and suppose that the c value of A determines the production of a given transmitter, which we will suppose is acetylcholine, for specificity. When c increases on successive trials, a unit input to A will liberate increasing total quantities of transmitter. This transmitter diffuses throughout the region bounded by B. The following questions immediately impose themselves concerning this diffusion: How can the transmitter which does not directly excite the recipient cell be expeditiously eliminated to prevent an inefficient lingering of cellular excitation due to slowly diffusing transmitter residues in B? How can the transmitter be prevented from diffusing through B into the cellular surround? The most obvious way to do this is to supply the glia forming B with an acetycholine annihilator, namely cholinesterase. How much cholinesterase? Enough to inactivate the expected transmitter residue, which is itself regulated jointly by c and the intensity of the input to A. Since the simple neural-glial interaction which we have discussed is also determined by c, we are led to the following conclusion: Just as increasing c generates an increase in acetycholine production in the nerve cell body, increasing c creates a change in the dual glial control mechanism which initiates a parallel increase in cholinesterase production in the glial cell. The total quantities of acetycholine and cholinesterase will hereby always be of an order of magnitude in a given neural-glial system appropriate to the effective annihilation of free transmitter.

How is the cholinesterase stored in the glia? Acetycholine is thought to be stored in the nerve cell body and is released when an electrical potential suitably polarizes the cell. If the neural-glial system were completely dual, we would expect the neural electrical potential, radiating volumetrically to the glial cell, to also be the impetus for cholinesterase release from B into the A region, according to the same law by which this electrical potential releases acetycholine from nerve cells. The greater distance of B then the nerve cell endbulbs from the strong coupling regions would allow the free acetycholine to take effect on the subsynaptic membranes before acetycholine-cholinesterase annihilation took place. The alternative mechanism is to simply passively store

cholinesterase along the glial membrane in a form suited to immediate annihilation with diffusing acetycholine. The latter alternative has the advantage that cholinesterase will not tend to flood the subsynaptic cleft during long input bursts. But even this flooding tendency can be controlled if the dual neural-glial control mechanisms produce the proper relative amounts of acetycholine and cholinesterase, as we have supposed. The experimental evidence does not seem to exclude either one of these possibilities conclusively.

The important qualitative conclusion arising from this discussion is that the neural-glial duality is a duality not only in terms of local cellular control mechanisms, but also in terms of the chemicals--determined by these control mechanisms--which these two cell structures provide in active neural interactions. If we assign a "+" sign to the nerve cell and its excitatory acetycholine store, then we must assign a "-" sign to the glial cell and its inactivating cholinesterase store. The neural-glial duality thus represents a splitting of a chemical process into two oppositely polarized functional parts which are sustained by two disjoint structural carriers. This splitting preserves the pleasant properties that (1) "opposites attract"--in terms of the formation of new functional boundaries, and that (2) "opposites neutralize one another"-- in terms of the acetycholine-cholinesterase annihilation. These remarks illustrate the fact that neural-glial duality is but one species of field-antifield duality.

In keeping with neural-glial duality, we expect that if the control mechanism of a given nerve cell does not produce a specific transmitter, then the impinging glial (or Schwann) cell will not produce a specific transmitter inhibitor. The sheath must always produce the substance appropriate for the annihilation of the c-controlled quantity which excites the subsynaptic membranes of the system. In the case of sheaths wrapped about axons that release no transmitter of any sort, we expect the cellular control mechanisms of the sheath to eliminate transmitter inhibitor production. This conclusion follows immediately from the fact that a c=0 value along an axon produces a zero value in the glial mechanism which controls transmitter inhibitor production.

The neural-glial duality is a useful concept, not only with which to explore the functioning of cellular systems, but also with which to be able to glean valuable dynamical information from a mere inspection of the relative geometrical relations between neural cells, their sheaths, and transmitter

concentrations in electronmicrographs. The proper setting for a quantitative study of this duality is a biochemical analysis, for which one c parameter is insufficient. Such a study, in its complete details, leads beyond the scope of this paper, although in the following sections we shall see that some information about biochemical ensembles can indeed be gleaned by natural extensions of our system. It is nonetheless remarkable that one can go so far towards qualitative systemic conclusions, and the reduction of the problem to a small number of alternative possible solutions, by merely asking how to draw a boundary!

120. Local Membrane Systems: Linear and Chaotic Coupling, and Intrinsic Measuring Devices

Before leaving entirely the subject of the decomposition of cellular ensembles into local units, we now sketch several idealized examples of local cellular decompositions. Our concern to the present has been largely limited to discussions of idealized volume conductors, which obey equations of the form

$$ds_i^+/dt = \alpha_i^+(M_i - s_i^+)I_i^+ - \alpha_i^-(s_i^+ - m_i)I_i^-,$$

$$dc_{ij}^+/dt = \gamma_{ij}^+(A_{ij} - c_{ij}^+)\max(s_i^+(t-t_{ij}^+) - u_{ij}^+, 0)\max(s_j^+(t-T_{ij}^+) - U_{ij}^+, 0)$$

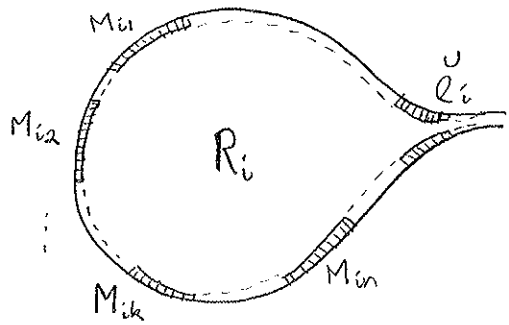
$$- \gamma_{ij}^-(c_{ij}^+ - a_{ij})(M_i - \max(s_i^+(\ldots)\ldots)(M_j - \max(s_j^+(\ldots)\ldots),$$

$$I_i^+ = \bar{I}_i^+ + r\Sigma_k \max(s_k^+(t-t_{ki}^+) - u_{ki}^+, 0)p_{ki}^+ c_{ki}^+,$$

which may be dualized in an obvious fashion to complete the system. We have included the mappings $c_{ij} \longrightarrow c_{ij} - a_{ij}$ and $s_j \longrightarrow \max(s_j(t-T_{ij}^+) - U_{ij}^+, 0)$ to emphasize that each component sub-process possesses its own threshold and transmission time lag. Indeed, one can easily envisage situations for which a mapping such as $c_{ij} \longrightarrow \max(c_{ij} - \Delta_{ij}, 0)$ is also appropriate, although it is often safe to assume that threshold dependencies in intercellular transmissions are principally carried by the point strength functions, to whose fluctuations the c_{ij} functions readily respond.

When one examines a cellular system that is not, in first approximation, a volume conductor, several important changes in the embedding equations occur.

Thus, let us be given an idealized cell diagram



R_i is a cell with total embedding spaces $M_{i1}, M_{i2}, \ldots, M_{in}$, and an output embedding space $\breve{l}_i$. The M_{ik} correspond to the membrane regions which receive, and actively respond to, the discharges of afferent fiber terminals. Denote the region corresponding to M_{ik} by $\mathcal{M}_{ik}$. The first problem that arises is: how do the various $\mathcal{M}_{ik}$ influence one another? The simplest departure from an idealized volume conductor is the following. When $\mathcal{M}_{ik}$ is activated, the state of many small subsystems of $\mathcal{M}_{ik}$ is altered. Some functional of this activation propagates to $\mathcal{M}_{ij}$ and tends to exhaust M_{ij}. We suppose that a mean propagation time t^i_{kj} and a mean propagation coefficient r^i_{kj} can be assigned to this process. Then the equation for the strength function s_{ij} of $\mathcal{M}_{ij}$ takes the form

$$ds^+_{ij}/dt = \alpha^+_i (M_{ij} - \Sigma_k s^+_{ik}(t - t^i_{kj}) r^i_{kj}) I^+_{ij} - \alpha^-_i (\Sigma_k s^+_{ik}(t - t^i_{kj}) r^i_{kj} - m_{ij}) I^-_{ij}$$

where we have replaced the s_{ij} term in the $(M_{ij} - s_{ij})$ and $(s_{ij} - m_{ij})$ terms of a volume conductor by weighted sums of temporally displaced s_{ik} terms. When $\mathcal{M}_{ik}$ transmits to $\mathcal{M}_{ij}$ in this way, we say that the directed pair $(\mathcal{M}_{ik}, \mathcal{M}_{ij})$ is linearly coupled. In particular, we can often suppose that in a cell for which all $(\mathcal{M}_{ik}, \mathcal{M}_{ij})$ are linearly coupled, the cellular propagation mechanism is so homogeneous that all $(\mathcal{M}_{ik}, \breve{l}_i)$ are also linearly coupled. In such a linearly coupled situation, the excitatory input I^+_{ij} from $\bigoplus_k R_k$ to $\mathcal{M}_{ij}$ is given by

$$I^+_{ij} = \bar{I}^+_{ij} + r \Sigma_k \max(\bar{s}^+_k(t - t^+_{k;ij}) - u^+_{k;ij}), 0) p^+_{k;ij} c^+_{k;ij},$$

where $\bar{s}^+_k$ is the strength function of the $\breve{l}_k$ region of R_k. By linear coupling,

$$\bar{s}^+_k = \Sigma_j s^+_{k(\breve{l}_k)}(t - t^k_{j(\breve{l}_k)}) r^k_{j(\breve{l}_k)}$$

where $(\breve{l}_k)$ is the index of the $\breve{l}_k$ region of R_k. To illustrate how these equations

reduce to the case of a volume conductor in a particularly simple case, let $r^i_{kj} = r_i$, $u^+_{k;ij} = u^+_k$, $t^i_{kj} \approx 0$, and $M_{ik} = r_i M_i$. Then, defining $s^+_i = \Sigma_k s^+_{ik}$ and $I^+_i = \Sigma_k I^+_{ik}$,

$$ds^+_{ik}/dt = \alpha^+_i (M_{ik} - \Sigma_j s^+_{ij} (t - t^i_{jk}) r^i_{jk}) I^+_{ik} - \ldots$$

becomes, upon adding over all k,

$$ds^+_i/dt \quad \alpha^+_i (r_i M_i - r_i s^+_i) I^+_i - \ldots$$

$$(\alpha^+_i r_i)(M_i - s^+_i) I^+_i - \ldots,$$

which has the embedding spaces of a volume conductor. Since, moreover, $\check{1}_i$ is linearly coupled to $\oplus_k \mathcal{M}_{ik}$, $\bar{s}^+_i \approx r_i s^+_i$, just so long as $\check{1}_i$ does not receive direct induced external inputs. Consequently,

$$I^+_{ij} = \bar{I}^+_{ij} + r\Sigma_k \max(r_k s^+_k (t - t^+_{k;ij}) - u^+_{k;ij}, 0) p^+_{k;ij} c^+_{k;ij}$$

whence, if the transmission time from any $\check{1}_k$ to each $\mathcal{M}_{ir}$ in $\oplus_k \mathcal{M}_{ik}$ is approximately the same,

$$I^+_i = \Sigma_j I^+_{ij} = \bar{I}^+_i + r\Sigma_k \max(r_k s^+_k (t - t^+_{ki}) - u^+_k, 0) \Sigma_j p^+_{k;ij} c^+_{k;ij},$$

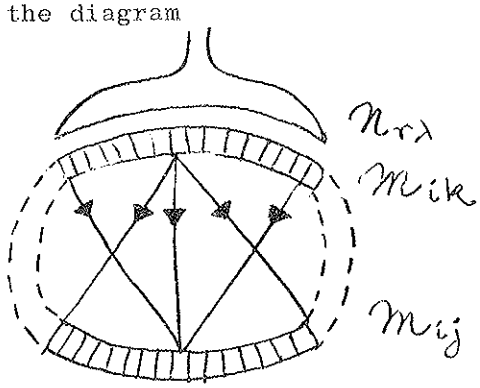
so that if $p^+_{k;ij} = p^+_{ki}$,

$$I^+_i = \bar{I}^+_i + r\Sigma_k \max(s^+_k (t - t^+_{ki}) - \bar{u}^+_k, 0) \bar{p}^+_{ki} c^+_{ki},$$

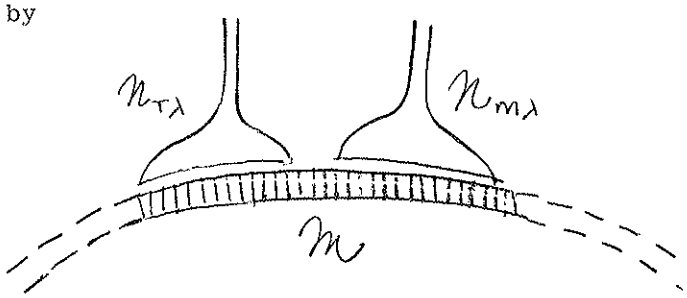
where $\bar{u}^+_i = u^+_i / r_i$, $\bar{p}^+_{ki} = r_k p^+_{ki}$, $c^+_{ki} = \Sigma_j c^+_{k;ij}$, and $s^+_k = \Sigma_j s^+_{kj}$. We have hereby recaptured the point strength equations for a volume conductor. Reversing this argument, we see that, given any system $\mathcal{M} = \oplus_{j,k} \mathcal{M}_{jk}$, we can decompose $\mathcal{M}$ into a system of volume conductors, in first approximation, if there exists a partition $\oplus_r \mathcal{B}_r = \mathcal{M}$ such that for every $\mathcal{B}_r$, (1) all pairs $(\mathcal{M}_{jk}, \mathcal{M}_{ml})$ of elements of $\mathcal{B}_r$ are linearly coupled, (2) there exists a $\mathcal{M}_{jk} \in \mathcal{B}_r$, denoted by $\check{1}_r$, such that all $\mathcal{M}_{ml} \in \mathcal{B}_r$ transmit to other $\mathcal{B}_l$ only by way of $\check{1}_r$, and (3) whenever necessary, structural field functions (such as connection densities and transmission time lags) between the sets $\mathcal{B}_r \setminus \{\check{1}_r\}$ and $\{\check{1}_m\}$ factor one another, at least approximately. One naturally seeks the maximal

partition $\bigoplus_r \mathcal{B}_r$ for which (1)-(3) hold.

In order to see when linear coupling holds, it is necessary to directly study the transport processes which carry $\mathcal{M}_{kj} \to \mathcal{M}_{kr}$ and $\overset{\smile}{1}_i \to \mathcal{M}_{kj}$ transmissions. Consider the diagram



in which an $\mathcal{N}_{r\lambda} \to \mathcal{M}_{ik} \to \mathcal{M}_{ij}$ transmission is schematically represented. The variables s_{ik} and s_{ij} are definable only with respect to the entire scheme of inputs and outputs which involve $\mathcal{M}_{ik}$ and $\mathcal{M}_{ij}$, respectively, whether these inputs be from adjoined nodes $\mathcal{N}_{r\lambda}$, from intracellular sources, or from other pertinent systems. In particular, in the above diagram, we must suppose that $\overset{\smile}{1}_r \to \mathcal{N}_{r\lambda}$ transmission excites the entire $\mathcal{N}_{r\lambda} - \mathcal{M}_{ik}$ border and that this broad excitatory front is propagated to all of $\mathcal{M}_{ik}$. Had the above diagram been replaced by



where $\mathcal{N}_{r\lambda}$ and $\mathcal{N}_{m\lambda}$ are independently excitable, it would have been necessary to split $\mathcal{M}$ into two subsystems, each with its own s function. Within the context of the present diagrams, these considerations might seem quite obvious, for the diagrams are intended to emphasize precisely those aspects of the construction of systems for which a point strength function exists. It must be remembered, however, that every $\mathcal{M}_{ik}$ region is composed of a very large number of relatively localized chemical subsystems of great complexity. The point strength functions which we are considering are not defined over each such subsystem, but are inextricably tied to the ensemble behavior of large sets of such subsystems. The justification for searching for relatively

macroscopic ensemble variables of this type lies in the fact that the input-output mechanisms which presently concern us themselves distinguish only this ensemble behavior. In this sense, the variables of the system which we are constructing depend for their definition on the complete set of measuring devices that operate on a given level of dynamical graining. These measuring devices are, in the present case, the intrinsic physiological input and output mechanisms themselves. If we, instead, introduced a microelectrode with particular ionic affinities, the system of inputs and outputs would be augmented. Such a microelectrode need not be sensitive to a macroscopic point strength function, while it might well be sensitive to local ionic fluxes which are averaged out in the ensemble behavior that defines a point strength function. One must be exceedingly careful in studying such interactions to clearly distinguish between the set of variables which a well-trained human theorist can detect, the variables which a microelectrode can detect, and the variables which various intrinsic physiological systems can detect. In particular, merely utilizing measuring devices with highly localized ranges and ionic specificities need not provide the most revealing source of information about intercellular transmissions, for even the cellular output mechanisms themselves are often constructed to be able to ignore such variables in their ensemble behavior. Of course, the physiological system itself can distinguish or eliminate all variables in a perfectly intrinsic way. The central point is that human beings thsemselves, as measuring devices several times removed from a physiological system, have a highly limited information transmission capacity on the conscious symbolic level. The notion of a variable is itself one attempt to use this capacity in an efficient way--which shall be further explicated in a later section--, and this notion itself can only be defined, ultimately, by considering the input-output relations of many individuals over time periods that are exceedingly long compared to the physiological relaxation times which we are considering. Since we cannot hope to use this capacity to speak about "all" interactions in a physical system at any time, we must find rules whereby we can <u>ignore</u> most of these variables at any one time, yet can <u>extend</u> the given system to a more comprehensive one at a later time. The passage from a system of volume conductors to a system of linearly coupled local systems is one such extension. In the presence of a microelectrode, interpolated between the physiological system and the human, socially trained "measuring device," even

the s_{ik} on the right hand side of the equation giving s_{ik} might have to be replaced by a functional of s_{ik}; for example, in the simplest cases, by

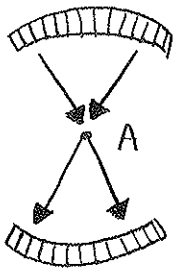
$$T_t(s_{ik}) = \int_0^t s_{ik}(t-w)\,dK(w).$$

Since nontrivial functionals always call for a field extension, it will then be necessary to dilate the surface-like membrane in an appropriate way. We shall later see that the natural dilation of the membrane is necessarily a system of parallel layers, such that every subsystem of the membrane becomes a rectangular parallelopiped which penetrates all of the membranous layers. Each parallelopiped system will carry a collection of local chemical variables whose interactions roughly produce $T_t(s_{ik})$ in the mean, taken over all parallelopipeds in $\mathcal{M}_{ik}$, where we ignore relatively small spatio-temporal differences on the parallelopiped level, just as we did in collapsing linearly coupled systems $\mathcal{M}_{jk}$ into volume conductors $\mathcal{R}_r$ according to (1)-(3).

These extensions will be given in increasing detail in later sections, but it is important to acquire a feeling for the method of attack at an early stage. One can think of this method as a reply to the question: How does an organism constructed according to a hierarchical plan, in which various levels of the hierarchy distinguish different varieties of input experiences, interact with other systems whose own construction is hierarchical in a similar sense? The method is therefore unthinkable in the absence of a particular organism, which is the ultimate measuring device of the problem. If a particular organism cannot distinguish some environmental fluctuation, this fluctuation is irrelevant to the analysis, just as if an impinging atomic "particle" cannot distinguish the fine structure of an atomic nucleus, this nucleus can be contracted to an "elementary" unit, surrounded by a resultant, contracted "field" which exhibits the interaction features that the particle can distinguish.

With these general remarks in mind, we can return to the case of linear coupling with a better understanding of why most of the cellular interactions are completely invisible to the analysis. In particular, we see that the distribution of $\mathcal{N}_{r\lambda} \to \mathcal{M}_{ik}$ transmissions must be broadly spread over many $\mathcal{M}_{ik}$ subsystems and that the position of subsystems attaining particular dynamical states must be distributed chaotically within $\mathcal{M}_{ik}$ relative to the other structural carriers of variables of the problem. Moreover, the method of propagation of potential changes, induced by each $\mathcal{M}_{ik}$, to other cellular subregions must be at least as

finely grained as the individual $\mathcal{M}_{ij}$ subsystems themselves. Otherwise, new $\mathcal{M}_{ij}$ subsystems could not be made to respond to the propagated potentials. Since the local fluctuations within the flow of potential between $\mathcal{M}_{ik}$ systems are small relative to the grain of these subsystems, this flow can be viewed as proceeding from subsystem to subsystem along paths, or rays, whose shape depends on intrinsic cellular formations. In the simplest case, these rays are straight lines, for the potentials pass right through local cellular systems without deformations that are significant to the $\oplus_k \mathcal{M}_{ik}$ level. These rays will also be supposed to satisfy a superposition property, for otherwise, it would be necessary to study systems with diagrams like



for which A must itself be viewed as a local $\mathcal{M}$ system. For specificity, it is assumed that the rays obey a diffusion equation, as is familiar from discussions in the literature of electrotonic potential propagation.

The manner in which this diffusion interacts with local $\mathcal{M}_{ij}$ subsystems can greatly alter the embedding equations for these subsystems. Thus, instead of a linear coupling, one often finds an equation like

$$ds_{ik}^{+}/dt = \alpha_i^{+}(M_{ik} - s_{ik}^{+})(\max(u_i(x_{ik}) - P_{ik}^{eq}, 0) + \ldots)$$
$$- \alpha_i^{-}(s_{ik}^{+} - m_{ik})(\max(P_{ik}^{eq} - u_i(x_{ik}), 0) + \ldots),$$

where $u_i = u_i(x,t)$, $x \in \mathbb{R}^3$, is the solution of the diffusion equation

$$\partial u_i / \partial t = \mathcal{D}_i(u_i; s_{il}^{+}, s_{i2}^{+}, \ldots, s_{in}^{+}).$$

Here P_{ik}^{eq} is the equilibrium potential of $\mathcal{M}_{ik}$, and the vector $(s_{il}, s_{i2}, \ldots, s_{in})$ provides a complete catalogue of boundary source conditions. x_{ik} is the position of $\mathcal{M}_{ik}$ in the cell. When an equation of this type holds, as above, for all ordered pairs $(\mathcal{M}_{ik}, \mathcal{M}_{ij})$, we say that the system $\oplus_r \mathcal{M}_{ir}$ is <u>chaotically coupled</u>. The two situations of linear and chaotic coupling are extremal points in the spectrum of possible couplings of local cellular systems. Mixtures of the two types of coupling often arise. When this happens, one finds equations of the

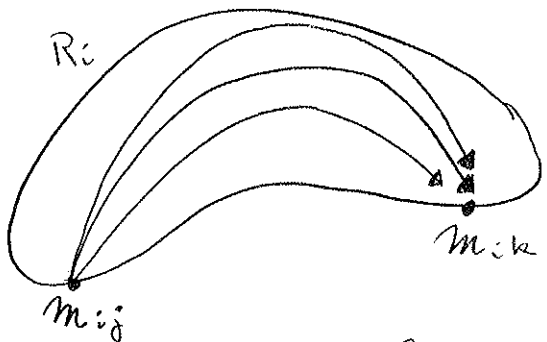
form

$$ds^+_{ik}/dt = \lambda_L\left[\alpha^+_i(M_{ik}-\Sigma'_j\, s^+_{ij}(t-t^i_{jk})r^i_{jk})I^+_{ik} - \alpha^-_i(\Sigma'_j s^+_{ij}(t-t^i_{jk})r^i_{jk}-m_{ik})I^-_{ik}\right]$$

$$+\lambda_C\left[\alpha^+_i(M_{ik}-s^+_{ik})(\max(u_i(s_{ik})-P^{eq}_{ik},0)+I^+_{ik}) - \alpha^-_i(s^+_{ik}-m_{ik})(\max(P^{eq}_{ik}-u_i(s_{ik}),0)+I^-_{ik})\right],$$

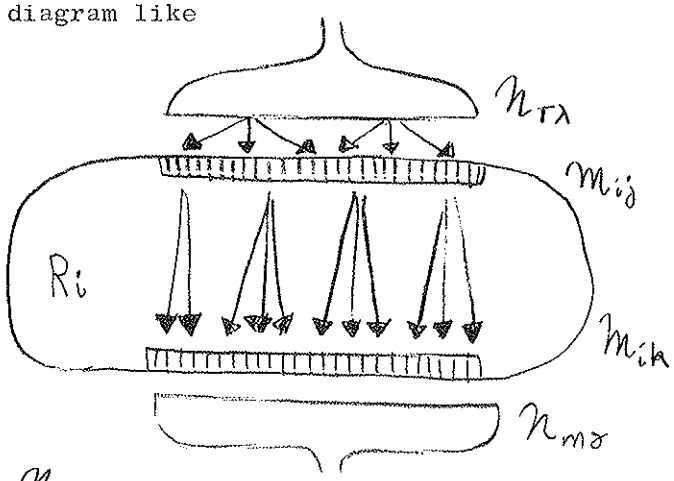
where $\partial u_i/\partial t = \mathcal{F}_i(u_i;(s^+)'')$, and $\lambda_L + \lambda_C = 1$, $\lambda_L, \lambda_C \geq 0$. Here Σ'_j means that the summation is restricted to the $\mathcal{M}_{ij}$ systems which are at least partially linearly coupled to $\mathcal{M}_{ik}$, and $(s^+)''$ means that the vector of boundary sources is restricted to the <u>complementary</u> set of $\mathcal{M}_{ij}$ systems which are at least partially chaotically coupled to $\mathcal{M}_{ik}$. The r^i_{jk} in this equation are also adjusted to weight the extent to which $(\mathcal{M}_{ij}, \mathcal{M}_{ik})$ is linearly coupled, and the $(s^+)''$ vector gives, not the total s^+_{ir} value of a chaotically coupled $(\mathcal{M}_{ir}, \mathcal{M}_{ik})$ pair, but only that fraction $R^i_{rk}s^+_{ir}$ of s^+_{ir} which contributes to the chaotic coupling.

When does a chaotic coupling arise? A partial answer is suggested by the fact that, in the term $u_i(s_{ik})$, we must refer to the position of $\mathcal{M}_{ik}$ as a point, x_{ik}, in $\mathbb{R}^3$. Thus, if $(\mathcal{M}_{ij}, \mathcal{M}_{ik})$ are almost exclusively chaotically coupled, $\mathcal{M}_{ij}$ must look like a point to $\mathcal{M}_{ik}$. This can happen either if $\mathcal{M}_{ij}$ and $\mathcal{M}_{ik}$ are separated by a distance that is large relative to the diameters of these systems, or if the transport process carrying potential from $\mathcal{M}_{ij}$ to $\mathcal{M}_{ik}$ is so inhomogeneous (large fluctuations) that the individual $\mathcal{M}_{ij}$ subsystems, as sources of potential variations, are entirely indistinguishable at $\mathcal{M}_{ik}$. Thus, the averaging rule underlying a chaotically coupled pair $(\mathcal{M}_{ij}, \mathcal{M}_{ik})$ is: contract each of $\mathcal{M}_{ij}$ and $\mathcal{M}_{ik}$ to a point, and let the transport of potential occur over the contracted space. We therefore find diagrams like

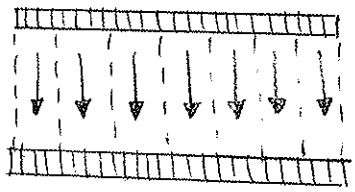


In particular, if $\mathcal{M}_{ik}$ lies in the cell body, $\mathcal{M}_{ij}$ will more likely be chaotically coupled to $\mathcal{M}_{ik}$ if it is in a distal dendrite than if it lies right across from $\mathcal{M}_{ik}$.

For linear coupling, the rules for averaging are different. Here one thinks of a diagram like



The idea is that $\mathcal{N}_{r\lambda}$ transmission alters the polarity of the $\mathcal{M}_{ij}$ subsystems in the usual way. These polarity changes are immediately translated into potential changes within R_i. The potential propagates in such a way that the polarity of the $\mathcal{M}_{ik}$ subsystems is altered in the same way as the polarity of the $\mathcal{M}_{ij}$ subsystems was, up to a time lage and an r^i_{jk} coefficient which gives the expected decrement in potential, per unit region, due to the transport process. Thus, instead of contracting each $\mathcal{M}_{ij}$ and $\mathcal{M}_{ik}$ independently, for linear coupling we envisage generalized tubes, encompassing specific $\mathcal{M}_{ij}$ and $\mathcal{M}_{ik}$ subregions and the transport paths leading from the $\mathcal{M}_{ij}$ subregions to the $\mathcal{M}_{ik}$ destination:



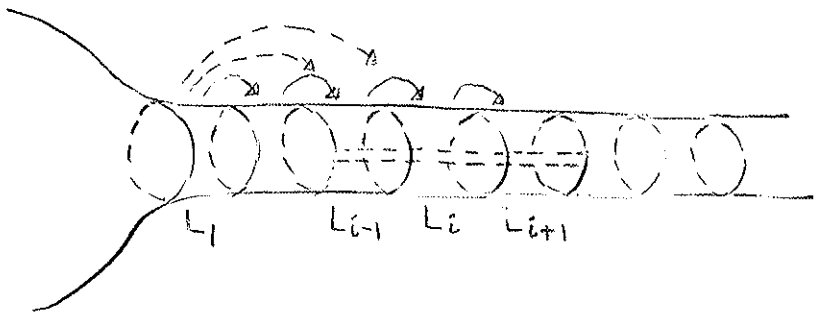
The space of all such tubes is the proper space over which to average, and when we do so we find a linear coupling. One can therefore think of $\mathcal{N}_{r\lambda}$ as applying an input directly to $\mathcal{M}_{ik}$ as well as to $\mathcal{M}_{ij}$; $\mathcal{M}_{ij}$ is a way-station in delivering this input which does not destroy the local structure of the input paths.

Both linear and chaotic coupling share a common important feature. For both, $\mathcal{N}_{r\lambda}$ transmitter releases an intrinsic membrane mechanism that alters the polarity of local $\mathcal{M}_{ij}$ subsystems in an amount proportional to the intensity of the $\mathcal{N}_{r\lambda} \rightarrow \mathcal{M}_{ij}$ transmission. This alteration in $\mathcal{M}_{ij}$ polarity is immediately transformed into a local cellular potential change. The cellular potential change propagates to another $\mathcal{M}_{ik}$ system, where it alters the polarity of its

subsystems in an amount proportional to the value of the transmitted potential. This polarity change, induced from the cellular interior, simultaneously alters the receptivity of the $\mathcal{M}_{ik}$ membrane to $\mathcal{N}_{m\gamma}$-induced transmissions from the cellular exterior. It is precisely this fact, whereby impinging interior potential changes and exterior transmitter processes induce equivalent effects in the membrane ensemble, that permits a harmonious interaction of many input sources in producing well-organized cellular outputs. Linear and chaotic coupling both obey these laws; they differ only in the order in which various segments of $\mathcal{M}_{ij} \oplus (\mathcal{M}_{ij} \to \mathcal{M}_{ik}) \oplus \mathcal{M}_{ik}$ are pre-averaged. In summary, linear coupling may be thought of as the coupling which arises when two local membrane systems are brought "infinitely close" together with respect to the intracellular transport process carrying potential variations, while chaotic coupling is the coupling which arises when these systems are taken "infinitely distant" from one another with respect to the membrane transport process.

121. Membrane Decompositions and Axonal Transmission

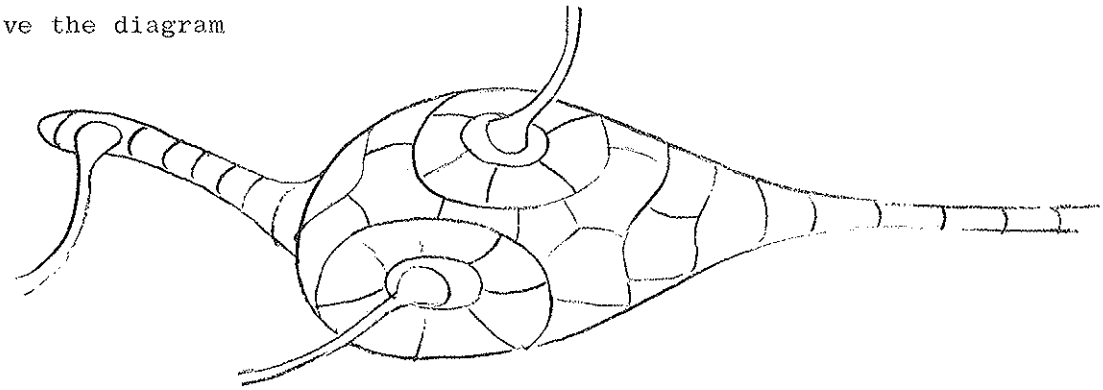
As a particular application of these ideas, notice that a region which is extremely well suited for linear coupling is the cell axon. Conceptually decompose the axon into a series of cylindrical segments L_i:



Each strip of L_i is in a perfect position to become linearly coupled with the adjacent strips in L_{i-1} and L_{i+1}. The cellular potential usually flows from L_i to L_{i+1}, by virtue of the asymmetric position of the cell body relative to $\oplus_i L_i$. Indeed, since the cell body is the usual physiological source of inputs to the axon, the total embedding space of L_{i-1} is usually considerably exhausted from immediately prior transmission when L_i is most active. The propagation of an impulse from L_1 to a terminal node can thus, in first approximation, be viewed as a succession of linear coupling along the chain $L_1 \to L_2 \to \cdots \to L_n \to \mathcal{N}$ of cylindrical strips. Increasingly chaotic secondary couplings directly join

L_i and L_j as $|i-j|$ is allowed to become large. The possibility of introducing antidromic impulses becomes obvious from this viewpoint, since the directionality of the input flow depends entirely on initial input asymmetries. The chain of linear couplings provides, in first approximation, a quantitative alternative to the classical saltatory hypothesis which eliminates many of its difficulties.

By continuing this process of membrane decomposition in a natural way, one finally arrives at a membrane which is fully partitioned into local subsystems whose shape depends on geometrical symmetries and on the dynamical laws (for example, $\mathcal{N} \to \mathcal{M}$, $\mathcal{M} \to \mathcal{M}$ transmissions) which interconnect these subsystems. Thus, we have the diagram

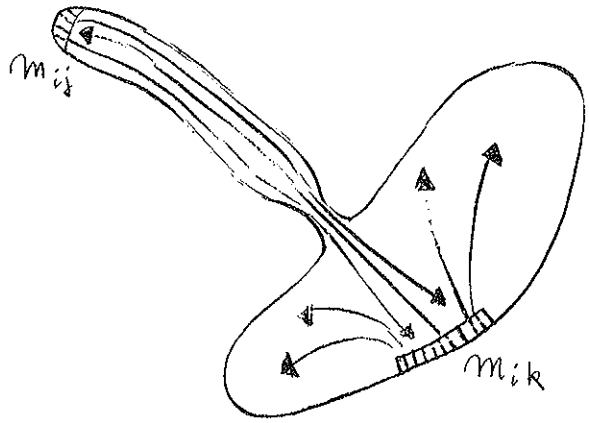


It is important to realize that we have been forced to pay a price for the possibility of completely decomposing the membrane into subsystems and viewing all interactions of these subsystems as combinations of linear and chaotic couplings. The price is manifest in the fact that we cannot even begin to speak about cellular dynamics until we make a choice of which coupling type holds between particular subsystems, and this choice is often based on intuitive and extratheoretical grounds. Indeed, the very choice of subsystems depends on our ability to guess when a certain kind of coupling will hold between the subsystems that we choose. The reason for this difficulty is that we are scrupulously avoiding a study of the fine structures of the membrane and the cell body, both of which are extraordinarily complex and involve large numbers of local variables that are either irrelevant or average out to form macroscopic variables in the ensemble picture. It is, however, only in terms of local variables that one can discuss how to divide the membrane and which averaging order to use in an intrinsic way. Since the subject matter of this paper is psychological, it is perhaps not unappropriate to remark that the possibility of making good guesses about membrane decompositions without becoming blinded by a plethora of local variables is tied to the fact that the joint information transmission

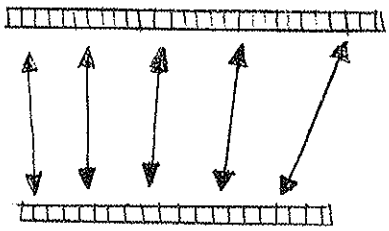
capacity of the human visual and symbolic systems is often much higher than that of the symbolic systems alone. The partition of the membrane into local systems is thus a particularly human convenience and liability; one cannot say that such a partition is "really there," yet without it, it becomes difficult for a human measuring device to draw any inferences whatever about the physiological ensemble. Since only symbols are accurately conveyed by scientific papers, however, adequate clues to a reader for performing these partitions himself can only come from a study of many specific examples. Further examples will follow in later works. The situation is somewhat simplified by the fact that it is usually not necessary to consider the entire membrane in the simplest physiological interactions, but only those subregions which receive direct transmissive inputs and which generate direct outputs. For these regions, the partition into subsystems is usually quite obvious. Although coupling between these subregions may be mediated by other membrane subsystems, one can often, in first approximation, absorb these effects into coefficients of the restricted problem.

122. Linear Coupling and Transport Reversibility

In performing membrane partitions, it is always interesting to ask: when does $r^i_{jk}=r^i_{kj}$ in a linear coupling? Since r^i_{jk} measures the expected propagation coefficient, per unit area of $\mathcal{M}_{ij}$ and of $\mathcal{M}_{ik}$, of the coupling, $r^i_{jk}=r^i_{kj}$ means that the transport process between $\mathcal{M}_{ik}$ and $\mathcal{M}_{ij}$ is reversible. Reversibility should not be considered an obvious property, as the following diagram shows:



Nonetheless, for this diagram we would expect $(\mathcal{M}_{ij}, \mathcal{M}_{ik})$ to be, at least partially, chaotically coupled! On the other hand, for the "canonical" linear coupling diagram



reversibility (per unit area) seems quite obvious. Thus, to the extent that we treat linear coupling as an idealized kind of membranous interaction, $r^i_{jk}=r^i_{kj}$ becomes a plausible working hypothesis, as does $t^i_{jk}=t^i_{kj}$. Hence the matrices $\mathcal{R}_i=\{r^i_{jk}\}$ and $\tilde{\mathcal{T}}_i=\{t^i_{jk}\}$ are symmetric matrices. The diagonal terms in $\mathcal{R}_i$ are all 1, while those in $\tilde{\mathcal{T}}_i$ are all 0.

123. Local Control Couplings

The introduction of local coupling systems brings with it new c coupling possibilities. The strong c couplings with which we originally dealt refer to a process that can be spread over the entire joint node-recipient cell system. Since volume conductors were used, the recipient cell was contracted to a single point, as was the node, whence local variations in the c coupling process were completely obscured. In particular, it was possible to let c determine a transmitter production process in the node without the need to independently study the carrier of c in the recipient cell. From the purview of a collection of local $\mathcal{M}_{ik}$ systems, however, the problem of the distribution of c couplings within the recipient cell becomes an important issue.

To the extent that each $\mathcal{M}_{ik}$ behaves locally like a volume conductor, our task is relatively simple. For example, if all $\mathcal{M}_{ik}$ are linearly coupled, then the $\mathcal{M}_{r\lambda}\to\mathcal{N}_{r\lambda}\to\mathcal{M}_{ik}$ c coupling equation becomes, for the simplifying choice of m_{ik} as the $\mathcal{M}_{ik}$ strong coupling threshold:

$$dc_{r\lambda;ik}/dt=\gamma^{+}_{r\lambda;ik}(A_{r\lambda;ik}-c_{r\lambda;ik})\max(s_{r\lambda}(t-t_{r\lambda;ik})-u_{r\lambda;ik},0)(\Sigma_j s_{ij}(t-t^i_{jk})r^i_{jk}-m_{ik})$$

$$-\gamma^{-}_{r\lambda;ik}(c_{r\lambda;ik}-a_{r\lambda;ik})(M_{r\lambda}-\max(s_{r\lambda}(t-t_{r\lambda;ik})-u_{r\lambda;ik},0))(M_{ik}-\Sigma_j s_{ij}(t-t^i_{jk})r^i_{jk}),$$

while if all $\mathcal{M}_{ik}$ are chaotically coupled, the term $\Sigma_j s_{ij}(t-t^i_{jk})r^i_{jk}$ is everywhere replaced by s_{ik} alone, with the understanding that $\mathcal{M}_{ij}\to\mathcal{M}_{ik}$ transport is once again determined by evaluating $u_i(s,t;s_{il},\ldots,s_{in})$ at $x=x_{ik}$. Mixtures of linear

and chaotic coupling call for the term $\lambda_L \sum_j s_{ij}(t-t^i_{jk})r^i_{jk} + \lambda_C s_{jk}$, with the usual conventions.

When all coupling changes are not directly attached to individual $\mathcal{N}_{r\lambda} \oplus \mathcal{M}_{ik}$ systems, the situation is more complicated. Indeed, one can easily envisage a system for which strong coupling in a particular $\mathcal{N}_{r\lambda} \oplus \mathcal{M}_{ik}$ system induces a change in recipient cellular control mechanisms that becomes broadly dispersed over the cellular volume and contributes secondarily to new $\mathcal{N}_{r'\lambda'} \oplus \mathcal{M}_{ij}$ couplings, instead of directly, as in the above examples. What is called for here is a splitting of each c function into (at least) two functions, one of which, c^1, is attached to control processes in $\mathcal{N}_{r\lambda}$, while the other, c^2, is attached to control processes in $\oplus_j \mathcal{M}_{ij}$. Since the data seems to be so inconclusive on these matters, we will merely sketch a small number of interesting alternatives to strong coupling, as a guide to future research.

To simplify matters, we study the coupling associated with a single $\mathcal{M}_{ik}$ subsystem, and suppose that the c^2 alterations propagate to other $\mathcal{M}_{ij}$ subsystems according to an appropriate mixtures of linear and chaotic couplings, either of the various s_{ij}'s or of the c^2 functions themselves. We can therefore use but two indices--m for the nodal source, and k for the $\mathcal{M}_{ik}$ system activated by this source. In this situation, the most profound splitting of c_{mk} into c^1_{mk} and c^2_{mk} is given by the equations

$$dc^1_{mk}/dt = \gamma^+_{mk1}(A^1_{mk}-c^1_{mk})\max(s_m(t-t_{mk})-u_{mk},0)$$

$$-\gamma^-_{mk1}(c^1_{mk}-a^1_{mk})(M_m-\max(s_m(t-t_{mk})-u_{mk},0),$$

and

$$dc^2_{mk}/dt = \gamma^+_{mk2}(A^2_{mk}-c^2_{mk})(s_k-m_k)-\gamma^-_{mk2}(c^2_{mk}-a^2_{mk})(M_k-s_k),$$

where the nodal-recipient cell transmission term $\max(s_m(t-t_{mk})-u_{mk},0)p_{mk}c_{mk}$ is replaced by $\max(s_m(t-t_{mk})-u_{mk},0)p_{mk}c^1_{mk}c^2_{mk}$. Here each c^i_{mk} function, i=1,2, is determined entirely by processes directly responsive to the potential over either node or recipient cell, but not both. Nodal-recipient cell transmission, on the other hand, depends on the product $c^1_{mk}c^2_{mk}$. If c^1_{mk} is taken to be proportional to available transmitter density, then this transmission law means that transmitter is released in a quantity jointly proportional to the nodal potential intensity and transmitter concentration. This quantity, in turn, activates

the subsynaptic membrane system in an amount proportional to its intensity and to the receptivity of the recipient membrane, which is determined by c^2_{mk} and, hence, by prior recipient cell activation. Suppose now that local s_{ij} variations propagate to other $\mathcal{M}_{ik}$ in one of the ways delineated above. Then activating a proper subset Ω of $\oplus_\lambda \mathcal{M}_{i\lambda}$ systems will cause c^2_{mk} to grow, for all $\mathcal{M}_{ik}$ which are nontrivially coupled to Ω. Thereafter, exciting a node of $\mathcal{M}_{ik}$ will yield a larger change in cellular potential than would have been possible in the absence of prior Ω excitation, even though this node did not in any way participate in exciting Ω. Strong coupling is hereby lost, for facilitative effects occur even if a particular node has always been quiescent in the past, just so long as its subsynaptic regions are internally coupled to previously activated subsynaptic regions. Consequently, the nodal-recipient cell systems no longer possess a concept of strict simultaneity of joint excitation; all that matters is the joint sizes of c^1_{mk} and c^2_{mk}, and each of these can be made to grow without too greatly exciting the other.

This example points out an important fact about drawing conclusions concerning whether a certain process is activated presynaptically or postsynaptically. Even here, where the coupling between presynaptic and postsynaptic cells is quite weak, a presynaptic volley will, intermediated by a transmitter release, activate c^2_{mk} to some measure. The effect of such a volley cannot, therefore, strictly be said to be presynaptic, for the c^1_{mk} and c^2_{mk} functions are indirectly coupled, whence all intercellular transmission factors will really depend on postsynaptic effects that are obscured by this presynaptic coupling. On the other hand, antidromic inputs will conceivably be much more highly restricted to the postsynaptic system, since, at least in the simplest systems of the above type, such an input does not induce as strong a presynaptic-postsynaptic coupling as the presynaptic input does. The distinction between presynaptic and postsynaptic effects thus really often is a distinction between the strength of the transmission between presynaptic and postsynaptic structures that mediates presynaptic-postsynaptic couplings which are vital to the integrity of both regions.

A somewhat more strongly coupled system is the following.

$$dc^1_{mk}/dt = s_m\left[\gamma^+_{mk1}(A^1_{mk}-c^1_{mk})\max(s_m(t-t_{mk})-u_{mk},0)\right.$$

$$\left.-\gamma^-_{mk1}(c^1_{mk}-a^1_{mk})(M_m-\max(s_m(t-t_{mk})-u_{mk},0))\right],$$

$$dc^2_{mk}/dt = rp_{mk}\max(s_m(t-t_{mk})-u_{mk},0)\left[\gamma^+_{mk2}(A^2_{mk}-c^2_{mk})s_k-\gamma^-_{mk2}(c^2_{mk}-a^2_{mk})(M_k-s_k)\right]$$

again with the transmission term employing $c^1_{mk}c^2_{mk}$ instead of c_{mk}. Here the postsynaptic potential level in the cell induces a process which nonspecifically excites the c^1_{mk} process, while the nodal potential level does the same for the c^2_{mk} process. Letting $c_{mk}=c^1_{mk}c^2_{mk}$, and choosing $A_{mk}=A^i_{mk}$, $a_{mk}=a^i_{mk}$, $\gamma^\pm_{mk}=rp_{mk}\gamma^\pm_{mk2}=\gamma^\pm_{mk1}$, and $M=M_k=M_m$ for simplicity, $i=1,2$, it follows that

$$dc_{mk}/dt = \gamma^+_{mk}(\mathcal{A}_{mk}-c_{mk})\max(s_m(t-t_{mk})-u_{mk},0)s_k$$

$$-\gamma^-_{mk}(c_{mk}-\mathcal{a}_{mk})(\mathcal{M}-\max(s_m(t-t_{mk})-u_{mk},0)s_k),$$

where

$$\mathcal{A}_{mk}=A_{mk}(c^1_{mk}+c^2_{mk}), \qquad \mathcal{a}_{mk}=a_{mk}(c^1_{mk}+c^2_{mk}),$$

and

$$\mathcal{M}=M(\max(s_m(t-t_{mk})-u_{mk},0)+s_k).$$

This is, we find a weak coupling equation whose total embedding spaces depend on the "total (instantaneous) energy" (for example, $c^1_{mk}+c^2_{mk}$) of the processes whose growth they determine. A joint nodal-cellular influence on transmission thus exists, above and beyond the mere fact of dependencies caused by transmission in the usual way from node to subsynaptic membrane, but this joint influence is still not quite as strong as a strong coupling. The addition of the premultiplying terms $\max(s_m(\ldots)-\ldots)$ and s_k on the right hand sides of the c^i_{mk} equations can be viewed as a kind of "coenzymatic catalysis" of each system by its juxtaposed system.

The next step in the construction of such catalysing systems is to suppose that they, too, obey a specific field-antifield duality principle rather than acting nonspecifically, whence, again using the above special assumptions on coefficients for simplicity, we find

$$dc^i_{mk}/dt = \gamma^+_{mk}(A_{mk}-c^i_{mk})\max(s_m(t-t_{mk})-u_{mk},0)s_k$$

$$-\gamma^-_{mk}(c^i_{mk}-a_{mk})(M_m-\max(s_m(t-t_{mk})-u_{mk},0))(M_k-s_k),$$

$i=1,2$, Thus, $c^1_{mk}=c^2_{mk}$ and we are faced with a bona fide strong coupling system with transmission term $\max(s_m(\ldots)\ldots)p_{mk}c^1_{mk}c^2_{mk} = \max(s_m(\ldots)\ldots)p_{mk}(c^1_{mk})^2$. Were we given data sufficiently fine to distinguish between transmissions of the form $\max(\ldots)p_{mk}c_{mk}$ and $\max(\ldots)p_{mk}(c^1_{mk})^2$, we could thus immediately infer the nature of the splitting in the underlying strong coupling.

We have hereby sketched a series of considerations illustrating nodal-cellular interactions at various levels of mutual dependence. Each of these levels has a specific interpretation and special theoretical virtues and deficiencies. The sketch illustrates a general method whereby new coupling types can be constructed with prescribed properties. It now remains for future experimental studies to distinguish the cases that actually occur. One fact is pervasive in all possible coupling systems: A simple flow of ions along a potential gradient does not suffice as a mechanism whereby to understand the total configuration of any coupling, especially with respect to the c couplings, which one envisages as spread throughout regions of the cell that are not restricted to the cellular membrane. One must search for relatively high energy processes, both in the cellular membrane and in the cellular interior, which involve metabolic control mechanisms with the capacity to coenzymatically induce control variations in adjacent cell systems by intercellular transport of ions.

The nodal-recipient cell interdependencies which we envisage above have important implications for an understanding of joint cellular homeostatic functions. In particular, suppose that a nontrivial nodal-cellular coupling exists which involves a pair of c^i_{mk} functions. Fluctuations in these functions represent not only variations in the state of cellular control processes, but also variations in the metabolic preparations which are necessary to realize the commands of these processes. Thus, if either all nodes leading to a single cell or the cell itself is destroyed, the total coupling system is fragmented, and one must expect compensatory changes in the surviving fragments. The qualitative features of many cellular destruction experiments, experiments which limit afferent excitation, and experiments which block specific intercellular transport paths follow directly from a study of which links in the coupling have been

eliminated by the experimental paradigm.

124. Use and Disuse, Transmitter Depletion, and Habituation

The above discussion introduces a number of delicately balanced intercellular couplings. These couplings may be placed into classes according to whether they respond more or less intensely to specific axonal inputs. Many of these effects can be read off from previous equations, but two of them--of great importance--require further discussion. The first effect relates a cellular control mechanism, say c^1_{mk}, to the amount of available transmitter, namely C^1_{mk}, in the node. In the simplest cases, where the production of transmitter is very rapid relative to all other relaxation times of the system, the approximation $C^1_{mk} = \lambda c^1_{mk}$ is adequate. λ is a positive constant. In general, however, we must expect lags between variations in the state of control mechanisms and the amount of the quantities whose production they control. We will consider only the simplest equation which accounts for these variations:

$$dC^1_{mk}/dt = u(c^1_{mk} - \lambda C^1_{mk}) - v(rp_{mk}\max(s_m(t-t_{mk})-u_{mk},0))C^1_{mk},$$

where u, λ, and v are positive rate constants, and the transmission law is $vrmax(...)p_{mk}C^1_{mk}c^2_{mk}$. That is, the production of transmitter increases at an exponential rate until it reaches an asymptote proportional to the control level c^1_{mk}, and the available transmitter is depleted at a rate proportional to its density and to the intensity of impinging afferent excitation.

This law has many important properties, which depend sensitively on the choice of rate constants. On the one hand, intense afferent excitation will tend to deplete the available transmitter, while a pause in excitation--a transient disuse of the node--will lead to a spontaneous recovery in transmitter level. Moreover, the intense afferent transmission will tend to increase c^1_{mk}, even as C^1_{mk} is decaying, whence a delicate balance between depleting and augmenting effects is introduced. Since the c^1_{mk} function is often more slowly varying than the transmitter depletion process, the tendency for C^1_{mk} to be exhausted by intense, even fairly brief bursts of excitation is evident. On the other hand, less intense and longer bursts of excitation lower the rate of transmitter depletion, thereby allowing transmitter recovery to increase, at the same time

that c^1_{mk} growth is enhanced, whence the asymptotic C^1_{mk} value also increases, so that the effective rate of C^1_{mk} recovery also increases. By properly choosing the intensity of afferent excitation and its prolongation in time, one can therefore envisage situations in which the two processes of depletion and augmentation are perfectly balanced.

When a node and its recipient cell are not excited during time intervals that are long compared to the time needed for maximal transmitter depletion, but short relative to the rate of c^1_{mk} decay, the transmitter level grows to a rather high asymptote. Consequently, after such an interval, the first few cellular spikes induced by a new nodal barrage will tend to be relatively large, but will diminish rapidly as the potential partially exhausts the superabundant transmitter supply.

In brief, whereas c^1_{mk} is potentiated by intense excitation, C^1_{mk} is depleted thereby, but tends to spontaneously receover to a density determined by the c^1_{mk} level. c^1_{mk} and C^1_{mk} thus respond in somewhat antagonistic ways to afferent excitation, although C^1_{mk} tends to reproduce the c^1_{mk} level asymptotically, up to a multiplicative constant. c^1_{mk} governs long term and initial transmission effects, while C^1_{mk} determines such short run effects as the rate of transmitter decay during prolonged bursts of excitation. The depletion of C^1_{mk} under prolonged input bursts may be viewed as a kind of presynaptic habituation to the input. These differences between c^1_{mk} and C^1_{mk} clearly show that, in discussing the effects of Use and Disuse in the nervous system, one must be careful to consider all features of the input; it is manifestly insufficient to consider either input intensity or input prolongation alone.

A simple type of habituation which is confined to the post-synaptic cell structure is given by the equations

$$dw_i/dt = k_i^+(M_i - w_i)(M_i - s_i) - k_i^- w_i s_i,$$

$$ds_i/dt = \alpha_i^+(w_i - s_i)I_i^+ - \alpha_i^- s_i I_i^-,$$

where we have set $m_i = 0$ for simplicity. w_i is slowly varying compared to s_i. w_i decreases most rapidly when s_i is most intense and recovers most rapidly when s_i is least intense. In this sense, w_i habituates to prolonged s_i excitation and spontaneously recovers in the disused state. w_i, and not M_i,

is the (habituated) total embedding space for s_i. Thus, $M_i - w_i$ may be viewed as a measure of the saturation of the capacity of the total embedding space M_i to polarize its local subsystems in response to prolonged electrical excitation. If a high s_i level is maintained, w_i gradually shrinks and thereby forces s_i to take smaller values. If the source of s_i excitation is removed, w_i spontaneously recovers. Since a cell which has been freed from excitation for a long period will have a high w_i value, delivering a new input will permit a rapid initial rise in s_i, which gradually stabilizes as w_i slowly decreases. This is the postsynaptic analog of the initial potentiation of spiking which is presynaptically induced by a high C^1_{mk} value before it, too, decays.

In summary, C^1_{mk} and w_i both decay as s excitation increases. C^1_{mk} responds rapidly to presynaptic excitation, while w_i responds slowly to postsynaptic excitation. Since s_i is often significantly coupled to c^1_{mk} values, the long-term w_i effects can propagate over the entire nodal-cellular system. Similarly, intracellular coupling between $\mathcal{M}_{ik}$ systems helps to propagate postsynaptic habituation effects to other postsynaptic regions, but here the habituation tends to free other postsynaptic regions from the excesses of an intense localized afferent discharge by reducing the s and u values which reach them.

Habituation of both the presynaptic and postsynaptic varieties here mentioned are specifically tied to particular cells. What happens when many cells are repetitively activated in such a way that all of them begin to exhibit habituation effects? If a particular space-time form whose local couplings exhibit such habituation processes is repeated in rapid sequence, the cell groups in this form will be specifically habituated. To the extent that a newly presented space-time form overlaps the cell groups of the old, its presentation will share the habituated cell groups and will thus generate a relatively meager cellular response. The presentation of forms with more nearly disjoint space-time carriers will, on the other hand, induce large potentials. Thus, the extent to which a given space-time form is afflicted with the same habituation effects as a previously presented form provides a measure of the distance between the two forms as space-time manifolds. This measure is the mean of the habituation effect of the first form divided by the mean of the habituation effect of the second form. It should also be noted that habituation can occur even if a form is repeated slowly over a fairly long time interval, for the w_i functions are slowly varying and exhibit cumulative effects.

Habituation effects can also be achieved if recurrent lateral inhibitory mechanisms are introduced in a proper fashion. How this occurs will be easier to see after we discuss global geometrical matters in greater detail in the following pages. The postsynaptic membrane habituation discussed above is the local membrane version of such global self-inhibitory interactions.

Using processes of the above type, and natural extensions thereof, one can introduce ever finer considerations into the study of local cellular interactions. In the following discussion of global geometrical matters, we will often restrict our remarks to strong couplings, both because such couplings are the simplest possible ones which conform to macroscopic behavioral requirements, and because the extant microscopic data does not definitively suggest a more complicated coupling system. The reader will be able to assign particular versions of this coupling; for example, linear or chaotic couplings, coupling with pre- and post-synaptic habituation, with little qualitative difficulty.

125. Genetic Repression, Feedback Inhibition, and Chromatolysis

Before leaving local cellular questions entirely, we consider the equation for C^1_{mk} from a deeper perspective. This perspective is rather speculative at present, for data bearing directly on the questions it raises does not seem to be readily available. Yet it presents such an appealing possibility, and such useful methods for generally looking at questions of this type, that it should not go unmentioned.

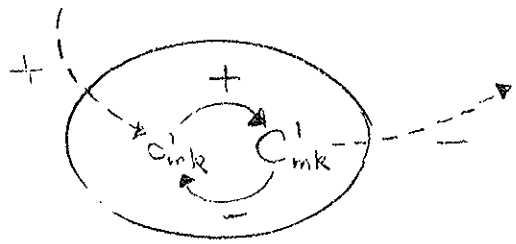
How can we interpret the growth term $u(c^1_{mk} - \lambda C^1_{mk})$ in terms of a cellular process? We suppose that the control process given by c^1_{mk} is realized by a large number of small systems distributed throughout the cell body. Not all systems which are, in potentiality, capable of producing transmitter are at any time actively doing so. The product of the number of active systems and their mean activity is given by c^1_{mk}. The term uc^1_{mk} in the equation for C^1_{mk} thus means that transmitter production occurs at each control system at a rate which depends directly on the activity of this system. The total transmitter produced is given by adding the rates of transmitter production over all such systems. Were this production process not kept in check, transmitter would accumulate at an exponential rate and would eventually upset the cell's normal functioning. The transmitter itself is therefore called upon to suppress the transmitter production process. It does this by reciprocally inhibiting some critical step, or steps, of the transmitter production sequence. We

suppose that the density of transmitter is the relevant variable in this suppression process. Since we also envisage a direct blocking of some stage of the production process, the total activity of transmitter production is no longer proportional to c^1_{mk}, but is rather proportional to the product of the mean activity and number of the uninhibited production systems, which is given by $u(c^1_{mk}-\lambda C^1_{mk})$, as in the C^1_{mk} equation.

The release of transmitter from the cell body, on the other hand, reduces the degree of transmitter blocking by an amount proportional to the quantity of transmitter released, which has been taken proportional to $r\max(s_m,\ldots)p_{mk}C^1_{mk}$. Thus, we have the full equation

$$dC^1_{mk}/dt = uc^1_{mk} - (u\lambda + vr\max(\ldots)p_{mk})C^1_{mk},$$

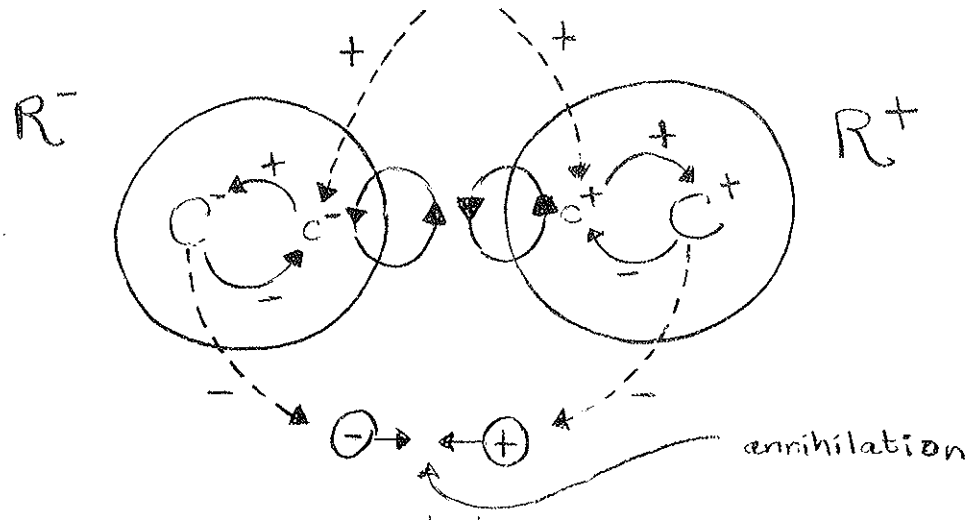
which now has the simple interpretation that the rate of total transmitter production is proportional to the effective mean activity of the transmitter producing systems multiplied by their total number. We can schematically diagram this situation as follows:



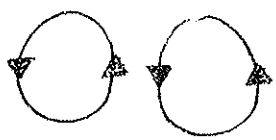
The solid arrows here represent (possibly complicated) chemical sequences. The dotted arrows represent membrane processes that are relevant to the (c^1_{mk},C^1_{mk}) coupling. The "+" sign on these dotted arrows indicates a process, released when the cell membrane is depolarized, which augments the total cellular c^1_{mk} value. The "−" sign represents the release of transmitter from the cell when the cell membrane is depolarized, and consequently depicts a reduction of cellular C^1_{mk}. The "+" and "−" signs are thus determined only relative to processes in the cellular interior. This diagram has several of the markings of field-antifield duality. For example, reversing the orientation of the dotted arrows relative to the cell membrane reverses their sign. Nonetheless, these arrows communicate with cellular (c,C) processes that, picturesquely, represent an internal "chemical vortex" within the cell which would tend to self-annihilation in the

absence of the field-antifield asymmetry which is introduced by coupling the directionality of the (c,C) process to a particular polarity within the cell membrane. The violation of field-antifield duality for such a cell thus has the useful effect of permitting a nontrivial amount of transmitter to accumulate within the cell.

Field-antifield duality can be better recaptured by looking at cellular systems which include more than one cell, in particular systems which obey neural-glial duality. Here a simple, somewhat hypothetical, situation is given by two cells R^+ (neural) and R^{--} (glial) with the diagram



where we have replaced (c,C) by (c^+,C^+). (c^{--},C^{--}) denotes the processes underlying the production of the transmitter C^{--}, which is a C^+ antagonist. The sign of the dotted arrows is again determined as above. When C^+ and C^{--} are emitted in close proximity, they annihilate one another in this system. Whenever a system is given for which such annihilation occurs, it is probable that the subsystems producing these products are reciprocally related as well. The close arrows

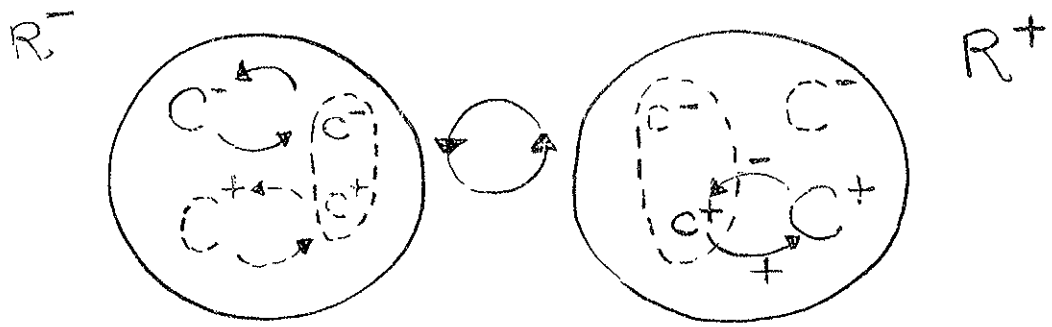


denote the electric currents of the membrane processes which activate this system. We assume that this current hyperpolarizes R^{--} whenever it depolarizes R^+. Moreover, although a depolarization is required to activate the (c^+,C^+) system, a hyperpolarization activates the (c^{--},C^{--}) system. That each of (c^+,C^+) and (c^{--},C^{--}) depends for its activation on a particular membrane polarity and that the two systems together exhaust the set of possible membrane polarities is a fact of

the greatest importance, and one which suggest fundamental reasons for the emergence of field-antifield dualities in a central position in biological systems. In brief, this duality is a multi-stage version of the duality between positive and negative states which exists in the physical fields out of which biological systems have gradually emerged.

We can draw interesting inferences from the dependence of individual cell processes on particular membrane polarities. One cannot, for example, fail to observe that, at least in the case of a single transmission type, the splitting of the cellular system into two pieces, R^+ and R^-, is accomplished in such a way that the transmission of impulses from cell body to node almost always produces the polarization which activates (c^-, C^-) in R^-. Thus, a depolarization in one R^+ cell depolarizes its axon, which carries this depolarization to a node where (c^+, C^+) is facilitated and C^+ is released, thereby depolarizing the next R^+ membrane, whence the process is repeated. By contrast, the R^+ depolarization is associated with an R^- hyperpolarization, a facilitation within R^- of (c^-, C^-) by this hyperpolarization, and a release of C^-, which hyperpolarizes R^+ and annihilates C^+.

Now recall that each of our (hypothetical) R^+ and R^- cells has gradually differentiated from a single (hypothetical) fertilized egg cell, which contained all of the genetic information from which, in interaction with the cellular environment, $R^+ \oplus R^-$ differentiation was induced. The complementarity of R^+ and R^- suggest, therefore, that each of R^+ and R^- possess control mechanisms which are capable of producing either C^+ or C^-. Yet in R^+, C^+ production is ordinarily repressed, as in the diagram



The specific and opposite polarity of the membrane excitation of each of R^+ and R^- provides a suggestion of one way in which differentiated C^+ and C^- production, respectively, is stabilized in these cells. To understand this

stabilization process fully, one must envisage in each cell similarly "polarized" asymmetries in all relevant fields, from the simplest physical fields to the fields organized by the cellular control processes themselves. These hierarchically organized fields are in constant feedback interaction, as the sequence (c^+ augmentation) ⟶ (electrical field depolarization) ⟶ (c^+ augmentation) illustrates. The simplest hypothesis is that specific ions are coupled to the membrane polarity and that these ions serve as coenzymes for intracellular control process variations. It is not, however, necessarily the case that if we reverse the usual electrical polarity of an R^+ mode then c^- will be activated, although it surely would be if a direct coenzymatic coupling with the membrane polarity exists. This is a plausible hypothesis, however, and one which should be studied carefully in the laboratory. The difficulty in firmly drawing the inference is that one cannot, a priori, assess how many intermediate genetic repressions have been required to realize the $R^+ \oplus R^-$ splitting. It is quite possible that a long sequence of linked genetic repressions is involved and that a simple reversal of a primitive electrical field asymmetry will be insufficient to reverse this sequence of genetic repressions. One can only say, a priori, that the various field asymmetries are compatible with one another. If, on the other hand, strongly hyperpolarizing (depolarizing) an R^+ (R^-) node over long time intervals does activate C^- (C^+) production, then we can conclude that the coupling between the electrical and the $c^{\pm}$ fields is rather direct and is intermediated, perhaps, by nothing more than specific membrane-coupled ionic species. Such a conclusion would not deny the possibility that long chains of sequential genetic repressions are necessary to ultimately differentiate R^+ and R^-, yet only small fragments of these chains would directly influence $c^{\pm}$ repressions and these fragments would be responsive to such simple field variations as variations in special ion densities. Such a possibility is compatible with the embedding equations, for in these one finds a rather direct coupling between variations in membrane polarity and in $c^{\pm}$ states. Nonetheless, the embedding equations suppress many local variables, especially variables which are linearly coupled to one another in long chains of rapid chemical reactions, and are merely asymptotic equations which describe stable neural ensembles, not the intricate chemical differentiation of individual cells.

A related prediction is the following. Let the $C_l^+ = ((c_{mk}^l)^+, (C_{mk}^l)^+)$ system

be positively coupled to the $C_2^+=((c_{mk}^2)^+,(c_{mk}^2)^+)$ system, and once again identify R^+ with a nerve cell and R^- will a glial cell that forms a functional boundary for R^+. If the R^+ axon is destroyed, then the C_1^+ nodal system of R^+ and the source of polarized inputs to $R^+ \oplus R^-$ are eliminated by chromatolysis. R^- is hereby released from one source of repression of its C_1^+ system, and C_2^+ loses its customary positive coupling with the C_1^+ system of R^+. The question is: can the de-repressed C_1^+ system of R^- substitute for the eliminated C_1^+ system of R^+. If it can, the glial sheath should, for example, gradually begin producing acetycholine if it originally produced cholinesterase. As a result, instead of finding miniature depolarizing spontaneous potentials at the subsynaptic membrane throughout the period after R^+ destruction, one would expect to find a period of no spontaneous potentials (during the void between R^+ destruction and R^- replacement) followed by a gradual emergence of miniature depolarizing spontaneous potentials in the absence of the customary R^+ source of these potentials. This result would provide further indirect evidence not only for a rather direct coupling between membrane polarization, mediated by ionic coenzymes, and the $(c_{mk}^1)^{\pm}$ control systems, but also for the existence of a significant $((c_{mk}^1)^{\pm},(c_{mk}^2)^{\pm})$ coupling.

126. Polarized Membranes, Coenzymes, and Cellular Control Processes

It is useful to carry the remarks on repressed control mechanisms and coenzymes a step further. In particular, imagine a cell which contains a pair of $(c^1)^+$ and $(c^1)^-$ control mechanisms whose activation and repression depend on transport processes which are directly coupled to the membrane polarity. These processes must depend on the deviation of the nodal potential from an equilibrium value. We assume that this equilibrium value, denoted by Q_{mk}^{eq}, equals the transmission threshold of the axon leading to the node. This assumption has the purely formal advantage that the term $\max\left[\max(s_m(t-t_{mk})-u_{mk},0)-Q_{mk}^{eq},0\right]$ is replaced by $\max(s_m(t-t_{mk})-u_{mk},0)$ in the equations for $(c^1)^{\pm}$. It also has two pleasant theoretical properties: (1) It is compatible with the assumption that the membrane resistance along the axon to and including the node is approximately constant, and (2) the node feels the electrical activity at its cell body source only through axonal transmission. Since the zero of axonal transmission is scaled to the transmission threshold, it is reasonable to expect

that the nodal processes dependent on cellular excitation are similarly scaled. Nonetheless, this assumption--which is not critical to our analysis--is purely hypothetical, and must be checked by experiment in every individual case.

Assuming a strong coupling for specificity, we find

$$d(c^1_{mk})^+/dt = \gamma^+_{mk}((A^1_{mk})^+ - (c^1_{mk})^+)\max(s_m(t-t_{mk})-u_{mk},0)\max(s_k(t-T_{km})-U_{km},0)$$

$$-\gamma^-_{mk}(c^1_{mk})^+(M_m-\max(s_m\ldots))(M_k-\max(s_k\ldots))$$

and

$$d(c^1_{mk})^-/dt = \gamma^+_{mk}((A^1_{mk})^- - (c^1_{mk})^-)\max(u_{mk}-s_m(t-t_{mk}),0)\max(U_{km}-s_k(t-T_{km}),0)$$

$$-\gamma^-_{mk}(c^1_{mk})^-(\max(u_{mk}-s_m(\ldots),0)-m_m)(\max(U_{km}-s_k(\ldots),0)-m_k).$$

Here, each of R^+ and R^- is supposed to carry a $(c^1_{mk})^+$ and a $(c^1_{mk})^-$ process. Denote that of R^+_{--} by $(c^1_{mk})^{++}_{--}$ and by $(c^1_{mk})^{+-}_{--}$, respectively. Employ a similar notational convention for the s_m functions as well. Thus, within each of R^+ and R^-, we find two antagonistic control processes, carried on separate geometrical sites.

As these equations stand, field-antifield duality is not satisfied within either of R^+ or R^- individually. This is easily seen by setting $(A^1_{mk})^{++}_{--} = (A^1_{mk})^{+-}_{-+} \equiv (A^1_{mk})^{+}_{-}$ and $(A^1_{mk})^{+}_{-} = (c^1_{mk})^{++}_{--} + (c^1_{mk})^{+-}_{-+}$, and by substituting these values in the equations. Nor can one, in general, salvage field-antifield duality by considering $R^+ \oplus R^-$. Duality can, however, be achieved under the special assumptions that

$$(a_1)\quad \max((s_m)^{+}_{-}(t-t_{mk})-u_{mk},0) = \max(u_{mk}-(s_m)^{-}_{+}(t-t_{mk}),0)-m_m,$$

$$(a_2)\quad M_m-\max((s_m)^{+}_{-}(t-t_{mk})-u_{mk},0) = \max(u_{mk}-(s_m)^{-}_{+}(t-t_{mk}),0)-m_m,$$

and

$$(a_3)\quad \max(s_k(t-T_{km})-U_{km},0) = \max(U_{km}-s_k(t-T_{km}),0),$$

$$(a_4)\quad M_k-\max(s_k\ldots) = \max(s_k\ldots)-m_k,$$

as one sees by letting A^1_{mk} $(A^1_{mk})^{+}_{-}$ and

$$(a_5)\quad A^1_{mk} = (c^1_{mk})^{++}_{--} + (c^1_{mk})^{--}_{++}.$$

We call (a_5) the crossing over condition, since the dual of a c function in one cell is the complementary c function of the complementary cell.

When does (a_1) hold? It holds whenever the R^+ and R^- membrane polarities are equal and opposite. This is the clue to the entire matter, in fact, for we have already discussed precisely such a splitting of membrane polarities in $R^+ \oplus R^-$; the system is actually constructed to always ensure just such a splitting under physiological inputs. The equations show that one polarity allows certain $(c^1_{mk})^+$ growth-inducing ions to enter the cell and extrudes certain $(c^1_{mk})^-$ growth-inducing ions, while the opposite polarity has the opposite effect. (a_2) has the same significance as (a_1), with the additional physical assumption that the scale of $(c^1_{mk})^{\pm\pm}$ growth and decay is measured relative to the extremal scale values m_m and M_m. Our implicit choice of $M_m = (M_m)^+$ and $m_m = (m_m)^+$ is, of course, not essential. (a_1) and (a_2) together are thus compatible with the existence, for both R^+ and R^-, of a single universal membrane type, which distinguishes R^+ and R^- entirely by membrane transport processes that are coupled to the expected polarities induced by physiological inputs.

(a_3) and (a_4) introduce a new consideration, for we have no reason to believe that the polarity of the postsynaptic membrane induces a process which has opposite "polarity" in presynaptic R^+ and R^-. If an (a_3) coupling is possible, it must induce opposite effects in the R^+ and R^- membranes, or some process coupled to these membranes, whence the universality of the $R^+ \oplus R^-$ membrane structure is lost.* If we wish to preserve membrane universality and field-antifield duality in this situation, we have essentially two alternatives: (1) we must weaken the presynaptic-postsynaptic coupling. The simplest way to do this is to render it a nonspecific catalytic coupling, realized by a premultiplying point strength term in each of the $(c^1_{mk})^{\pm\pm}$ equations, (2) Replace the terms $\max(...)-m_k$ and $\max(U_{km}-s_k(...),0)$ in the $(c^1_{mk})^-$ equation by $(M_k-\max(s_k...))$ and $\max(s_k(...)-U_{km},0)$, respectively. Since (2) provides the simplest strong coupling that is compatible with membrane universality and field-antifield duality, we make this change here. (2) is a strong coupling term which does not distinguish $(c^1_{mk})^+$ from $(c^1_{mk})^-$, and is therefore nonspecific in this restricted sense.

This example shows how a proper application of such general phenomenological concepts as field-antifield duality and membrane universality can exert a forceful control over the choice of possible couplings, which often

*Also see REMARK following this discussion.

becomes unique when a small amount of data on the general nature of a specific coupling is available. Were it true, for example, that membrane universality, field-antifield duality, and the original coupling equations held, we would be forced to embed $R^+ \oplus R^-$ into a larger cellular system to see duality, and thus the complete set of theoretical variables, clearly. In this way, general principles and a small amount of local data provide the theorist with useful hints on how to complete a cellular system.

Since we have supposed in this example that the two antagonistic $(c^1_{mk})^+$ and $(c^1_{mk})^-$ processes exist in each of R^+ and R^- ready to be activated by a suitable membrane polarization, it becomes immediately possible that internal $((C^1_{mk})^+, (C^1_{mk})^-)$ annihilations can take place. This is accounted for in the adjusted equations

$$d(C^1_{mk})^{++}_{--}/dt = u(c^1_{mk})^{++}_{--} - (u\lambda + rvmax(...)p_{mk})((C^1_{mk})^{++}_{--} - (C^1_{mk})^{+-}_{-+}),$$

where $(C^1_{mk})^{++}_{--} - (C^1_{mk})^{+-}_{-+}$ denotes the effective transmitter supply in R^{+}_{--}. In this situation, one therefore finds transmitter annihilations both interior and exterior to individual cells. The exterior annihilations may be viewed as the residual annihilations of the net transmitter content remaining from interior annihilations. The opposite membrane polarizations ensure that all annihilations are not interior ones. On the other hand, if the membranes are kept at equilibrium potential for all time intervals, all antagonistic c quantities decay to a perfectly balanced state.

Although the data remains insufficient to recognize these various systems in vivo, each such system provides an idealization of possible relationships between important theoretical properties, in whose pursuit new experiments can profitably be organized. Moreover, more realistic--and complicated--theoretical systems can be constructed on the model of these idealizations once the underlying method of attack is understood. It is of special importance that one realize that, once a set of phenomenological concepts is chosen and a minimal amount of data available on the nature of the coupling in any given situation, it becomes immediately possible to make inferences about the nature of the membrane transport process which services the cellular control processes being studied. This is a fact of the greatest importance, for it provides a method for <u>inferentially</u> studying the ensemble interactions of stable control macromolecules with their fluctuating environmental field surround, which is a problem of such central importance for all of biology, and one that is made

especially difficult to attack directly with experiments by the fact that the coupled quantities lie in widely separated regions of the cell and enjoy different relaxation times.

We end these considerations by calling attention to the system for which (1) each of R^+ and R^- is its own dual, (2) each of R^+ and R^- contains a $(c^1_{mk})^+$ and a $(c^1_{mk})^-$ process which is directly responsive to opposite membrane polarizations, (3) the process is strongly coupled, (4) membrane universality holds. By (1) and (2), $A^1_{mk} \equiv (A^1_{mk})^+ = (A^1_{mk})^-$, and $A^1_{mk} = (c^1_{mk})^+ + (c^1_{mk})^-$. The growth term of the process is

$$d(c^1_{mk})^+/dt = \gamma^+_{mk}(A^1_{mk} - (c^1_{mk})^+)\max(s_m(t-t_{mk})-u_{mk},0)\max(s_k(t-T_{km})-U_{km},0) \sim \ldots$$

Thus, the entire process is

$$d(c^1_{mk})^+/dt = \gamma^+_{mk}(A^1_{mk} - (c^1_{mk})^+)\max(s_m \ldots)\max(s_k \ldots)$$

$$-\gamma^-_{mk}(c^1_{mk})^+\max(u_{mk}-s_m(t-t_{mk}),0)\max(s_k(t-T_{km})-U_{km},0),$$

where the last $\max(s_k \ldots)$ follows by (4). Thus

$$d(c^1_{mk})^+/dt = \max(s_k \ldots)\left[\gamma^+_{mk}(A^1_{mk} - (c^1_{mk})^+)\max(s_m \ldots) - \gamma^-_{mk}(\ldots)\ldots\right],$$

whence the requirement of strong coupling, in addition to (1), (2), and (4), implies that the $(c^1_{mk})^{\pm}$ functions are only <u>weakly</u> coupled.

Also notice that if $s_m(t-t_{mk}) = u_{mk}$ or $s_k(t-T_{km}) = U_{km}$, then $d(c^1_{mk})^{\pm}/dt = 0$. Thus, if either the pre- or post-synaptic cells are maintained at their $(c^1_{mk})^{\pm}$ equilibrium potentials, the $(c^1_{mk})^{\pm}$ remain perfectly constant. There are no decay effects due to disuse in this system. Strictly speaking, certain spontaneous decay terms are possible. These are essentially of the form

$$g((c^1_{mk})^+(c^1_{mk})^-)\cdot\left[f((c^1_{mk})^- - f((c^1_{mk})^+)\right],$$

where f and g are nonnegative, monotone increasing functions with $f(0) = g(0) = 0$, which are customarily chosen as either identically constant or as the identity map.

Temporarily ignore spontaneous decay. A positive membrane polarity must then allow particular ions to pass through the membrane to $(c^1_{mk})^+$ sites, where they coenzymatically activate a chemical system---wholly contained in all other respects in each of R^+ and R^- ---that directly augments $(c^1_{mk})^+$ growth. The coupling which these coenzymes interpolate between the membrane polarity and the

"template" on which the $(c^1_{mk})^+$ process is activated is chaotic (in ensemble), but the template-$(c^1_{mk})^+$ interaction is itself linear. These remarks are all directly read off from the individual coupling terms and the fact that c values are stable at equilibrium potential. Indeed, when $s_m(...)=u_{mk}$, the quantities injected into the cell by a specific membrane polarization are no longer intracellularly available in high concentrations, whereas the $(c^1_{mk})^{\pm}$ levels remain completely stable.

One also sees that reversing the membrane polarity reverses the injection of antagonistic coenzymes. An active competition for A^1_{mk} sites to produce $(c^1_{mk})^+$ or $(c^1_{mk})^-$ takes place, as the equation $A^1_{mk}-(c^1_{mk})^+ + (c^1_{mk})^-$ shows. The spontaneous decay f term (g=constant), if it occurs at all, represents a process, superimposed on the preceding coenzymatic competition, whereby the active $(c^1_{mk})^+$ and $(c^1_{mk})^-$ spontaneously exchange states. A nonconstant premultiplying g term means that this exchange is induced by a chaotic interaction between the $(c^1_{mk})^+$ and $(c^1_{mk})^-$ states. This example thus provides a simple case of how one can easily draw interesting, and even unexpected, conclusions about the internal interactions of systems which obey such naturally posed conditions as (1)- (4).

REMARK: It might be thought that even if the R^+ and R^- membrane is universal, if activating the postsynaptic membrane releases both $(c^1_{mk})^+$ and $(c^1_{mk})^-$ growth-augmenting ions, yet the $R^{\pm}$ membrane polarity filters out just one type of these ions completely, then a bona fide effective strong coupling is still possible. Such a strong coupling is said to be induced by the presynaptic membrane. For this to occur, the postsynaptic membrane must release pairs of ions at each postsynaptic membrane polarity, with one member of each pair a $(c^1_{mk})^+$ activator and the other a $(c^1_{mk})^-$ activator. The polarity of the presynaptic membrane, in turn, must determine which of these ions penetrates into the presynaptic cell. Such a presynaptic induction process could not depend wholly on the flow of ions along electrochemical gradients. The difficulty with this idea is that the postsynaptic pairing of ions must be matched by a presynaptic pairing to preserve membrane universality, which contradicts the possibility of presynaptic discrimination of singlets of the pair.

A general fact emerging from this speculative analysis is that the existence of field-antifield duality on the cellular ensemble level has led us to seek a comparable duality on the level of cellular control processes, which are

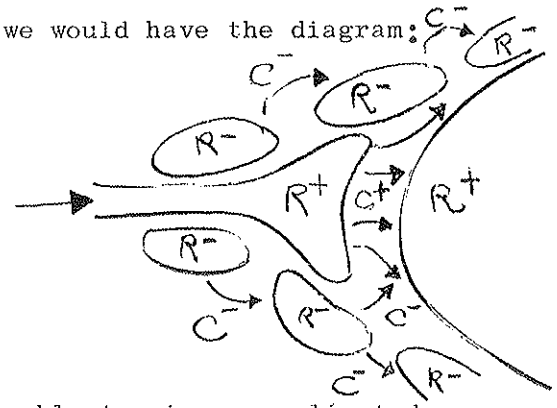
governed by macromolecules such as DNA and the various RNA's, whose frequent appearance as linear coils of paired nucleic acids seems ideally suited to duality. Both forms of the duality principle seem to take their origin from a similar situation in the physical fields out of which biological systems have evolved.

It should be explicitly realized that we have erected field-antifield duality to the status of a postulate. One cannot take it to be a proven fact, for it is supposed to apply to systems which one has never had the opportunity to study, and even if the postulate seems to be violated in a particular system, this ostensible violation might well be a reflection of the fact that the theorist is studying an incomplete set of dynamical variables, as several of our examples have tended to show. Whatever real violations in field-antifield duality do appear should ultimately be traceable to a comparable asymmetry in the physical fields on which the biological system in question is superimposed. We will give examples of such violations in a later work.

Field-antifield duality would remain a useful yardstick for studying biological systems even if real asymmetries were detected. One may view its usefulness, perhaps, as one views the usefulness of straight lines and planes in the study of general curves and surfaces, or of diffusion processes in the study of general membrane processes. It provides a theoretically meaningful standard against which to view a given system as a deviation, or envelope, thereof. Even more significantly, it supplies a criterion for a complete set of theoretical variables for any given system. Thus, if will appear in our later examples of violations of duality that these violations occur only if one misunderstands the function of the physiological system as it is immersed in a particular asymmetrical physical field. The asymmetry in the physical field simply restricts the physiological symmetries to proper subsets of the total physiological field subserving a particular modality.

It remains uncertain, and doubtful, whether a real nerve cell-glial version of $R^{+} \oplus R^{-}$ obeys this duality in all respects. Several of the reasons for these doubts are: (1) R^{-} (glial, Schwann cells) are often far smaller than the R^{+} cells that they couple with. This difficulty is removed, however, if the local dynamical variables of these cells obey mass action laws in each R^{-} and if a superposition principle joins all R^{-} to their underlying R^{+}. These properties are compatible with the embedding equations. (2) After modal depolarization, a

net C^+ concentration is necessary at R^+ subsynaptic membranes if their depolarization is to occur. To satisfy duality, it is therefore necessary to suppose that some type of an R^- subsynaptic membrane exists for which a C^- hyperpolarizing concentration occurs after "nodal" C^- hyperpolarization. Were this true, we would have the diagram:



(3) One must be able to view unmyelinated axons and the free Schwann cells which eventually surround them as systems on an equal theoretical footing in all respects, including the distribution of potential, without considering the Schwann cell merely as a supplementary aid to neural transmission and rigidification.

I am of the opinion that one must look to systems larger than $R^+ \oplus R^-$ to dualize " all" $R^+ \oplus R^-$ variables. For example, to every "on"-"off " field one must be able to associate an "off"-"on " field. Indeed, as increasing numbers of local variables are considered, these variables must be thought of as being embedded in increasingly large regions of brain and supplementary tissues to preserve duality, until, in the limit, the entire nervous system, with all supplementary systems, must be compared with its own field-antifield dual. Nor should it be overlooked that, for species indulging in sexual reproduction, pairs of individuals must be studied instead of singletons to reveal many important symmetries. Carrying forward this extension process, one sees that, in any practical problem, some field-antifield asymmetries must be expected, although in principle it is to be hoped that they can eventually be eliminated. Whether the entire Universe is self-dual is a question on which I do not feel qualified to comment, although the trend of the present argument clearly suggests that as increasingly penetrating local questions are asked, it will be found that increasingly large sectors of the Universe must be studied to salvage whatever symmetries do exist, a conclusion which is compatible with the Heisenberg Uncertainty Principle for quantum fields. Indeed, one is left with the strong impression that all dynamical hierarchies are actually evolutionarily induced by

local deviations from field-antifield duality, and that, in particular, our very existence is tied to hierarchies of local field asymmetries which extend from minute dual splittings to the most slowly varying gravitational fields. If they are carried out conscientiously, one comes away from such seemingly never-ending speculations with a better understanding of the statement that neural systems, like all biological systems, are "open systems" whose ultimate symmetries can be deeply understood only by embedding them in the very rich field environment with which they participate.

127. Spike Discreteness: Electrode vs. Cellular Measuring Devices. The Incompleteness of Dynamical Systems

These discussions of various c function fluctuations show that the smoothness of s fluctuations can be meaningfully measured only with respect to the relative sizes of the c unit variation induced by an s unit variation. In particular, notice that an $R_{kv} \rightarrow N_{kv;ij}$ transmission term of the form $\max(s_{kv}(t-t_{kv;ij})-u_{kv;ij},0)$ varies smoothly as a function of s_{kv}, yet it is a familiar fact that axonal transmission is realized by discrete spike potentials running wavelike to their nodal destinations. It cannot be too strongly emphasized that the spikelike, discrete quality of these potential waves appears only when one uses an electrode measuring device whose time scale is strongly compressed and which can return to its equilibrium value quickly relative to the time scale of ensemble neural events. On the other hand, when one considers a c function as an intrinsic measuring device of ionic fluctuations linearly coupled to local membrane polarity variations, we have no a priori reason to suppose that a c function has the same time constants as an electrode. Indeed, were this the case, the line residual "memory" of our system would be as inconsequential as that of the electrode itself, which is manifestly not so. Since the ensemble behavior giving rise to c functions is slowly varying compared to the variations of s functions, one can therefore, in principle, easily envisage an elementary spike as such a small dynamical unit that the c functions vary continuously with respect to the strings of spikes that underlie neural frequency modulation. Since the product of s and (c- and s-coupled) C functions, in turn, is the effective source of postsynaptic potential changes, the roughness of the s grain is the determining factor in controlling the size of unit s perturbations. One can thus interpret the embedding equations as having

solutions whose smoothness is wholly determined by the s functions; the embedding equations are equations of distribution type whose test functions are grained as roughly as expected s function fluctuations. When one considers entire cells as one's fundamental dynamical units, these s functions are so smooth within a single cell relative to the s values of different cells--all s values governed by the same graining-- that one can, in first approximation, think of the embedding equations as having smooth solutions in the classical sense.

These conclusions had, of course, already been at least partially reached in our preliminary study of control subforms. The new feature is that it is only because the relative grains of s and c fluctuations have a particular ordering, which fits the ordering of the events necessary to complete an entire interneural transmission, that we can write down differential equations for these functions at all. In particular, if the c functions were rapidly varying relative to the s functions, no such equations could be written without introducing a whole new level of dynamical graining, and more theoretical variables. If one assumes, for example, that the p functions are even more slowly varying than the c functions, the p functions must be thought of as being primarily coupled to the c^1 and c^2 functions of the cells whose axons they are carried by, and not to the s functions of these cells. A p function which varies more rapidly than the c functions but less rapidly than the s function of a cell must, on the other hand, be primarily coupled to the s function and not to the c functions. A study of p coupling details will be provided in a later work.

The important point for the present is that the possibility of restricting attention to the s, p, c,and C functions alone in itself partially determines the possible laws which these functions can obey, even though the graining factors determining these laws are themselves invisible in the formal rules of the differential calculus. The assessment that a set of variables is a complete set of variables for a given level of theoretical description is itself not representable within the theoretical equations, while the assumption of completeness, toghether with the equations, provides information about the system that is not contained within the equations. It might be supposed, by contrast, that one can represent the process whereby the various combinations of completeness properties, dynamical equations, perceptual information, and the like are combined to draw theoretical conclusions within the theorist's brain

as a dynamical system in its own right. The various completeness properties, etc., would themselves form subsystems or inputs to this system. It is in this total system, one might think, that the incompleteness of the original dynamical equations is eliminated. This is not so. For just so long as one tries to express the process as a dynamical system, one must introduce variables, and it is the very process of choosing variables which creates the incompleteness of the total system. One can, however, always try at a future time to include these variables in a still larger theoretical frame in which they are coupled to still newer variables, which are primitives in the extended system. Such a method of extension is a continuation of the extension method which began with the simple dual point p_W. This extension procedure now emerges as a method whereby our information about nonlinear systems can be continually increased in spite of the incompleteness of our capacity to represent them symbolically. Such principles as field-antifield duality serve as guides in this extension process whereby we can guess whether, at any time, we have found a set of theoretical variables which is (approximately) complete--up to a given level of dynamical graining.

On a less general level, observe that the transmission term $\max(s_{kv}(t-t_{kv;ij})-u_{kv;ij},0)$ is not, strictly, the only term that is compatible with the (asymptotic) equivalence of geometrical and dynamical indistinguishability under the law of superposition of inputs. More generally, a term such as $T(s_{kv}(t-t_{kv;ij}))$, where

$$T(s) = \mathcal{L}(s)\mathcal{H}(\max(s-u_{kv;ij},0)),$$

is also possible. $\mathcal{L}$ is a linear function of s and $\mathcal{H}$ is the Heaviside function

$$\mathcal{H}(x) = \begin{cases} 1 & x > 0 \\ 0 & x \leq 0 \end{cases}.$$

$\mathcal{L}(s)$ customarily takes the form $\mathcal{L}(s) = s-\lambda_{kv;ij}$, $\lambda_{kv;ij} \leq u_{kv;ij}$. If $\lambda_{kv;ij} = u_{kv;ij}$, this law reduces to the original transmission law. If $\lambda_{kv;ij} < u_{kv;ij}$, a discontinuity in axonal transmission occurs when the transmission threshold is reached, and this discontinuity must be thought of as existing relative to the unit rate of growth of the s and c functions. We will always consider the special case $\lambda_{kv;ij} = u_{kv;ij}$ for simplicity. The case $\lambda_{kv;ij} < u_{kv;ij}$, which is logically possible, introduces obvious modifications.

128. Embedding Surfaces With Fixed Sinks

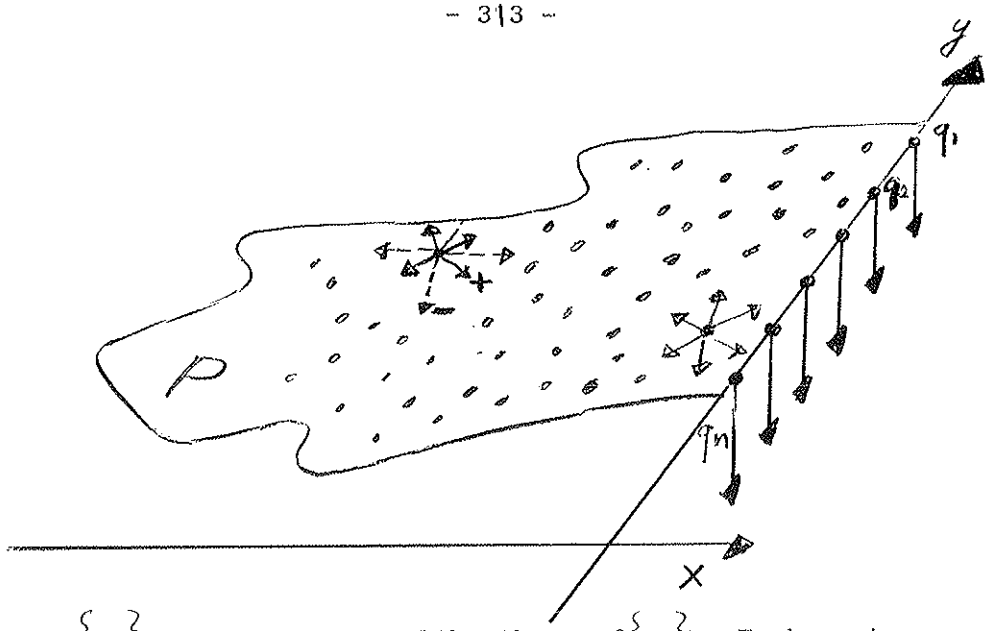
We now leave the question of the extension of volume conductors to local, partially independent membrane systems and return to a discussion of aspects of the global geometry of increasingly realistic fields. This discussion will proceed by illustrating in stages the various inadequacies of certain simple natural guesses at field structure. These inadequacies will suggest field extensions. Once we have considered several of these extensions, a general method of extension will be intuitively clear.

A natural guess at field structure is the following one. Let us be given a surface P in $\mathbb{R}^3$. Suppose that P is a planar region in $\mathbb{R}^2$ for simplicity. Let the points $\{p_i\}$ be contained in the interior of P as Euclidean points, and let each p_{ij} be radially symmetric. That is, $p_{ij}^{\pm} = p^{\pm}(|p_i - p_j|)$, where $|p_i - p_j|$ is the Euclidean distance between p_i and p_j. Suppose further that there exist constants x^+ and x^- such that

1) $p^{\pm}(x) = 0$ whenever $x \geq x^+$,
2) $\max(x^+, x^-)$ is small compared to the diameter of the set $\{p_i\}$,
3) each of $p^+(x)$ and $p^-(x)$ have at most one maximum as a function of x. This is not a critical assumption. Also suppose, for specificity, that p^+ and p^- form proximal "on" and distal "off" regions, respectively,
4) $p^{\pm}$ are compatible with the convergence of field functions to equilibrium in the free case,
5) the transmission velocity is chosen to be the same in all lines, for simplicity.

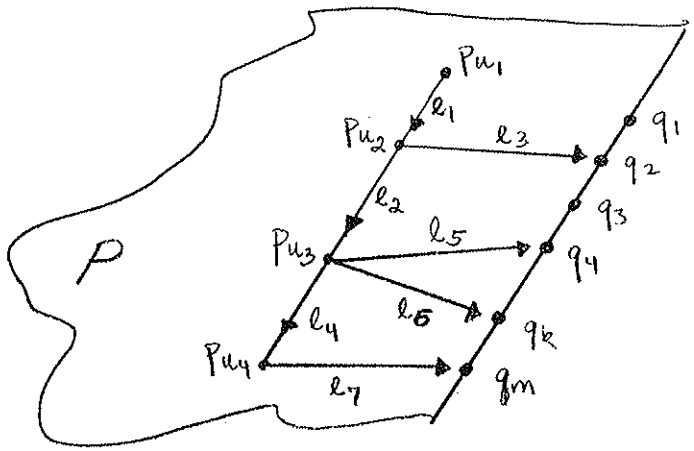
Thus, we are given a plane sprinkled with a large number of points that exhibit radially symmetrical interactions of mixed type with nearby points.

Now let P be embedded in the complex plane $\mathbb{C}^2$ such that $P \subset \{z: \mathrm{Im}(z) \leq 0\}$ and let P have a boundary on $\mathrm{Im}(z) = 0$. Let the points q_i be embedded in $\mathrm{Im}(z) = 0$ with a regular spacing.



Endow $\{q_i\}$ with mixed lines like those of $\{p_i\}$. Each q_i is a control form for some part of a behaviorally visible motion. One can suppose that q_i projects, perhaps in several stages, to a certain collection of muscles with a fixed structural density distribution, and that no two q_i project to the same muscles with precisely the same distribution. Relative to the dynamics of P, $\{q_i\}$ may be intuitively thought of as a set of strength sinks leading from P. Inputs to $\{p_i\}$ are the strength sources of P.

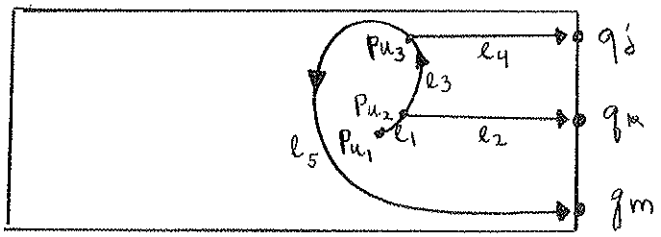
Our problem is to determine what combinations of muscular patterns can be evoked solely by transmission from embedded line residues in $\mathcal{T}(p)$ to $\mathcal{T}(q)$. The simplest nontrivial muscle patterns are muscular sequences. Since the transmission velocity is everywhere the same in P, we can schematically represent flows in P which generate such sequences by directed curves. For example,



says that exciting p_{u_1} induces a highly contracted strength flow along l_1. This flow splits a p_{u_1} into two channels. p_{u_2} is therefore called a branch point of the flow. l_3 excitation by this branch point eventually excites q_2. l_2 excitation reaches the branch point p_{u_3} which simultaneously excites

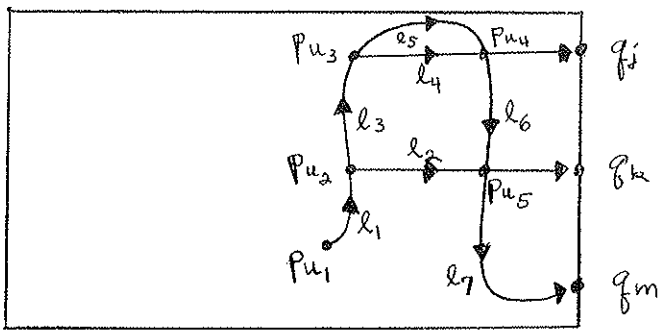
l_4, l_5, and l_6. l_5 excites q_4, l_6 excites q_k, and l_4 excites l_7 by way of p_{u_4}. l_7, in turn, excites q_m. Each l_i can be composed of several successive points arranged in an excitation chain. Supposing that l_5 and l_6 are of equal length, the net outcome of this situation is that exciting p_{u_1} excites $\{q_i\}$ in the order q_2, $\{q_4, q_k\}$, q_m, where $k<m$, with latencies that depend on the lengths of the l_i.

What happens if we try to embed a line distribution capable of generating a sequence q_k, q_j, q_m, where $j<k<m$? Here we need diagrams like



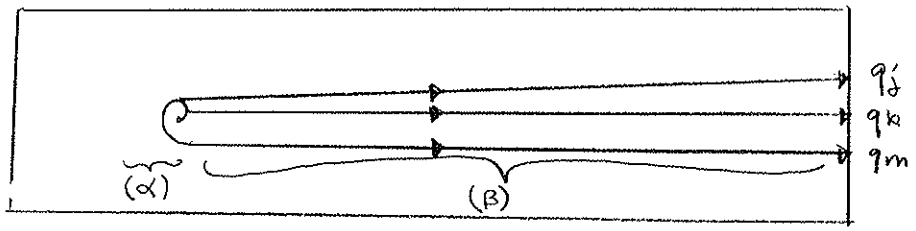
(I)

or



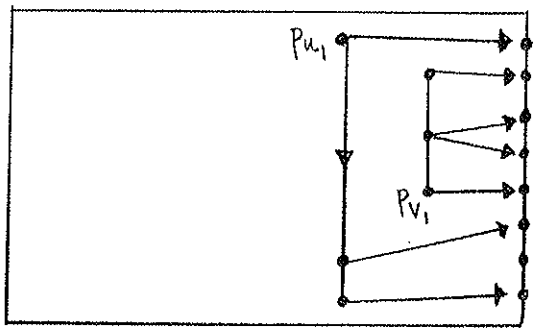
(II)

These diagrams are self-explanatory. The l_5 loop in (I) is necessary to avoid the multiple crossings that occur at p_{u_4} and p_{u_5} in (II). (I) succeeds in generating the sequence q_k, q_j, q_m, but because of multiple crossings, (II) gives the sequence $q_k, q_m, q_j, q_j, q_k, q_k, q_m, q_m$. (I) is, in fact, essentially the only way to avoid the difficulties of (II), up to homotopies of $\bigcup_i l_i$ which do not themselves introduce multiple crossings. In avoiding (II), however, we are forced in (I) to use an l_5 line which is necessarily longer than the shortest possible $l_1 \cup l_2$, or $l_3 \cup l_4$. Thus the time intervals between successive p_{u_1}, q_k, q_j, and q_m excitation onsets can be made equal only by lengthening the l_i, $i < 5$, with small wiggles. This difficulty can be partially compensated by using a diagram like



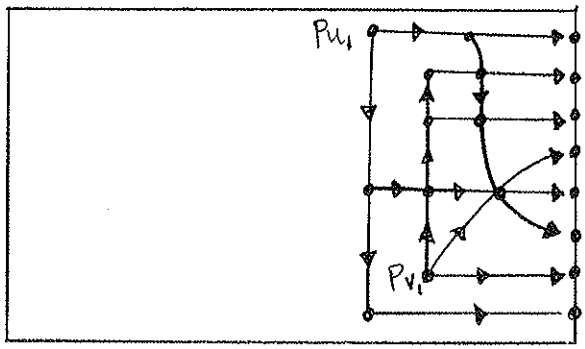
in which the relative length of the (α) region of the l_5 loop is much less than the length of the (β) strips running directly to q_j, q_k, q_m. The fairly complicated (α) region cannot, however, be made smaller than the natural grain of P imposed by the density of the p_i and the p^{+}_{-} distributions. The (β) strips must therefore involve many successive transmissions, which decay at an approximately multiplicative rate with each new transmission. Such long strips thus transmit only small strength quantities to $7(q)$. Moreover, it is very hard to embed such long strips in a plane, and such strips represent a most inefficient waste of the area of P. From such considerations, it becomes apparent that simple changes in the temporal ordering of the muscle sequence to be controlled impose strong restrictions on the possible line embeddings. None of these embeddings is particularly easy to embed and none control the muscle sequences efficiently.

The most profound difficulties arise when we try to imagine embeddings that control even just two different sequences of $\{q_i\}$ points, which one obviously must be able to do in any conceivable organism. Here one faces alternatives like



(I)

and

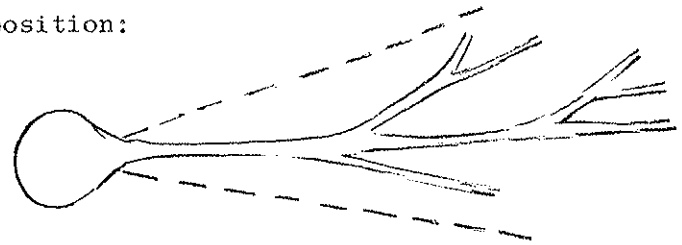


(II)

(II) induces catastrophic behavioral confusions, while (I) requires that a certain muscle group be used only at a single position in a single muscle sequence! A surface is therefore a hopelessly inadequate field for controlling reasonable behavior patterns.

129. Indistinguishable Packets and Input-Output Relations

We can begin to see how to improve on surfaces by asking: how can radially symmetric p^{+}_{-} functions be realized even in a surface? Radially symmetric p^{+}_{-} are desirable because the introduction of spatial isotropy into our spaces enlarges the number of possible embeddings, even though this enlargement is not sufficient when it is restricted to a surface. Yet we are most familiar with cells that emit axons which grow in strongly polarized directions relative to the cell position:



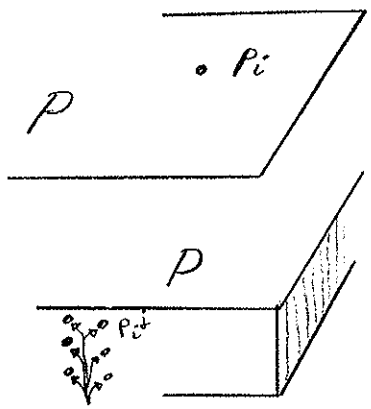
The most obvious way to reconcile these two facts is to suppose that each p_i represents a packet of cells $\{p_i{}^j\}$. Each $p_i{}^j$ can possess a polarized axonal branching, but the distribution of orientations in a horizontal plane is uniform within each packet. Such a use of packets is analogous to the decomposition of field points into packets to produce a spatially self-similar field with homogeneous columns. Replacing each p_i by a packet means that the individual elements of each packet must be dynamically indistinguishable relative to the individual elements of each of the other packets. To ensure dynamical indistinguishability of packets, the elements of each p_i must, for example, be so arranged relative to input mechanisms to p_i that no $p_i{}^j$ is in a privileged

position relative to all other $\{ p_i^k : k \neq j \}$. In particular, the phases of input onsets to the p_i^j must either be random or must introduce only small time lags relative to the transmission times from each p_i^j to $\oplus_{k \neq i} p_k \oplus \oplus_m q_m$. One way to achieve this condition, given homogeneously distributed $\oplus_{i,j} p_i^j$, is to construct input mechanisms whose transmission velocity far exceeds the largest transmission velocity between the packets. This condition on transmission time lags is a condition on the output from each p_i to the other packets.

Indeed, such conditions on the input-output relations of subsets of points form the content of the notion of dynamical indistinguishability. Given any point set $Q \subset P^*$, it is very useful to find that partition of $Q, Q = \oplus_i Q_j$, for which the individual Q_i are maximal sets such that Q_i is dynamically indistinguishable mod $\mathcal{T}(\oplus_{j \neq i} Q_j)$. The meaning of such maximal decompositions is that the individual elements of the decomposition perform as ensemble units with respect to the input-output mechanisms of each field. The input-output mechanisms, which are after all constructed from cells, are themselves clustered in locally indistinguishable sets to form ensemble units. Such decompositions serve vital functions. We have, for example, just observed that cells cannot always be naturally made which send rich axonal distributions in all directions, but that it is sometimes desirable that they behave as if they could. Solution: cluster the cells in packets $p_i = \{ p_i^j \}$ which are dynamically indistinguishable mod $\mathcal{T}(\oplus_{j \neq i} p_j) \oplus \mathcal{T}(\oplus_k q_k)$ such that the cellular orientations within each p_i in the horizontal plane are random. This principle, whereby ensembles of dynamical units can be made to exhibit properties beyond the capabilities of individual units, is quite universal, and should always be kept in mind in interpreting neuroanatomical arrangements.

Our plane P is therefore not a true plane at all, but is merely a set of packets of dynamically indistinguishable points.

is replaced by

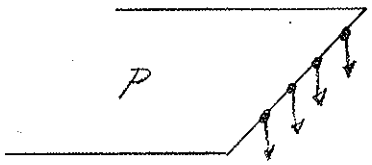


130. Cell Layers

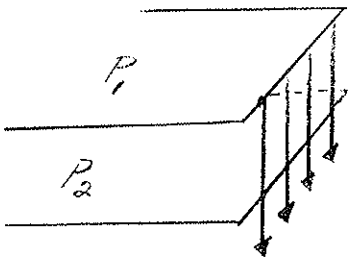
The discussion about controlling muscle sequences means, in this context, that we must loosen the bonds tying together the cells of each p_i packet. If we try to do this without changing the cells themselves, we can only weaken the input bonds tying the cells together. For example, we can replace

by

Thus, a single tightly binding input mechanism is replaced by two such mechanisms. In terms of surfaces, this means that we replace



by



That is, two surfaces, P_1 and P_2, now lead to $\{q_i\}$ instead of just one surface. Since we can arrange these surfaces to be parallel to one another, we call them layers. If we want each q_i to be directly responsive to $P_1 \oplus P_2$, as it was to P alone, we must elongate the q_i cells in the vertical direction. Our previous remarks on the deformation of cells now suggest that after this elongation of q_i is performed, P_1 and P_2 will exert different kinds of influence on q_i. Since I_{ij} is closer to P_2 than to P_1, P_2 cells will exert a more direct influence on q_i firing, while P_1 cells will tend to set the general excitatory tone of q_i — unless special provisions are made in the shape of q_i to accentuate the P_1

contribution. Layers are, indeed, found in great abundance in the cerebral cortex of higher animals, and it is well known that six parallel layers can often be observed. We shall, in fact, show in the following pages that by joining together several layers in appropriate ways, a very sensitive embedding mechanism can be achieved, at least for the handling of local embedding problems.

In passing, notice that our original surface P is immediately decomposed into two layers if we impose the requirement of field-antifield duality. These two layers interact with each other locally to produce lateral inhibitions. If $\mathcal{F}(q)$ is also a mixed field, two layers split q also. These splittings do not enrich the class of possible embeddings in a significant way, although they do provide an interesting example of dynamically indistinguishable extensions. If both layers in such a splitting send lines to the output cells, it is important to notice which layer lies nearer to the ν's, for reasons that we have already considered. Superimposing the $P_1 \oplus P_2$ splitting on this one can given up to four layers, depending on where the input lines end, but one should not confuse the splitting into excitatory and inhibitory components with the layers which are observable in gross anatomical preparations.

Another way to understand the emergence of layers is the following. Let us be given a set $\{p_i\}$ of identical cells and consider the growth of lines from a cell q_1 to $\{p_i\}$. Let $q_1 \oplus \oplus_i p_i$ be embedded in $\mathbb{R}^3$ and let every p_i be surrounded by a spherically symmetrical domain of attraction D_i. That is, we are given a spherically symmetrical function D defined on $\{|x| : x \in \mathbb{R}^3\}$ such that

1) $D(|x|)$ is monotone decreasing,

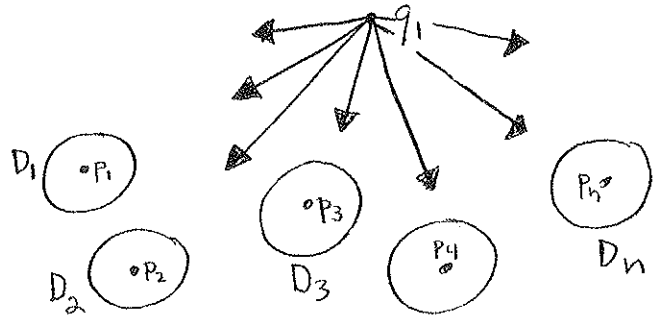
2) there exists $\bar{x} \in (0, \infty)$ such that $D(\bar{x}) = 0$,

3) $D_i = \{x: D(|x - p_i|) > 0\}$.

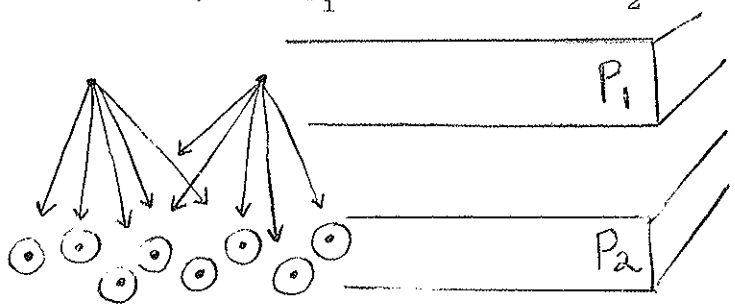
$D_i(x) = D(|x - p_i|)$ is a kind of potential surrounding p_i, possibly a chemical potential. The source of the potential is not critical for our purposes.

Lines growing from q_1 are diverted once they enter some D_i and are redirected at point x with a force that is a monotone increasing function of $\text{grad}(D_i(x))$. q_1 is a standard cell, or indistinguishable collection of cells, which sends out growing lines in all directions with spherical symmetry. We want these lines to reach the p_i with equal probability. $\{p_i\}$ must therefore be distributed with spherical symmetry about q_1. Not all p_i need be at equal distances from q_1 if

1) all p_i lie within a distance that can be reached with equal probability by the q_1 lines in the absence of $\{p_j : j \neq i\}$
2) the D_i of the p_i near to q_1 do not block the free growth of lines to distal p_i. Thus, the set D_i is starlike relative to q_1:



Now let us be given many q_1, say the set $\{q_i\}$, and try to impose the above conditions. The natural symmetry condition to impose on $\{q_i\}$ is that $\{q_i\}$ and $\{p_i\}$ be locally pairwise homogeneous sets. It then follows that as the set $\{q_i\}$ is enlarged, with the various q_i uniformly distributed in space, the spherical symmetry of the original $\{p_i\}$ distribution is flattened until locally parallel layers P_1 and P_2 emerge. q_i is embedded in P_1 with uniform density and p_i is embedded in P_2 with uniform density:



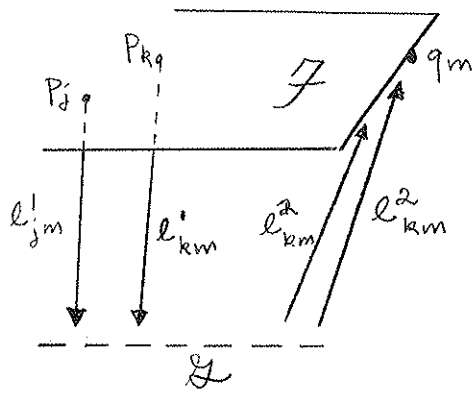
The layers P_1 and P_2 need not be globally parallel, but the curvature of both layers must exhibit parallel changes and must be slowly varying compared to the average diameter of the projection domains of each point.

This example shows that simple laws of growth between two types of cells imply the emergence of layers. These layers reflect a condition on the local input-output relations of the cells. The previous example showed that several layers are needed leading to a fixed set of response control forms. This imposes a condition on the global input-output relations of the cells. Locally parallel layers are thus compatible with the maintenance of large collections of locally pairwise homogeneous point sets whose task is the embedding of the higher control forms of muscle groups.

331. Action at a Distance

A serious deficiency of embeddings in a single surface is that slight changes in the muscle sequence impose strong constraints on the onset times of the muscles. This deficiency is not removed even if we use several layers which all share comparable transmission rates. The ultimate reason for the difficulty is that such layers can only propagate transmissions towards the strength sinks in a local way with respect to the Euclidean topology of the individual layers. Consequently, an input to a point p_i for which $\inf_j |p_i - p_j|$ is large cannot be as effective in generating muscular motions as an equal input to a p_k for which $\inf_j |p_k - p_j| \ll \inf_j |p_i - p_j|$. Only the points which border on $\{q_i\}$ have a strong dynamical grip on $\mathcal{F}(q_i)$ in the surface topology.. To use all p_j in an efficient way, we must therefore extend the field in such a fashion that the local surface topology is strongly violated.

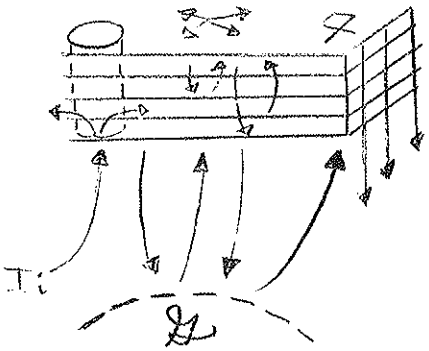
We would ideally like inputs to all p_j to be able eventually to excite a given q_k in the same amount of time and with equal effectiveness, without creating a chaotic field interaction pattern. It is immediate that we need a field extension like



where the dotted line represents an as yet unknown field structure $\mathcal{G}$. An important feature of $\mathcal{G}$ is that the lines $l^1_{jm}, l^1_{km}, l^2_{jm}$, and l^2_{km} are directed for such long distances in an almost vertical direction that $|p_j - p_k|$ is relatively small compared to their lengths. These "long distances" are dynamically measured in terms of relative transmission time lags. Our purpose can be accomplished either with short lines and slow transmission velocities or long lines and rapid transmission velocities. Since each p_j is a dynamically indistinguishable set of points, the latter alternative is strongly preferred, for we have already seen that the existence of rapid input-output pulses makes it easier to construct indistinguishable sets. The widths of the various layers of $\mathcal{F}$ are thus much smaller than the lengths of the

$\mathcal{7}(p) \rightarrow \mathcal{H} \rightarrow \mathcal{7}(q)$ paths. The $\mathcal{7}$-$\mathcal{H}$ interaction is thus a kind of "action at a distance" compared to interactions between the $\mathcal{7}$ layers. Once such a $\mathcal{H}$ is imagined, we immediately see from our experience with fields of type M that it is also desirable to enable the various p_i to interact with one another via $\mathcal{H}$. For after a certain subset $Q_i \subset \{p_i\}$ comes to control $\mathcal{7}(q)$ sequences, we might well want to ensure that new subsets R_j can be embedded to control various combinations or subsets of the Q_i for the purpose of rapidly embedding new sequences on the old. Such a many-many field cannot be constructed free from the old difficulties with surfaces unless we employ $\mathcal{7} \leftrightarrow \mathcal{H}$ interactions.

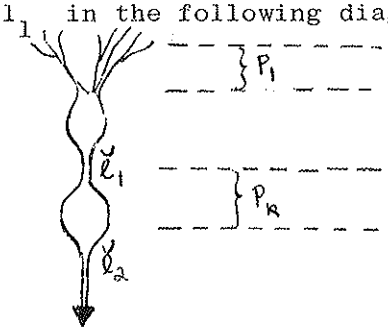
$\mathcal{H}$ thus must sustain topographically ordered reciprocal interactions with cytoarchitectonically well-delineated subregions of $\mathcal{7}(p) \oplus \mathcal{7}(q)$. We have already seen in the discussion of fields of type M that the embedded control forms in such reverberatory fields need not be a single p_{u_1}, as we found with surfaces, but can rather be a sequence of input distributions to well-determined sets of points. The general picture of this situation is



We will now sketch in the details of this picture more completely in several steps. Our sketch will not exhaust the possibilities of field construction. Nor will it include careful lists of all local cell distributions. Such tasks require many more symbols than are useful in a qualitative discussion. Nonetheless, field structures shall emerge that bear strong and unmistakable resemblances to known neuroanatomical arrangements, important functions of these arrangement shall be evident, and the fields can be further extended whenever finer details are desired.

132. Horizontal Fibers

We earlier saw that by elongating the output cells when several parallel layers are juxtaposed, the layers furthest from $\breve{1}$ come to exert a less direct control on output transmissions than the nearby layers. Unless special cell deformations like $\breve{1}_1$ in the following diagram are introduced,



the flow of excitation from P_1 to P_k propagates in a primarily diffusive, or electrotonic, fashion to $\breve{1}_2$, where it interacts with more direct inputs from the lower layers. Thus, the upper layers might well be expected to contribute to the control of excitatory field tone and related subliminal matters. Subliminal tone obviously becomes an effective mechanism only if it is distributed over a broader region of space than the mechanism which exerts direct control over output transmission. We will therefore expect to find rather long horizontal fibers in P_1. If the lines sent out by each P_1 point terminate with uniform density throughout P_1, and the transmission velocity is proportional to line length, the entire embedding surface is effectively contracted to a point with respect to the setting of excitatory tone. Such a global contraction is not useful when we want the simultaneous excitation of several P_1 points to preferentially subliminally excite a localized set of output mechanisms. Thus we expect to find radially symmetric distributions (indistinguishable sets!) of horizontal lines whose density diminishes with increasing length. Radial symmetry is, of course, not a critical assumption. In particular, when a field is compressed in one direction, elliptic line distributions are more appropriate.

What shall the spectrum of transmission velocities be within P_1? This cannot be determined independently of the transmission structure of the other layers, but certain conclusions can be made if we grant reasonable general assumptions. All transmission dimensions must be determined by geometrical parameters of the lines, and these parameters are controlled from the cell body. For without such local control, the global interaction structure could not be regulated to mesh harmoniously with changes in local cellular dynamics. When the lines are, for example, of uniform circular shape throughout their length,

all transmission time lags T are a function of line diameter D and length L. When all lines are of equal diameter but of variable length, we can let the transmission velocity be independent of line length. All transmissions here flow continuously with respect to the local surface topology and we can draw directed flow curves between points. $\mathcal{G}$ will, however, be constructed to violate the surface topology. If we think of the subliminal P_1 horizontal transmissions as preparing proper subsets of P_1 points for transmission to $\mathcal{G}$, it is possible that the transmissions within P_1 also violate the surface topology. By such preparations, the transmission flow to and from $\mathcal{G}$ and the preparation of points to participate in this flow interact harmoniously. A P_1 for which this is true will be functionally more a part of $\mathcal{G}$ than of $\mathcal{T}$.

The simplest way to violate the surface topology is to suppose that all horizontal P_1 transmissions reach their destination with a time lag independent of the length of the transmission line. We already know a simple way to accomplish this. Letting V= transmission velocity, set $V=k_1D$, $D=k_2L$, so that $T=L/V=1/k_1k_2$. If several cell types Ω are available in P_1, k_1k_2 can vary with cell type. Let k_j^u be the k_j parameter for cell type $v_u \in \Omega$. If all cell types are included in each indistinguishable packet of cells, it follows that exciting a single P_1 packet can be made to have the following effects on points p^1 to which p_1 sends lines: (1) a series of excitatory waves occur at p^1. Their relative onset times are chosen from the set

$$\left\{ \left| 1/k_1^{u_1}k_2^{u_1} - 1/k_1^{u_2}k_2^{u_2} \right| : v_{u_1}, v_{u_2} \in \Omega \right\}$$

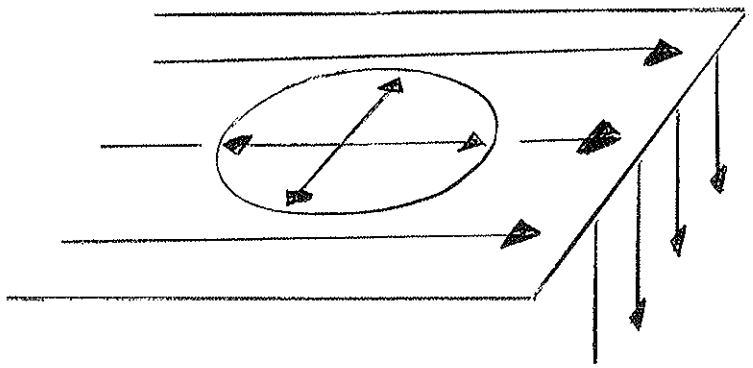
(2) the number and intensity of staggered excitatory waves received from p_1 by some p^1 can be made to vary with $|p_1-p^1|$ if the spatial distribution of p_1 lines of type v_u varies with u. Indeed, these inputs are of the form

$$\Sigma_u s_1(t-1/k_1^uk_2^u)p_u(|p_1-p^1|)c_u(|p_1-p^1|)$$

$$\approx \int s_1(t-T(u))c(u,w)dP(u,w)$$

where $dP(u,w)=p_u(w)$, $w=|p_1-p^1|$, and $T(u)=1/k_1^uk_2^u$. They can obviously be made to exhibit a broad spectrum of functional behavior by varying the field geometry.

Since the q output points are not uniformly distributed in P, the circular symmetry of the law $T=1/k_1k_2$ is upset by the polarity induced by these points:



$\mathcal{G}$ must compensate for this asymmetry if the law $T = 1/k_1 k_2$ is to accomplish its purpose of violating the surface topology. We will shortly see how to do this.

First we must briefly discuss how the P_1 horizontal fibers influence the output fibers. The most central and immediate observation is that when points $p_1, p_2, \ldots, p_n \in \mathcal{P}(P_1)$ are excited at times $t_1 < t_2 < \cdots < t_n$, we find a succession of radiating subliminal waves from p_1, then from $p_2, \ldots$. If the various waves of the individual points overlap in space-time, the output mechanisms which lead from the regions of the greatest overlap will be the most intensively excited, and will often be the easiest cells from which to induce transmissions by inputs from the lower layers. This fact is immediate from the form of the total input to a point $p_k \in \mathcal{P}(P_1)$:

$$\sum_{i=1}^{n} \sum_{u} \int s_i(t-T(u)) c_{ik}(u, w_{ik}) dP(u, w_{ik}),$$

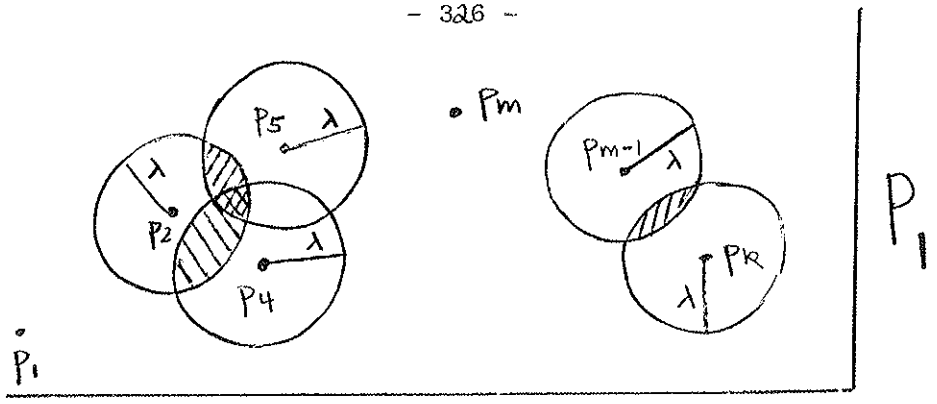
where $w_{ik} = |p_i - p_k|$. For example, let $c_{ik} \equiv 1$,

$$p_u(w_{ik}) = \begin{cases} 1 & \text{if } 0 \leq w_{ik} \leq w \\ 0 & \text{otherwise} \end{cases}$$

and suppose that

$$s_i \approx \begin{cases} k & \text{if } t_i \leq t \leq t_i + T_o \\ 0 & \text{otherwise} \end{cases}$$

where k is a positive constant, and t_i is again the onset of p_i activation. We can here draw diagrams like

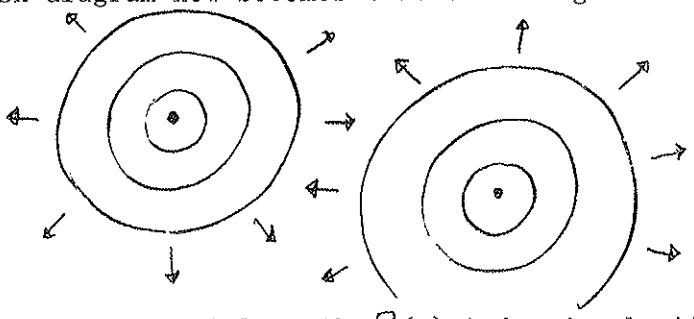


Each disk of radius λ about some p_i is a region to which p_i contributes an excitatory intensity of k at time $t \in [t_i+T(u),\ t_i+T(u)+T_o]$. When m disks overlap, the total excitatory intensity is mk. Since we imagine that these excitations are delivered to those portions of each output cell p_i which are the furthest away from $\overset{\cup}{l}_{ij}$, these individual k excitations can be made to combine almost additively within p_i, and to propagate to $\overset{\cup}{l}_{ij}$ according to an approximately linear partial differential equation. On the other hand, the transmission thresholds are constant, so that, in particular, when all $t_i=t_j$, the points covered by the largest number of disks will be the easiest points from which to evoke transmission.

These disk diagrams are reminiscent of Venn diagrams from logic. The analogy is a significant one. For example, the intersection of all overlapping Venn regions denotes the region which has all of the "properties" of any of the regions, and this fact is rewarded by making these regions of maximal overlap the behaviorally most salient ones. Regions of lesser overlap are also automatically distinguished by the number of overlapping sets. The Venn diagrams provide a pictorial model for certain logical systems, but this model is far less subtle than the time-dependent "logic" of dynamical conditioning in even a single idealized field layer. In P_1, the intersection of regions depends not only on which regions overlap, but also on when they overlap, the distance of overlapping regions from source points, the particular inputs chosen, and so on.

The above P_1 transmission mechanism violates 7's surface topology. A P_1 that interacts with the rest of 7 without violating the surface topology is achieved by simply requiring that the transmission velocity through the horizontal fibers be independent of fiber length, rather than being proportional to the fiber length. Exciting a horizontal fiber locus now generates a radial excitation wave, spreading with uniform velocity through all horizontal lines emerging from this locus. Several velocities can be included, but the normalized

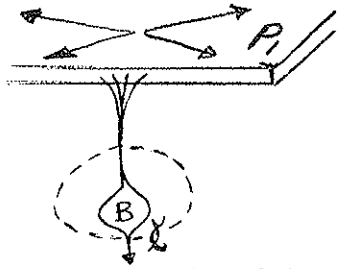
distribution of line lengths must be the same for every velocity. This is just the dual of the former case, where the normalized distribution of velocities must be the same for every line length. All formulas, therefore, carry over from the former case, with obvious dual modifications. The notion of a disk diagram now becomes that of a diagram with radially symmetric rings:



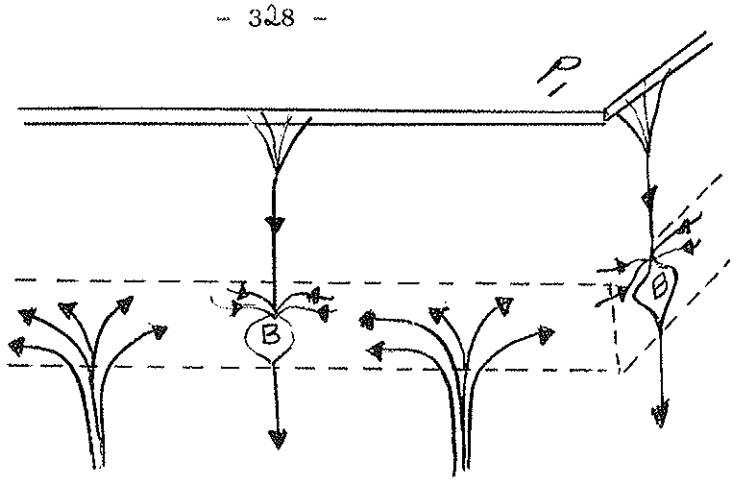
Ring diagrams also violate the $\gamma(q)$-induced polarity of $\gamma(p)$. A ring diagram acts like a disk diagram only when the velocity of transmission to the rings is so much larger than that of any other surface-bound transport process that it is effectively infinite.

133. Input Lines and Output Cells

How do the output cells look? Since P_1 inputs behave as if they are far from 1, these cells must look like



where the dotted region contains as yet undetermined input lines and dendrites. Since the cell body B cannot be too close to a source of excitatory tone such as P_1, the input lines which most directly influence B must also terminate in greatest density far from P_1, and another set of dendrites might well appear, for familar reasons, growing from B in this region.



Input lines need not terminate directly on B. When they do, the control of output from B by direct inputs can be made very strong, reliable, and time-independent. But the possibility of interpolating new input-output line connections is minimized. Such a strong direct control is useful, therefore, either when (i) the input-output circuits are available since shortly after birth, and when (ii) the input lines themselves grow from sources which are stable response control forms, but one transmission-step further removed from the muscles. Such connections should, in particular, prevail more frequently along the central fissure ($\mathcal{F}(q)$) than within Region 6 of man.

134. Interpolated Cells: $\mathcal{M}$ Cells and $\mathcal{I}$ Cells

Since the input lines terminate near the layer of the output cells, we must postulate the existence of cells--called $\mathcal{M}$ cells--which are interpolated between the input fibers and horizontal fibers, and which carry impulses from the inputs to the horizontal fibers. The bodies and dendrites of these cells should lie near the input layer and their axons must rise vertically, terminating in the layer of horizontal fibers, where the axon is distributed in a horizontal direction.

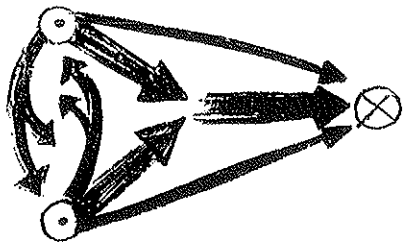
When the growth of new input-output connections is possible, another type of cell must be interpolated between the input and the output lines. Some useful characteristic of such interpolated (or $\mathcal{I}$) cells are the following: (1) We want input-output connections to be possible in all directions. $\mathcal{I}$ cells should therefore be receptive to excitation from all directions. (2) $\mathcal{I}$ cells should have sufficiently small cell bodies that they can be densely distributed within the input and output lines. Large cell bodies could hardly be very sensitive to small fluctuations in input direction, for inputs from a wide angle would always converge additively on their broad cell surfaces. The distribution of the input within this angle would therefore be hard for the cell to distinguish.

(3) Notwithstanding the small size of these cells, it is advantageous that they have a large surface area so that they can receive and respond to many input fibers. The most obvious way to reconcile (2) with this requirement is to endow the $\mathcal{J}$ cells with a profuse dendritic bush. (1) implies that this bush must be almost spherically symmetric, or at least must exhibit profuse branches within a large angle. If the total surface area of the dendrites far exceeds the surface of the cell body, then the input contributions to the $\mathcal{J}$ cells will transform more like additive increments than as volumetrical increments.
(4) The output lines of the $\mathcal{J}$ cells must also be distributed in all directions in which an output line can be found. One way to do this is to let each axon break up into a broadly distributed set of branches. Such cells thus have a double bush. If the $\mathcal{J}$ cell output line sends out highly polarized axonal branches, however, we must introduce dynamically indistinguishable packets of cells, in which the output lines are randomly distributed in all directions towards which the input lines are equally dense. This requirement also provides another reason why $\mathcal{J}$ cells (with only one bush) must be small. We must be able to stack up so many of them vertically that many polarized axonal directions are found in each stack, and this must be done without forcing the upper and lower ends of the stack out of the transmission range to the output cell body. Another way to effectively randomize the axonal directions is to send dendritic branches out from the output cell body in various directions into the $\mathcal{J}$ cell mass. Inputs to such dendrites, however, will behave more additively than volumetrically, of course.

The $\mathcal{J}$ cells thus appear in multitudinous meshes, with their profuse dendritic bushes and (un)polarized axons so arranged that large numbers of directed $\mathcal{J}$ cell chains pass from each set of localized input lines to each nearby set of localized output lines. The successive excitation of $\mathcal{J}$ cells in an $\mathcal{J}$ cell chain can be made to approximate a random process with additive, exponentially decaying increments at every link in the chain, up to variations in the c functions of these cells. When many localized input lines are simultaneously excited, or a few localized lines are excited intensively, populations of immediately adjacent $\mathcal{J}$ cells will begin to transmit, and this transmission pattern will be passed on to their successor cells. We naturally choose transmission thresholds and other field parameters so that the strength functions return to equilibrium values in a free field. The excitation transmitted

over $\mathcal{I}$ cell chains therefore decreases as the length of the chain increases, at least as fast a a power of the number of links in the chain, whence new input-output connections will be easiest to establish between nearby input and output mechanisms.

We can draw pictorial representations of $\mathcal{I}$ chain excitations. For example, let a directed curve represent a chain of $\mathcal{I}$ cells, "$\odot$" an excited input point , and "$\otimes$" an excited output point viewed in a vertical direction. Then we can draw



to represent the flow through $\mathcal{I}$ cell chains from two excited points to a symmetrically placed output point. The thickening in the arrows represents the heightened intensity achieved by summation in the dendrites that receive inputs from both input sources. Implicit in the realization of such thickenings is the natural supposition that every dendritic region of a given $\mathcal{I}$ cell can be reached by equally distant and properly polarized $\mathcal{I}$ cell axons with equal probability. An essential difference between diagrams of $\mathcal{I}$ cell chains and disk diagrams in P_1 is that the former are closely tied to the surface topology by the fact that transmissions are propagated stepwise over the links of the chain. The dynamics of $\mathcal{I}$ cell chains are thus easier to represent by a local physical field theory than the dynamics of P_1 disks. This bond with the surface topology is not complete, however, due to the many-one relation of input sources and transmissions. The almost additive input contributions tend to overcome even this difficulty when the transmission threshold is quite low.

135. Stellate Cells, Pyramid Cells, and Cells of Martinotti

The description of desirable properties for $\mathcal{I}$ cells closely resembles the neuroanatomical description of the stellate cells; we therefore identify the two. The stellates, are indeed, found with greatest density in association cortex

and in increasingly large numbers in the highest mammals. Both of these facts are compatible with the excellent preparation of $\mathcal{S}$ cells for the construction of new input-output chains. Similarly, the shape and function of the output cells suggest that they are abstract realizations of pyramid cells. The $\mathcal{M}$ cells are identified with the cells of Martinotti.

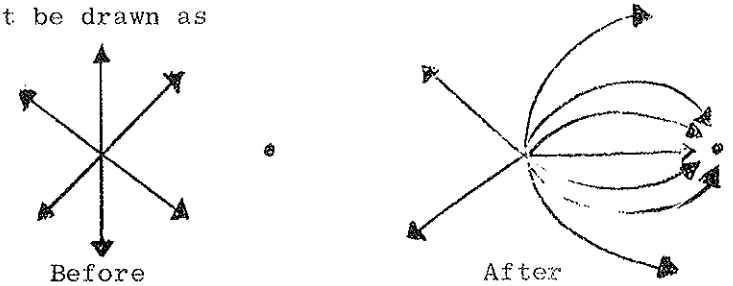
It is not critical that the stellates terminate directly on the pyramid cells, just so long as they terminate on cells which reach the pyramid cells in a very small number of transmission steps. In either case, if we want the body B of a pyramid cell to act as a volume conductor, it is important that many lines terminate over the entire surface of B. When B does behave as a volume conductor, the effectiveness of a given cell on transmission from B is determined by the total number and size of endbulbs which it sends to B. To construct B's which are sensitive to inputs from many directions, many different cells must send lines to a single B, and we can expect to see profuse nests of small cells terminating on B, as indeed are often found in neuroanatomical preparations.

One can proceed in this fashion to add new subtleties to the interactions between the layers. Lateral inhibitory cells can, for example, be attached to the output cells as "on"-"off" regions in familar ways, as can recurrent pyramid collaterals to help bind together the excitatory output of local pyramid cell collections. One can also introduce vertically oriented cells whose transmissions help to bind together columns of cells of similar type into dynamically indistinguishable sets, and horizontal cells several times longer than stellate cells to facilitate the growth of excitatory stellate chains; cf., spider cells. Pyramid cells of varying sizes that are sensitive to excitation in different layers can also be included. Since the excitation sustained by a pyramid cell over much of its range is transmitted with a locally determined velocity, introducing vertically oriented pyramid cells of different sizes does not violate the surface topology in the vertical direction. Several input layers can also be easily contemplated. Further details can be added without qualitative difficulty, and will differ in detail according to the function intended for the surface as a whole.

136. Weighted Lines of Force

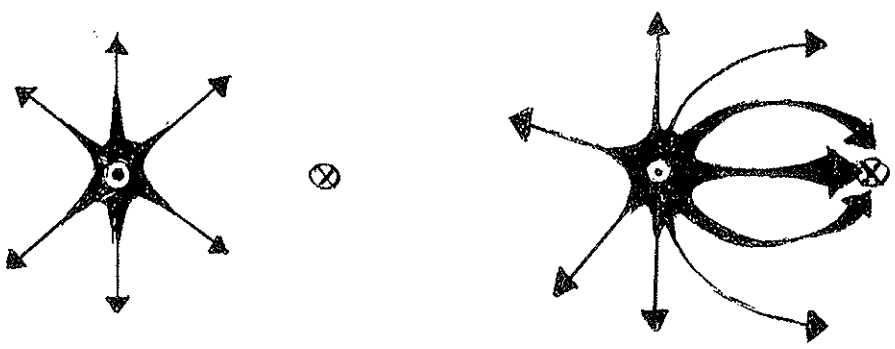
An important feature of this discussion is that new input-output chains cannot form unless the output cells $\mathcal{O}$ are excited simultaneously with several end links of the stellate chains impinging on $\mathcal{O}$ --at least if the strongly coupled embedding equations hold. Thus, some input-output connections must exist from the very beginning of the organism's independent life and must be part of the infant's overt behavioral repetoire. Independent life begun as a tabula rasa ends as a tabula rasa. To form new input-output chains, we must deliver inputs to points p which send out radially spreading excitatory waves. If an extant input-output cycle is activated before these waves decay, the simultaneous activation of $\mathcal{I}$ cells in contact with the output cells will cause the $\mathcal{I}$ cells' c functions to grow. All of the c functions of cells in the chain are not equally facilitated by this mechanism. The c functions of the cells Q which are nearest to the output cell bodies are facilitated first. This local facilitation is really quite satisfactory to provoke new learning, for all that really matters is that a later input to p generates a larger transmission to the appropriate output cells over the facilitated terminal links Q.

We do not get a full picture of the facilitation process by thinking merely in terms of one $\mathcal{I}$ chain. A better picture is obtained by comparing a field of $\mathcal{I}$ chains to a classical field theory, say a stationary magnetic field. In such a magnetic field, the lines of force before and after successive inputs and outputs might be drawn as



Before After

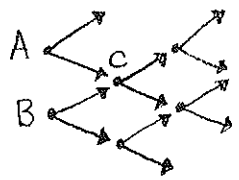
For embedding chains, the comparable picture is



Before After

The distribution of the global objects in the embedding picture, such as the cells, does not change. During short time intervals at least, the change occurs in the microscopic dynamical states of oriented subsets of these objects. The intimate relations of the multitudes of stellates to their neighbor stellates and to every nook and cranny of impinging input and output cells, as well as their locally timed transmission law, are enough to ensure an amazing sensitivity of populations of these cells to even slight variations in the input sequence. New input-output $\mathcal{J}$-derived chains must be clustered near the q_i points in an infantile organism if $\mathcal{G}$ interactions are not available. With increasing experience, the input source of an effective chain can be taken farther from $\mathcal{T}(q)$, since many links in the chain will have formed preferential directions by that time. The randomly distributed collection of $\mathcal{J}$ cells gradually form sets of generalized channels with increasing experience. The existence of a $\mathcal{G}$ field makes the emergence of long $\mathcal{J}$ channels somewhat unnecessary, as we shall soon see. New input-output connections are not necessarily restricted to the $\mathcal{J}$ cells, but can occur whenever the strong coupling conditions of the embedding equations are realized.

Notice that the growth of weighted lines of force in a stellate field is augmented considerably by the extensive convergence and divergence of stellate fibers. For example, in the schematic diagram



simultaneous excitations of A and B excite a localized region of C. This joint excitation is distributed to each of the A$\longrightarrow$C and B$\longrightarrow$C junctions, and therefore hastens the strong coupling at these junctions more than would have been possible if just A or B had been excited. A later excitation to A (or B) alone will reflect this augmentation in the A$\longrightarrow$C (B$\longrightarrow$C) coupling by exciting C more than would have been possible if only A (B) occurred on the first trial. A (B) hereby tends to reproduce the C excitation which had earlier been produced by A and B together. Extrapolating from this diagram, it is intuitively clear that a rich plexus of converging and diverging stellates is well suited to reproduce many joint effects of several overlapping past inputs when only a proper fraction of these inputs occurs on a later trial.

137. Motions Through Layers and Refinements in the Space of Equivalent Potential Distributions

If we want P_1 to behave as a functional part of 7, in particular with respect to $\mathcal{J}$-cell behavior, we must choose the transmission velocities to be independent of line length, in the sense of the second P_1 case above. We thus choose ring diagrams instead of disk diagrams. We also want the velocity of transmission over horizontal fibers to be at least as large as the velocity of propagation through stellate cell chains. In fact, we choose the relative velocities in ensure that an input to p_i will be transferred by the cells of Martinotti over the horizontal fibers to the apical dendrites of the p_j pyramid cells and down to the region of stellate-pyramid contact so quickly that excitation propagated from the p_i input fibers over the stellate chain to the p_j stellate-pyramid contacts arrive when the p_j pyramid cells are active. The prerequisites for strong stellate-pyramid coupling are hereby satisfied.

Letting the pyramid cell $\overset{\smile}{1}$ regions be reached by inhibitory interneurons in short-range "on"-"off"distributions, we see that the cell body of a pyramid cell can be volumetrically inhibited without inhibiting the nonvolumetrically bound dendrites, including regions of the apical dendrites. Thus, new $\mathcal{J}$-cell--dendritic connections can be formed subliminally in the absence of pyramidal output transmission. More will be said about this phenomenon in a comparable situation in 8. The formation of new connections in 7 is thus a geometrically graded process. The superficial layers, like P_1, contribute to initial, relatively coarse, and subliminal line connections. These connections are gradually stabilized in $\mathcal{J}$-cell chain formations, which are actually better described in terms of weighted lines of force. Thus, the establishment of new connections tends to proceed on successive trials from the superficial layers of cortex to the lower layers. This motion through the layers is only relative, since all layers react with a columnar symmetry to new inputs. Nonetheless, the weighted lines of force in the lower layers, which are the bulwark of the truly delicate new connections, do take on greater relative importance later in the connection process. A useful heuristic way to think of this motion through the layers derives from the realization that the $\mathcal{J}$ chains are the most direct and stable input-output connections possible in 7. The motion from superficial to deep layers thus parallels a contraction of the functional distance between fixed input and output mechanisms. It also parallels a refinement, or successive conditioning, within the associational cells of all kinds until they

can discriminate inputs selectively before transmitting to their respective output mechanisms. This conditioning contracts the functional distance between fixed input and output distributions.

The superficial layers of cortex can only discriminate gross differences between the input distributions they receive, while the deeper layers can discriminate even minute differences. Let the set of possible space-time excitatory distributions of a cortical column p_i be decomposed into classes such that two distributions are in a single α class if and only if they cannot be distinguished by the α^{th} cortical layer. Then the motion of excitation from peripheral to deep cortical layers induces successive refinements in the classes of p_i. Since only one space-time distribution ω is actually realized, this successive refinement of the α classes may also be viewed as a contraction of the set of possible space-time distributions to which ω belongs. As the excitation passes through the layers, therefore, the column p_i becomes "more certain" of which space-time distribution has actually been received. The ultimate mark of a column's "decision" as to which space-time distribution it has received is its output transmission pattern. Because the peripheral layers only coarsely discriminate space-time distributions, they can only subliminally excite the output cells. But because the $\mathcal{J}$-cell weighted lines of force can very finely discriminate space-time distributions, they exert a direct and powerful influence on the output cell body's excitatory potential. It should not be overlooked that by virtue of such a motion through the cortical layers, many different input distributions to the cortical column leave behind distinctive line residues which control subtle output variations. A simple excitation flow between columns has none of this flexibility.

A single sweep through the cortical layers therefore accomplishes a remarkable contraction in the collection of space-time distributions as it simultaneously accumulates near the $\overset{\smile}{1}$ terminals derived excitations which determine the form of pyramidal transmission to the measure that they can determine which input distribution actually has impinged on the cell! Such automatic, successive contractions joined by a parallel weighting of importance in determining the output are pervasive in neural interactions. One can picturesquely think of them as the neural analog of a perfectly rational man (column!) who acts forcefully only when his information is complete. When only superficial layers are excited, his information about the input is highly limited, so his activity is meager. But when the beautifully sculpted $\mathcal{J}$-chains determine the input with

high probability, he acts with vigor, and produces the act appropriate to the information.

The analogy with successive refinements in one's information suggests a consideration of the neural correlate of the information functional: lateral inhibition. Indeed, if all p_i are uniformly excited, we expect none of them to transmit, for otherwise the organism will be flooded with insignificant background excitation. If the p_i form a homogeneous field, for example, this means that we must surround each p_i by a radially symmetric lateral inhibitory mechanism Δ_i, distributed to a ring of cells outside p_i's excitatory surround, such that $\oplus_i \Delta_i$ is a complete set of antagonists for $\mathcal{F}(\oplus_i p_1)$. Each Δ_i can be realized, as we noticed above, by a set of inhibitory interneurones joining input axons to pyramid cell bodies, whose axons terminate on the pyramidal $\check{I}$ regions. This mechanism permits the more sublimical excitations always to exert an effect on peripheral dendritic regions, which are the regions incapable of making fine input discriminations and incapable of decisively controlling pyramidal output, anyway. By contrast, inputs from cells in the lower layers, like the $\mathcal{J}$ cells, are much more volumetrically bound to $\check{I}$, and will be strongly inhibited if they do not determine specific deviations from the homogeneity of $\mathcal{F}(\oplus_i p_i)$ excitation. Thus, both the excitatory and the inhibitory horizontal interactions between specific layers of cells become more finely sculpted as the layer is moved from the periphery inward.

It follows from this discussion that pyramid output cells whose cell bodies and dendrites lie in a layer above the most densely packed stellates will asymptotically be capable of distinguishing only certain coarser features of input distributions than the sensitive pyramids whose cell bodies lie in a layer containing extensive input line terminations and a rich stellate field. The transmissions of such more superficial pyramids are determined by these coarse input features. If both coarse and sensitive pyramids from a single cortical column project to a single input-output locus, displaced at a distance, the coarse transmissions generally arrive before the sensitive transmissions do. The recipient point is hereby allowed to itself begin the process of refining its space of equivalent potential distributions in a gradual way. By the time the more highly discriminating transmissions arrive, the locus has already begun the refinement process and can efficiently process the new input details.

In particular, if the recipient locus is itself a cortical column, it is natural to expect the coarse (sensitive) pyramids to send lines preferentially to the recipient layer of coarse (sensitive) pyramids to ensure maximal distinguishability of inputs. Such an expectation is compatible with the knowledge that the sensitive pyramids can distinguish all of the features of the coarse inputs, for the sensitive pyramids send dendrites to the layers of the variously coarse-grained pyramids. If, on the other hand, all output cells from the superficial layers projected their early outputs rather directly to the most sensitive pyramidal cell bodies, the M-s values of these pyramidal bodies would be small before the more finely discriminating inputs arrived. These input details would therefore have only a negligible dynamical effect on output firing. This difficulty is partially offset by staggering input fibers, as above, along the pyramidal dendrites and cell body, so that the coarsest inputs arrive at the dendrites furthest from the pyramidal cell body. The most distal inputs thus reach the cell body with the greatest electrotonic decrement, whence the fine input details are preferentially weighted.

The set of layers to which a given pyramid sends its dendrites thus provides a direct geometrical measure of its dynamical sensitivity. For example, if all pyramids send apical dendrites to the layer of horizontal cells, then all pyramids are initially activated by the crudest subliminal potential shifts. The structure of fields of associational neurons through which the dendrites and cell body of a given pyramid pass, the density of the pyramidal links with the associational loci, and the method of propagation of potentials from these links to the cell's $\check{1}$ region determine the cell's asymptotic sensitivity.

Another way to ensure that a deep pyramid is not flooded by preliminary coarse excitation is to ensure that its cell body has a relatively large volume in which coarse dendritic inputs are diluted. Thus, the pyramids with the longest vertical dendrites should have the largest cell body volumes. These remarks show that a spatial self-similarity of the vertically oriented pyramids contributes to a sensitization of the entire cortical column to subtle input variations, including such simple variations as contractions in the temporal scale of the input paradigm. A quantitative discussion of this spatio-temporal self-similar interaction will be given in another place.

138. Direct Betz Cell Projections vs. Multi-synaptic Chains

An important qualitative consequence of such a spatio-temporal self-similarity can, however, be immediately drawn. If the class of pyramidal cells really does enjoy a type of spatial self-similarity, then the pyramids which have the longest dendrites and the largest cell bodies will also have the longest axons. These axons are often sufficiently long to project almost directly to spinal motor centers, while the smaller pyramids manifestly cannot reach such loci directly, and must depend on multi-synaptic links. These directly projecting pyramids are precisely the pyramids which can best discriminate cortical inputs. We are therefore confronted with a very wise method of distributing cortical outputs. For it is safe to let some of the largest pyramids project directly to spinal motor centers because these pyramids possess a very stable firing pattern which is highly sensitive to variations in cortical inputs. Picturesquely stated, these large pyramids do not "make mistakes" of input indentification in their firing pattern, so that it is unnecessary to dynamically shield the spinal motor centers from them with a multi-synaptic chain of neurones Indeed, these direct pyramidal spinal projections emerge in increasing numbers as the phylogenetic scale is ascended. This fact underlines the highly adaptive, but nonetheless evolutionarily advanced, character of these very sensitive and direct spinal projections.

It is natural to ask whether a multi-synaptic chain of neurones accomplishes a similar task, and the answer is "yes". For the interpolation of many synapses markedly increases the opportunities for convergence and divergence of neural connections between the peripheral input and spinal output control centers. We have just seen, however, in considering the formation of weighted lines of force in stellate fields that increasing the convergence and divergence of stellate cells increases the stellate field's capacity to distinguish input variations in its distribution of line residues. The argument is formally identical for any system of converging and diverging neuronal chains, including a chain initiated by a small pyramid cell. The innumerable examples of converging and diverging neuronal structures, indeed, provides a convincing global geometrical indication that a local dynamical strong-coupling system must exist. Moreover as we saw in studying fields of type M, one can adjust the threshold and input line densities of a multisynaptic link so that a nontrivial fraction of lines must be simultaneously excited for transmission to occur. This is the output

analog of increased input distinguishability.

The comparison between a multi-synaptic pyramidal chain and a directly routed Betz pyramid reveals a general evolutionary neural tendency. In a multi-synaptic chain, the dynamical mechanism for increasing the distinguishability of inputs is spread over a broadly distributed set of neurons, which in the lowest invertebrates approximates an almost homogeneous mesh of undifferentiated associational neurons randomly juxtaposed between input sources and output sinks. In a Betz cell, by contrast, the entire associational mechanism has been compressed into a remarkably small and efficient strip of neocortex, totally eliminating the need for many interpolated chain links. This deformation of the associational mechanism into comparatively localized fields is paralleled by the emergence of neocortex, in general, and the specific sensory and motor fields, in particular.

It is an outstanding feature of this development that the influence which many cells come to exert on the transmission of a single cell is determined by a delicately balanced, and inhomogeneous, interaction structure that can distinguish far more inputs than the equipotential interaction structure in the most primitive neural nets. Physical theories, to the present, have very often limited themselves to formal constructions of the physical analogs of such equipotential nets by studying experimental situations for which a covert, or explicit, chaotization principle is appropriate. In a highly polarized and inhomogeneous cortical column, and its physical field analogs, a global chaotization principle is manifestly unappropriate, as is a dependence wholly on a homogeneously grained dynamical superposition principle. Moreover, although a cortical column acts as a tightly-bound input-output unit, it is composed of many smaller parts whose relative geometrical relations are thoughtfully constructed to transform the entire column into a stable and highly refined input-output mechanism. These smaller parts therefore behave as "hidden variables" when one considers the cortical column as a "black box" in first approximation. Such an approximation is possible when only physiological inputs are delivered to the column. For every physiological input has a natural dynamical grain that is predetermined by the spray of fibers from every source to its cortical columnar terminal locus, and every spray configuration is itself partially determined by the geometrical symmetries and

customary level of excitation of its terminal locus. If, on the other hand, cortical inputs are introduced in a man-made, nonphysiological manner, small clusters of cortical cell units will introduce local, "unhidden" nonlinearities for which an asymptotic column input-output operator is unsuitable. This emergence of local nonlinear variables must be expected whenever man-made experiments locally perturb a stable nonlinearly-bound physical system in an "unphysiological" way, and the measuring device of this perturbation is itself highly focalized.

139. A Remark on Artificial Epileptic Foci and Cortical Conditioning

Another qualitative conclusion following from these considerations is that disrupting the normal activity of the superficial layers of a critical region of cortical columns early in conditioning might interfere more with the animal's expected behavior after training than a similar disruption after conditioning has already occurred. For early in the conditioning process, the sensitive $\mathcal{J}$-chains of the deep layers have not yet been stably established, and line residues will reflect coarse averages throughout the cortical column. After conditioning has occurred, the stable $\mathcal{J}$-chains of the lower layers assume relatively greater importance in controlling subtle behavioral variations, so that disrupting the superficial layers at this time causes strength scatter within a relatively less important subfield of lines residues. Such a superficial disruption of normal activity can be effected by inducing an artificial epileptic lesion, and the expected disruptive effects have been observed by F. Morrell. It must be remarked, however, that natural conditioning of cortical columns inevitably is accompanied by companion conditioning within the specific thalamic nuclei with which these columns interact. Even after the superficial cortical layers become dynamically chaotic, a considerable subcortical line residue remains. Whether or not this subcortical component also becomes chaotic depends on the particular transmission pattern of the superficial layers to their subcortical foci induced by the lesion.

By similar arguments, one can see that inducing a man-made potential within localized cortical columns with the physiological polarity while these columns are receiving behaviorally generated inputs creates a statistically significant strong coupling situation if the dendritic branches of the larger pyramids are differentially excited. Cortical conditioning can therefore often be expected. Such a potential also, of course, permits the pyramid cells to fire under less intense physiological inputs.

The interpretation of this conditioning is not always straightforward because polarization does not imitate the usual sensitively graded excitation of all columnar layers which would have followed if the polarizing inputs had been delivered through natural fiber bundles which were strongly coupled to the column before conditioning began. Moreover, the behavioral inputs form line residues which are concentrated on the small number of artificially polarized columns, and it is very rare that localization of this kind occurs without companion global renormalizations under physiological conditions. Indeed, global subcortical renormalizations of the cortical fields and cortico-cortical lateral inhibitions can entirely obscure the effect. Nonetheless, the possibility of such cortical conditioning under fortuitous physiological background conditions is a simple consequence of our discussion of laminar interactions. It should also be observed that unless the pyramidal cell bodies are directly excited, the conditioning will seem to be restricted largely to the dendrites. Even if the cell bodies are excited, the fact that stable $\mathcal{S}$-chains form only after repetitions of well-sculpted input distributions will tend to place deceptive emphasis on the more apical dendritic couplings. Such dendritic effects have been studied by F. Morrell.

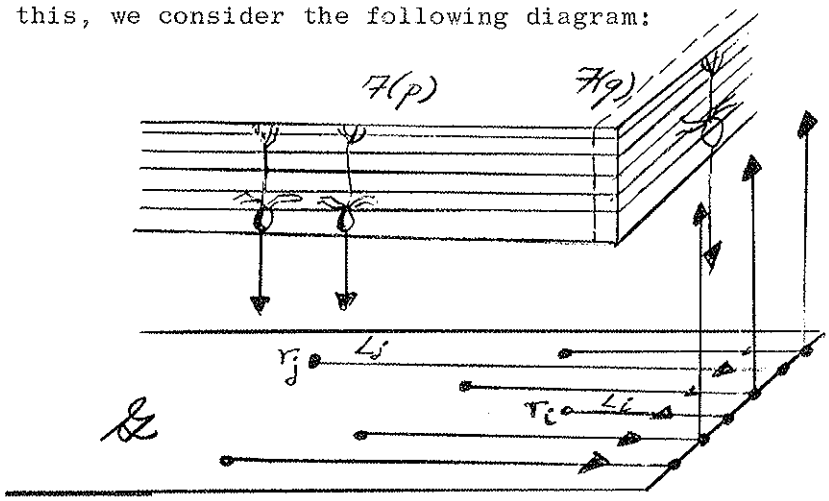
140. Respondant Conditioning

It is clear that this discussion of intra- and inter-columnar cortical interactions is completely compatible with the essential features of the familiar learning paradigm of respondant conditioning, and provides new insights into the finer aspects of this paradigm. Operant conditioning requires a direct consideration of global renormalization factors, to which

we shall later turn. It is important to realize that we have, in fact, implicitly determined a field renormalization in this discussion, for the inputs in our thought experiments have, in all cases, been allowed to reach the cortex without serious decrement.

141. Parallel Fibers

The structure of $\mathcal{G}$ will now be sketched. We want $\mathcal{G}$ to violate the surface topology of the P_k layers so that all regions of $\mathcal{T}$ can be used to effectively control the inhomogeneously distributed output points $\{q_i\}$. In particular, we want to remove the $\mathcal{T}(q)$-induced polarity of $\mathcal{T}(p)$, with which a radially symmetric distribution of horizontal fibers is incompatible. To do this, we consider the following diagram:

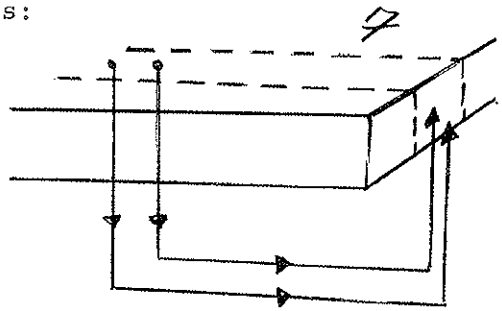


The major structural components of $\mathcal{G}$ are points r_i which send lines L_i to the points $\bar{q}_i$. The L_i lines run parallel to the layers of $\mathcal{T}$, and the $\bar{q}_j$ points lie vertically below the q_j points. We assume that the transmission time velocity within all the L_i is the same, and that L_i of every length are found with equal probability coursing from below some p_i point to some $\bar{q}_i$. These conditions immediately remove the $\mathcal{T}(q)$-polarity of $\mathcal{T}(p)$ when we consider the

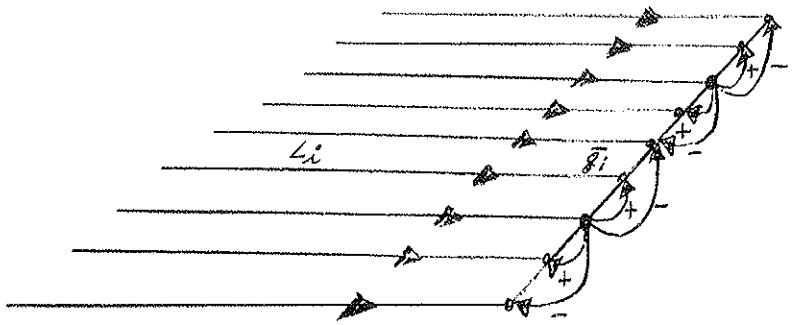
total field $\mathcal{T} \oplus \mathcal{R}$. For example, if a single p_i point is excited, it sends out radially symmetric waves to nearby p_j points. All points equidistant from p_i receive the same wave at the same time. If one such point transmits to $\mathcal{R}$, all such points do, and all transmissions from a given output cell type reach $\mathcal{R}$ at the same time, by symmetry. Since the transmission time from any r_i to $\mathcal{T}(\bar{q})$ is independent of distance, the transmissions induced by the various radially symmetric p_j points all arrive at $\bar{q}_i$ and hence at $\{q_i\}$ at the same time. In the same way, we see that any two identical, simultaneous inputs to $p_1, p_2 \in \mathcal{P}(\mathcal{T})$ induce transmissions which arrive simultaneously at $\bar{q}_i$, and hence at $\{q_i\}$, with equal intensity. These two transmissions are, in fact, the same up to a shift of locus along $\bar{q}_i$ axis. We call the L_i parallel lines, or parallel fibers, because they run parallel to one another in great profusion. The parallel fibers increase the effective receptive area of the q_i by an enormous factor.

142. Lateral Inhibition in $\mathcal{R}$

The cycle of output line transmission from $\mathcal{T}(p)$ to $\mathcal{R}$, followed by parallel line transmission, and transmission from $\mathcal{T}(\bar{q})$ to $\mathcal{T}(q)$ effectively decomposes $\mathcal{T}$ into a series of parallel strips with respect to the control of $\mathcal{T}(q)$ muscle groups:



It is obviously important that when many parallel fibers are simultaneously excited, only the most intensively activated fibers be allowed to transmit to $\mathcal{T}(q)$. Otherwise, $\mathcal{T}(q)$ would be flooded by background excitation. An ordered lateral inhibitory mechanism is therefore called for. The value of such a structure is also apparent from the fact that $\mathcal{T}(p \oplus q)$ itself exhibits a mixed line structure of the "on"-"off" type. Since parallel fibers which are close to one another excite nearby $\mathcal{T}(q)$ points, and are excited by nearby $\mathcal{T}(p)$ points, the lateral inhibitory mechanism in $\mathcal{Q}$ should also be of the "on"-"off" variety. Radial symmetry in $\mathcal{T}$ becomes transverse symmetry in $\mathcal{Q}$, so that we expect to find a mechanism like:



in $\mathcal{Q}$, where the parallel directed lines are parallel fibers, and the (+) and (−) signs refer to excitatory and inhibitory lines, respectively. To preserve the transverse symmetry of the bundles of parallel fibers, we choose these "on"-"off" distributions in such a way that they are identical replicas of one another when their source points are shifted transversally with respect to the parallel fibers.

We would like this transverse lateral inhibitory mechanism to operate before massive $\mathcal{T}(\tilde{q}) \longrightarrow \mathcal{T}(q)$ transmissions become possible. The threshold for transverse lateral inhibition must therefore be less than the threshold for $\mathcal{T}(\tilde{q}) \longrightarrow \mathcal{T}(q)$ transmission. If all parallel fibers are simultaneously excited by the same input function, the transverse lateral inhibitory mechanism will

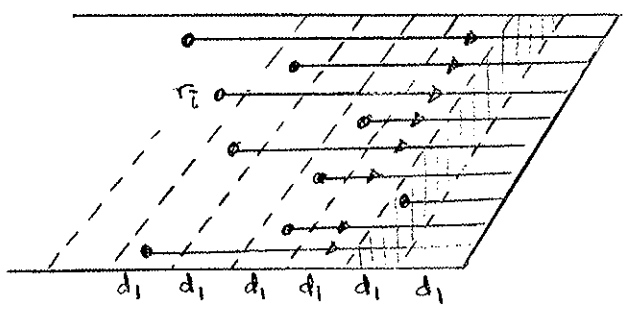
inhibit all terminal $\bar{q}_i$ points with equal intensity. We suppose that the inhibitory lines are sufficiently dense that for all admissible inputs, uniform excitation of $\mathcal{T}(\bar{q})$ is suppressed before $\mathcal{T}(\bar{q}) \longrightarrow \mathcal{T}(q)$ transmission occurs. The set $\{\bar{q}_i\}$ thus forms a complete set of antagonists relative to the set of all parallel fibers. Only those inputs to the parallel fibers which mix a proper measure of intensity and nonuniformity of distribution can hope to excite $\mathcal{T}(q)$. This restriction is physically very reasonable, for the uniform excitation of an entire field of muscle control forms can only spell behavioral confusion.

A difficulty is, however, immediately encountered under such a condition. Since the lateral inhibitory lines are strong enough to quell even maximal uniform excitation of the set of parallel lines, when only a small number of parallel fibers are excited, it is quite possible that a much smaller lateral inhibitory transmission will suppress $\mathcal{T}(\bar{q}) \longrightarrow \mathcal{T}(q)$ transmission. This is possible even if the normalized distribution of the less intensive excitation is more distinctive--has a higher average information value--than the normalized distribution of the excitation of many fibers. We must remove this undesirable suppression of less intensive excitation distributions, and we must do so while retaining (i) a weakened notion of complete sets of antagonists, and (ii) some form of transverse symmetry in the new inhibitory mechanism. The simplest way to do this is to consider the following sets:

$$R(d) = \left\{ r_i : \quad \min_j \left| r_i - \bar{q}_j \right| = d \right\},$$

$$\mathcal{L}(d) = \left\{ L_i : \quad r_i \in R(d) \right\}$$

$R(d)$ is the set of all r_i which are at distance d from the set $\bar{q}_j$, and $\mathcal{L}(d)$ is the set of all parallel fibers which are emitted by points in $R(d)$. Instead of grouping all points in a single complete set of antagonists at $\{\bar{q}_j\}$, we suppose that a complete set of antagonists is adjoined to every set $\mathcal{L}(d)$. Since only a finite number of points exist, we can replace the continuous variable d by a discrete set of distances $d_1, 2d_1, 3d_1, \ldots, nd_1$, $d_1 \ll d$, which induce a partition of the r_i's as follows:



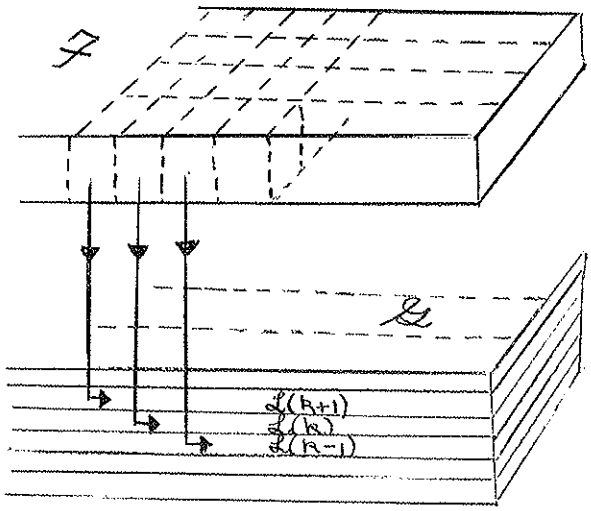
Corresponding to this discrete partition, we replace the sets $\mathcal{L}(d)$ by the sets

$$\bar{\mathcal{L}}(m) = \left\{ L_i : \quad (m-1)d_1 \leq \min_j \left| r_i - \bar{q}_j \right| < md_1 \right\} .$$

Since we shall no longer have occasion to refer to the $\mathcal{L}(d)$ lets, we write $\mathcal{L}(m)$ instead of $\bar{\mathcal{L}}(m)$ without danger of confusion. Each $\mathcal{L}(m)$ shall be given its own complete set of antagonists. The set $\{\bar{q}_j\}$ must also be expanded if it is to carry the n new complete sets of antagonists. The obvious way to do this is to replace $\{\bar{q}_j\}$ by n sets $\{\bar{q}_j^{\,k}\}$, $k=1,2,\ldots,n$, where the set $\{\bar{q}_j^{\,k}\}$ carries the antagonists for $\mathcal{L}(k)$. The $\{\bar{q}_j^{\,k}\}$ inhibitory mechanism affects a rectangle of r_i points, namely $\mathcal{L}(k)$, rather than all r_i points. The various $\{\bar{q}_j^{\,k}\}$ sets must be arranged in some regular order relative to one another which permits their individual internal inhibitory mechanisms to operate without interfering with each other. The simplest way to do this is to let them lie in layers, with the $\{\bar{q}_j^{\,k}\}$ layer lying above the $\{\bar{q}_j^{\,m}\}$ layer if and only if $k > m$. The parallel fibers therefore also lie in layers consisting of the $\mathcal{L}(k)$ sets. These "layers" really mesh smoothly with one another, since the length of the fibers is not strictly quantized by d_1. The decomposition using a d_1 parameter imposes convenient conceptual boundaries, but the cellular interactions themselves are not really strictly circumscribed within these boundaries. This deformation of the L_i into $\mathcal{L}(k)$ layers leaves all temporal relations invariant if the transmission velocities of the input-output lines are chosen proportional to line length.

143. Cellular Parallelopipeds

The decomposition of the parallel fibers into layers has the desirable consequence that lateral renormalizations only mutually affect p_i's which lie at approximately equal distances from $\mathcal{T}(q)$. The surface $\mathcal{T}(p)$ is therefore effectively decomposed into transverse regions relative to transmission through $\mathcal{G}$ to $\mathcal{T}(q)$. But $\mathcal{T}(p)$ has already been decomposed into parallel longitudinal regions as well, by the fact that all parallel fibers in a given column lead to the same $\mathcal{T}(q)$ point. Combining these two decompositions, we see that $\mathcal{T}(p)$ is now effectively decomposed into small rectangular parallelopipeds relative to $\mathcal{T}(q)$:



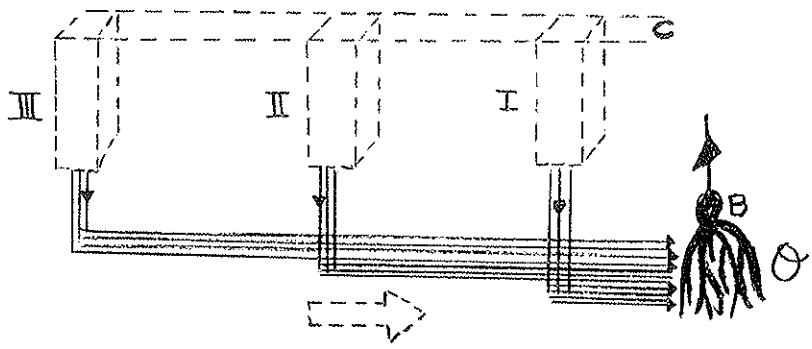
By strongly violating the surface topology of $\mathcal{F}(p)$, we have nonetheless arrived at a typically Euclidean kind of functional decomposition of $\mathcal{F}(p)$! This decomposition of $\mathcal{F}(p)$ is obviously a very efficient use of the available space, and it is accomplished without any essential loss in symmetry.

144. Dendritic Bushes

Individual $\bar{q}_j^k$ points need not serve both as output points of $\mathcal{F}(\bar{q}) \rightarrow \mathcal{F}(q)$ lines, and as lateral inhibitory and excitatory sources. Indeed, when field-antifield duality holds, as we always suppose, they cannot. Our previous experience with inhibitory interneurons suggests that the lateral inhibitory and output roles repose in different cells. How can we construct these cells to take advantage of the functional decomposition of $\mathcal{F}(p)$ into rectangular parallelopipeds? The roughness of the $\mathcal{F}(p)$ parallelopiped grain must be reflected in the structure of the lateral inhibitory and output lines. This roughness may also be expressed by saying that these cells must respond to local averages of parallel fiber excitation, and these averages must be taken over roughly parallelopiped-shaped sections of parallel lines. We thus need a cellular structure which automatically computes averages over parallelopipeds of closely packed parallel fibers. Rich dendritic bushes are such structures. The lateral inhibitory and output cells thus send out dendritic bushes to parallelopiped-shaped regions of parallel fibers.

The splitting of the two processes of lateral inhibition and output transmission into two different sets of cells allows us to restrict the complete sets of antagonists roughly to $\mathcal{L}(k)$ sets without restricting the dendritic bushes of the output cells to such sets. We can therefore construct output cells whose

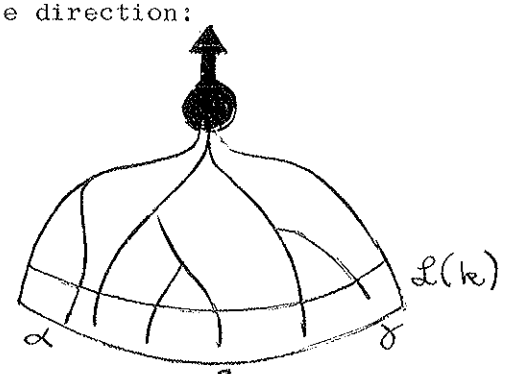
profuse dendritic bushes are responsive to all parallel fibers which lie in a given column of parallel fibers. Although these output cells $\mathcal{O}$ are sensitive to an entire column of p_i cells, since only closely juxtaposed p_i parallelopipeds mutually inhibit one another in the $\{\bar{q}_j^k\}$ sets, the $\mathcal{O}$ cells can be made to transmit even when only a limited number of p_i cells transmit. It is advantageous for another reason that a single $\mathcal{O}$ cell be sensitive to an entire column of p_i cells. Each q_i is organized into a tightly bound column of cells. If a series of $\mathcal{O}$ cells projected from $\mathcal{K}$ to each q_i, this well-organized columnar structure, which is so well-suited for stable output transmission, would be split into many parts of lesser stability. Moreover, the $\mathcal{O}$ cells whose dendrites reach all $\mathcal{L}(k)$ combine sensitivity to a large sector of the $\mathcal{I}(p)$ field with no loss of specificity. In fact, consider the diagram:



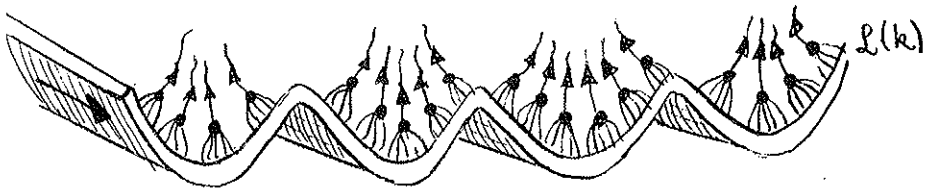
145. Folia

The dendrites of $\mathcal{O}$ are spread out broadly in a transverse direction and throughout the $\mathcal{L}(k)$ layers to be sensitive to excitation averages transmitted by all of C. Therefore, (II)'s transmissions can be completely inhibited by lateral inhibition without in the least affecting the transmissions of (I) and (III). Notice that the longest fibers arborize closest to the cell body B of $\mathcal{O}$. If $\mathcal{O}$'s dendritic bush is uniformly distributed, (III) will exert a greater influence on $\mathcal{O}$'s firing than (I) or (II) will. On the other hand, if short parallel fibers were to exist with higher probability than long fibers, the advantage of (III) in arborizing near B would compensate for the relative sparseness of long fibers. (III)'s advantage can, in fact, be easily reduced in several ways, the simplest way being to suppose that long fibers are less numerous than short fibers. Another way is to ensure that the density of the dendritic surface varies inversely with its distance from the cell body B of $\mathcal{O}$. When the dendritic surface density does depend on distance from the cell body, it will be difficult to assure that all fibers in a fixed $\mathcal{L}(k)$ have an

equal influence on B if $\mathcal{L}(k)$ lies in a planar slab. For we have assumed that all parallel fibers of equal length are equally dense transversally, while some $\mathcal{L}(k)$ fibers must terminate on the relatively sparse peripheral branches of $\mathcal{O}$. It is easy to correct this unpleasant situation. We simply curve the $\mathcal{L}(k)$ slabs slightly in the transverse direction:

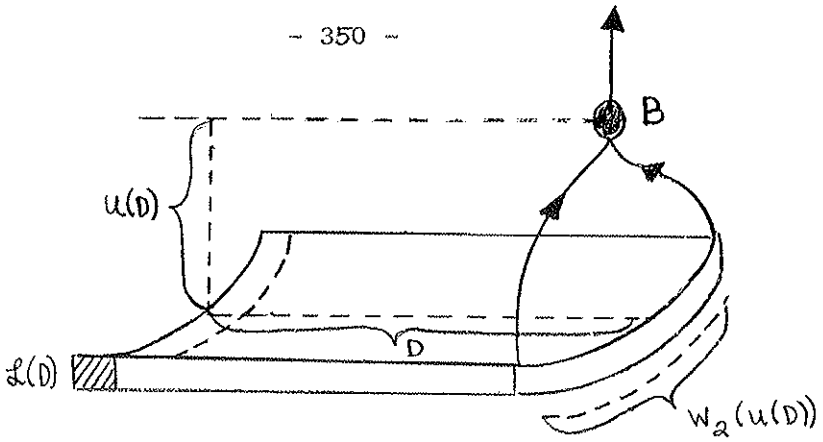


Now the β fibers have no advantage over the peripheral α and γ fibers. When we extend this arrangement to the entire collection of $\mathcal{O}$ cells, we find the following diagram:



$\mathcal{L}$ is hereby split into a series of identical parallel folia. Such folia also permit a much closer packing of $\mathcal{O}$ cells in Euclidean space--without changing the functional geometry--than would have been possible with planar layers.

More generally, let $w_1(D)$ be the density of parallel fibers at a distance D from B, $w_2(D)$ be the curved breadth of the dendritic bush at distance D from B, and $w_3(D)$ be the total effective surface area of dendritic bushes in a transverse row D units distant from B. Also let $\mathcal{L}(D)$ lie at a mean distance u(D) from the longitudinal projection of B along :

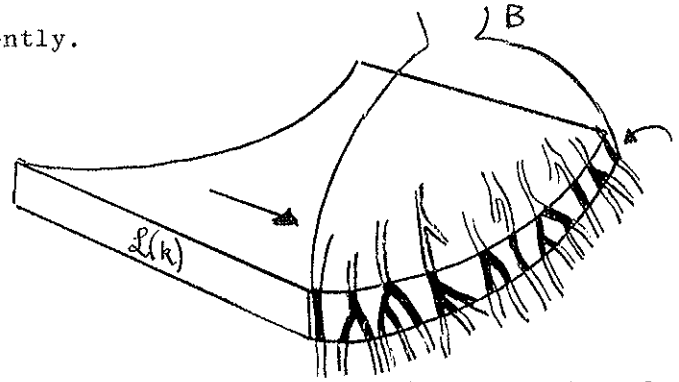


If (1) all dendritic branches of $\mathcal{O}$ contribute equal additive excitations to B when they receive equal transmissions (equipotentiality), and (2) equally excited parallel fibers contribute equal excitation densities to $\mathcal{O}$'s dendritic bush, then every $\mathcal{L}(D)$ will carry an equal weight in determining the dynamics of B if and only if

$$w(k) = w_1(u(kd_1))w_2(u(kd_1))w_3(u(kd_1))$$

is independent of k=1,2,...,n. This condition is, of course, true only in a statistical sense, since each $\mathcal{L}(k)$ is a thin band of parallel fibers. We also know how to immediately generalize this condition when (1)-(2) are not satisfied. When (1) is violated, we simply include in w(k) a factor that expresses the inverse of the expected relative decrement which an excitation from $\mathcal{L}(k)$ undergoes while being transmitted to B over $\mathcal{O}$'s dendritic bush. If (2) is violated, we include a factor, as we did in discussing spatial self-similarity, that shows how excitation densities are transformed over the system $r_i \longrightarrow L_i \longrightarrow \bar{q}_j^{\,k}$.

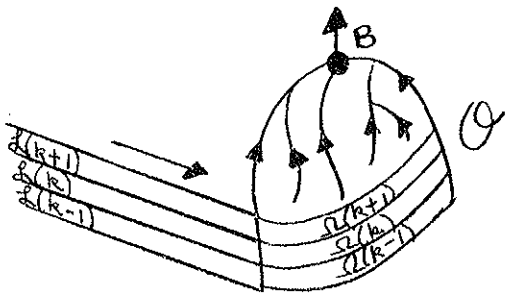
The successful imposition of such conditions on $\mathcal{L}(k)$ sets depends on peculiarly dendritic properties. In particular, the dendritic segments reached by each $\mathcal{L}(k)$ lie in a large number of different dendritic branches, and can therefore be viewed as local mechanisms which are capable of acting independently.



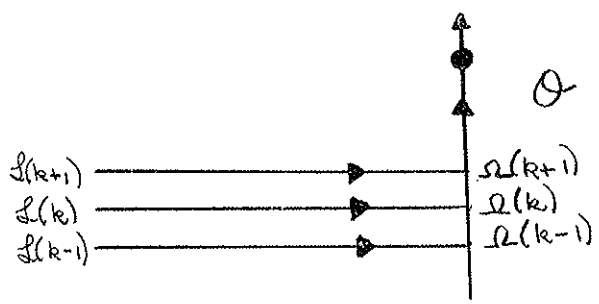
Were this not the case, the averaging operations leading to such quantities as w_1, w_2, and w_3 would not commute, and our conditions would become senseless.

146. Dendritic Cross-Sections

To clarify the abstract situation underlying these conditions, we introduce some definitions. Our concern is with the way in which p_i cells influence q_j cells. This concern leads, after a brief discussion of how lateral inhibitory mechanisms should be introduced into $\mathcal{G}$, to a natural decomposition of parallel fibers into horizontal $\mathcal{L}(k)$ bands and to the construction of output cells $\mathcal{O}$ whose profuse dendrites run transversally through all of the bands which serve a given q_i point. Each $\mathcal{L}(k)$ decomposes the $\mathcal{O}$ dendrites into transverse bands, which become the effective geometrical units of the dendritic bush. We call these bands dendritic cross-sections. The cross-section associated with $\mathcal{L}(k)$ is called the k-cross-section, or $\Omega(k)$. The $\Omega(k)$ are the natural regions into which to decompose the dendrites of $\mathcal{O}$ when we view $\mathcal{O}$ as an averaging device. This is true for several reasons: (1) We already noted that the individual dendritic segments in $\mathcal{L}(k)$ behave quite independently, at least when they are compared to the more volumetrically bound subregions of B, just so long as B does not send strong currents into the dendritic bush. These segments can therefore be viewed locally as the structural carriers of an additive random process, whence all $\mathcal{L}(k)$ contributions to $\Omega(k)$ can immediately be lumped into the contribution of a single virtual point. (2) We must, however, lump very carefully, for the $\Omega(k)$ contribution is not delivered directly to B. Rather, it must be propagated over $\bigcup_{m=1}^{k-1}\Omega(m)$. Whenever this propagation process is so homogeneous across each $\Omega(m)$ that the transverse additivity of the individual $\Omega(k)$ components is left invariant under it, the $\Omega(k)$ decomposition still has a functional interpretation when it is applied to dendritic impulses up to the time they reach B. It is in this sense that



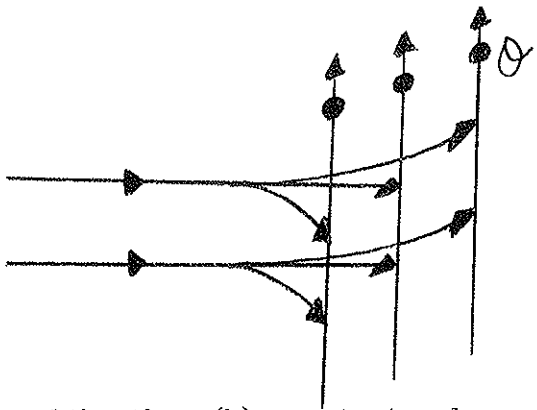
can be contracted to



(∝)

which is the minimal field for the dendritic bush and provides an immediate insight into the structure of the transmission process. If we placed a single output line on some α dendrite, this minimal field decomposition would become invalid. For the computation of such minimal fields and their dynamically indistinguishable sets is motivated entirely by a desire to see in the most parsimonious way how given input mechanisms interact with an interpolated field to excite given output mechanisms. By varying the distribution of input or output mechanisms, even without changing the interpolated field, we alter the problem completely. Such an alteration can be accomplished even without changing the local field geometry. For example, if 100,000 wildly distributed input lines are always activated together, we will be strongly inclined to search for a minimal field in which all 100,000 wildly distributed input lines are collapsed into a single input mechanism.

The transmission lines from each p_i characteristically terminate on many transverse p_j. This dynamical overlap is reflected in the transmissions from $\gamma(p)$ to β, and finally to the α cells. We therefore expect that the transverse dendritic bushes of nearby α cells will also overlap. If we require the p_i cells to send out fibers to p_j points at increasingly large transverse distances, we must also require increasingly large numbers of α cells to send their dendrites to common parallel fibers. When we allow α cells to share parallel fibers, the simple diagram (α) is not sufficient. It can be replaced by



In discussing rules like the w(k)=constant rule, we must now also be careful to distinguish the parallel columns in which α's dendrites lie. Whenever the superposition of local dendritic components is a valid approximation to reality, this new distinction produces no additional difficulties, and we can easily compute the relative excitatory structural contribution of dendrites from each column of a given α cell. When an input is delivered to a fixed column of parallel fibers, α cells in several adjoining columns will be excited, but this excitation will diminish as the transverse distance of the α cell from the excited column

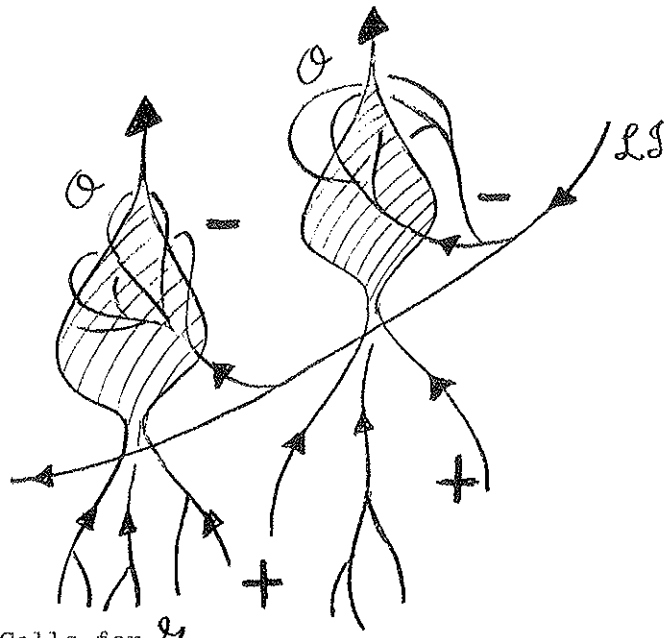
increases. The partition into parallel folia thus does not ensure equipotentiality for all p_i projecting to a given $\mathcal{O}$ in this more general case of overlapping $\mathcal{O}$ cells. Such a violation of equipotentiality is useful because graded $\mathcal{T}(p)$ interactions should map into graded $\mathcal{O}$ interactions. The folia still serve a useful purpose, however. When folia are present, the violation of equipotentiality is determined by the local cellular control mechanisms which determine the shape of the $\mathcal{O}$ dendritic bush. It is not determined by functionally accidental variations in the curvature of the $\mathcal{L}(k)$ bands. Moreover, even when all $\mathcal{L}(k)$ contributions are not equipotential, each $\mathcal{L}(k)$ carries an additive random process, even though its increments under random inputs are not equally weighted. In fact, when the dendritic lengths of a given $\mathcal{O}$ are broadly distributed, the dendrites contribute excitations to B which are graded temporally as well as spatially with respect to the $\mathcal{L}(k)$ deomposition.

Perhaps the most important qualitative conclusion to be derived from this discussion is that whenever many cells act "in series" to determine a given input-output flow, altering the distribution of one of these cells immediately imposes a restriction on the possible distributions of the other cells, no matter how widely dispersed these cells are in Euclidean space. The particular restrictions introduced depend on particular symmetries of the geometrical arrangements of the cells, but do not introduce new qualitative considerations.

147. Inhibitory Baskets

We can now introduce the lateral inhibitory cells $\mathcal{LI}$ without difficulty. Their distribution will be broad whenever the transverse "off" regions of the p_i cells are broad. The distribution of $\mathcal{LI}$ cells determines whether the complete sets of antagonists are derived from columns, parallelopipeds, or other $\{p_i\}$ configurations. We choose parallelopipeds here because the p_i cells themselves interact as parallelopipeds. The $\mathcal{LI}$ cells obviously send out transverse branches to the $\mathcal{O}$ cells. When both lateral inhibition and $\mathcal{O}$ output were controlled by $\bar{q}_i$ cells, we needed the requirement that the $\mathcal{T}(\bar{q}) \rightarrow \mathcal{T}(q)$ transmission threshold exceeds the lateral inhibitory transmission threshold. The idea underlying this condition was that the transverse lateral inhibition must renormalize $\bar{q}_i$ cell excitation before output transmission occurs. Now that we

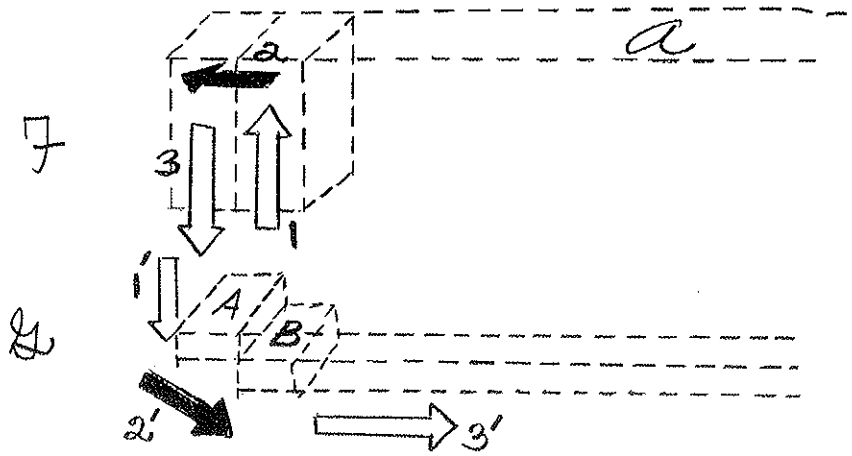
have both $\mathcal{O}$ and $\mathcal{LI}$ cells, we can easily improve on this threshold mechanism by noticing that the quickest way to renormalize an $\mathcal{O}$ cell's output transmission is to let inhibitory fibers terminate near its $\check{1}$. We thus expect the $\mathcal{LI}$ cell axons to split into fine bundles, or baskets, which are spread over the body of $\mathcal{O}$ cells, particularly near the initial segment of the output lines.



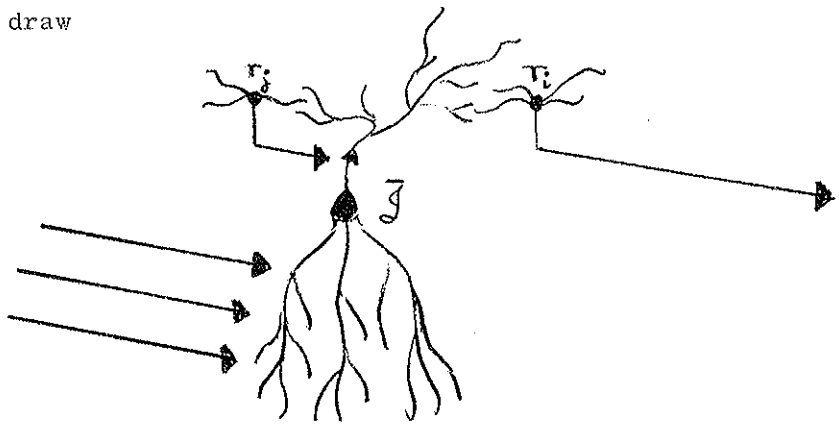
148. Interpolated Cells for $\mathcal{Y}$

Juxtaposed parallel fibers lead from mutually excitatory p_i points to overlapping dendritic bushes. It is therefore desirable to bind such fibers into units also, whence we expect to find excitatory $\mathcal{J}$ cells, often distributed in a transverse direction, with fibers not so long that they block $\mathcal{LI}$ inhibitory interactions.

Is there a natural analog of an $\mathcal{I}$ cell in $\mathcal{Y}$? The answer is yes. The shape of these cells, which we call $\mathcal{J}$ cells, will not be the same as that of the $\mathcal{I}$ cells because the details of the geometries of $\mathcal{T}$ and $\mathcal{Y}$ differ. But their function should be the same. To see how the $\mathcal{J}$ cells must look, consider the diagram

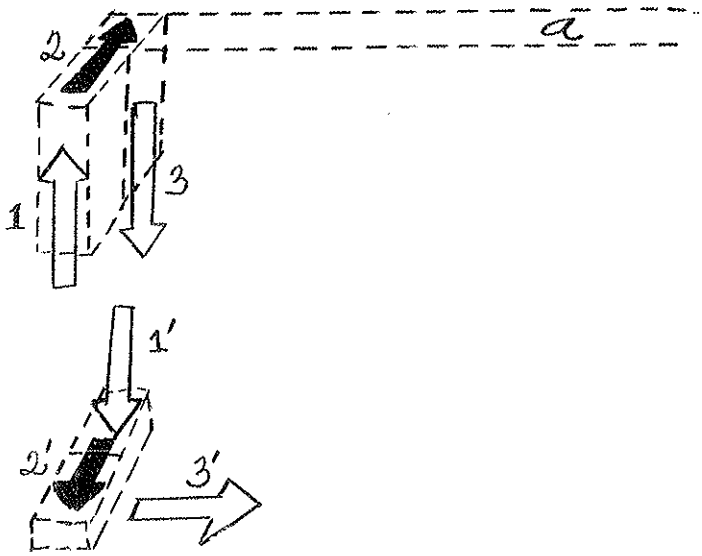


The arrows (1) and (3) are input and output arrows for successive parallelopipeds in the $\mathcal{P}(p)$ column denoted by a. The (2) arrow gives the orientation of the $\mathcal{I}$ chains subserving the (1)-(2) cycle. The (i') arrow in $\mathcal{Q}$ corresponds to the (i) arrow in $\mathcal{P}$, i=1,2,3. A horizontal (2) arrow in $\mathcal{P}$ becomes a diagonal (2') arrow in $\mathcal{Q}$. The $\mathcal{I}$ cells corresponding to (2') must therefore send dendrites to the r_i cells of A and axons to the r_i cells of B, which lie in a lower layer. Whenever long $\mathcal{I}$ chains in $\mathcal{P}$ can effectively be excited, then $\mathcal{I}$ cells must be able to join widely separated $\mathcal{L}(k)$ layers in $\mathcal{Q}$. Horizontal symmetry in a longitudinal direction in $\mathcal{P}$ is thus translated into diagonal symmetry in a longitudinal direction in $\mathcal{Q}$, with the same orientation. The $\mathcal{I}$ cells thus send dendritic trees through a $\mathcal{Q}$ parallelopiped. These trees terminate in a cell body which sends axons to the r_i dendrites of a different $\mathcal{L}(k)$ layer, diagonally juxtaposed. The r_i dendrites cannot be distributed throughout many $\mathcal{L}(k)$, for the specificity of the parallelopiped interaction in $\mathcal{P}$ will be lost by such a distribution. Thus we can draw



The dendritic bush of $\mathcal{I}$ can be decomposed in the same way in which the bush of $\mathcal{O}$ was decomposed.

Now consider

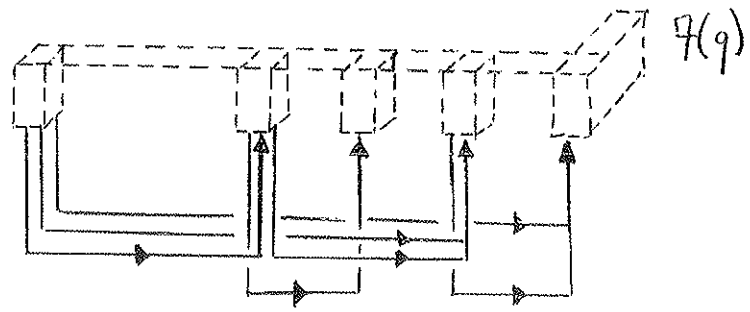


Here horizontal (2) motions are transformed into horizontal (2') motions. We already have seen a cell type which behaves like (2'), namely the $\mathcal{J}$ cells. The $\bar{\mathcal{J}}$ cells can also be made to behave like (2') by simply shifting their orientation to the transverse direction. Thus, both $\mathcal{J}$ and $\bar{\mathcal{J}}$ cells contribute in $\mathcal{G}$ to realize aspects of $\mathcal{I}$'s behavior in $\mathcal{7}$.

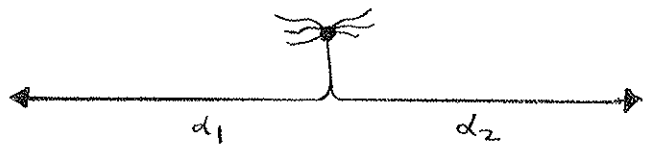
149. Stationary Cross-Sections

To understand the roles of $\mathcal{J}$ and $\bar{\mathcal{J}}$ more clearly, we recall that $\mathcal{7}(p) \rightarrow \mathcal{G} \rightarrow \mathcal{7}(p)$ interactions must exist, whereas we have considered only $\mathcal{7}(p) \rightarrow \mathcal{G} \rightarrow \mathcal{7}(q)$ interactions to the present. It is easy to overcome this deficiency in three steps: (1) Let parallel fibers terminate beneath every $\mathcal{7}(p)$ parallelopiped rather than merely at $\mathcal{7}(\oplus_{j,k} \bar{q}_j^k)$. Similarly, let transverse distributions of $\mathcal{O}$, $\mathcal{LI}$, $\bar{\mathcal{J}}$, $\mathcal{J}$, and r_i cells lie beneath every $\mathcal{7}(p)$ parallelopiped. How can the distribution of these new cells be determined? The simplest way is as follows. Shift the $\mathcal{O}$, $\mathcal{LI}$, $\bar{\mathcal{J}}$, and $\mathcal{J}$ distributions from $\mathcal{7}(\oplus_{j,k} \bar{q}_j^k)$ along the parallel fibers, and let new $\mathcal{O}$, $\mathcal{LI}$, $\bar{\mathcal{J}}$, and $\mathcal{J}$ cells be created at the shifted locus of the old cells. A new locus is determined under every p_i parallelopiped. These new $\mathcal{O}$, $\mathcal{LI}$, $\bar{\mathcal{J}}$ and $\mathcal{J}$ distributions therefore have stationary cross-sections. What about the distribution of new parallel fibers and r_i cells? Here again, imagine that all the parallel fibers terminating at $\mathcal{7}(\oplus_{j,k} \bar{q}_j^k)$ and their r_i sources are shifted longitudinally away from $\mathcal{7}(\oplus_{j,k} \bar{q}_j^k)$

for a distance of approximately d_1. Create a new set of parallel fibers $\mathcal{L}^1$ where the shifted fibers lie and a set R^1 of new r_i points. Now shift the parallel fibers and r_i cells longitudinally for approximately another d_1 units and create a new set $\mathcal{L}^2$ of parallel fibers and a new set R^2 of source points for them. Continue this process until $\mathcal{G}$ is covered by $\mathcal{L}^i$ and R^i sets. By this shifting process, we also ensure that all R^i and $\mathcal{L}^i$ sets have stationary cross-sections. We now have the following transmission flow diagram:



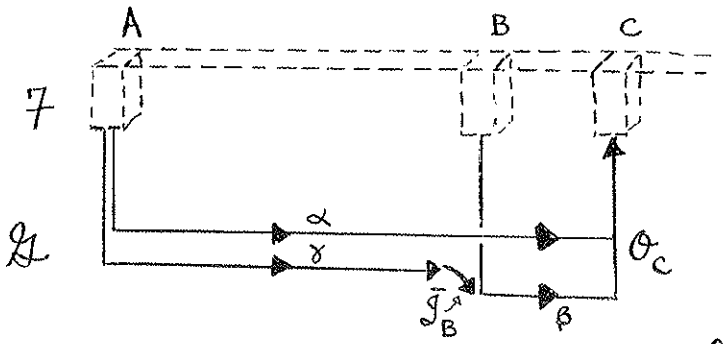
(2) The previous diagram reveals an imperfection: All $7(p) \rightarrow \mathcal{G} \rightarrow 7(p)$ interactions are polarized in the $7(q)$ direction. We can remove this imperfection quite easily. Simple let each cell in $\oplus_i R^i$ send out axons which split into two opposite longitudinal directions before coursing along as parallel fibers.



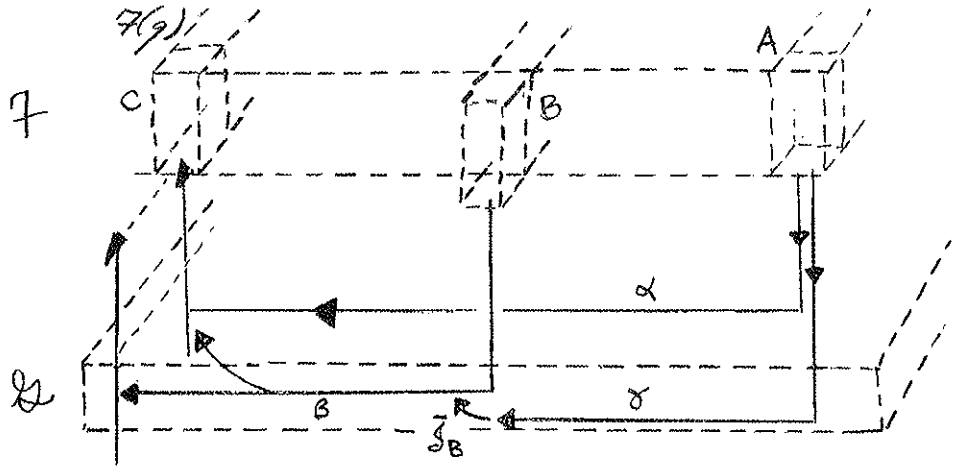
The distribution of the α_1 fibers is thus the same as the distribution of the α_2 fibers, and the $7(q)$ polarity is immediately removed.

150. Recircuitry

(3) Before we constructed the $\oplus_i R^i$ and $\oplus_i \mathcal{L}^i$, the $\mathcal{J}$ and $\bar{\mathcal{J}}$ cells could only bind input cells to r_i cells, or r_i cells to output cells. Now it is also possible for new connections among different R^i cells to be established. For example, consider the following diagram:



If A, then C, are excited, only the α terminals on $\mathcal{O}_C$ will be markedly facilitated. If A, then B, then C are excited, both α and β terminals on $\mathcal{O}_C$ will be facilitated. Moreover, transmission over γ will come to exert a stronger influence on transmission over β because of the facilitation of an $\mathcal{J}_B$ link joining γ to β. If A is excited for a second time T time units after the A-C cycle, the output over $\mathcal{O}_C$ will be larger than it was the first time A was excited. But if A is excited for a second time T time units after the A-B-C cycle, and A,C are separated by the same time interval in both the A-C and A-B-C cycles, then $\mathcal{O}_C$ transmission will be larger still, because of the additional contribution of the newly incorporated $\gamma - \mathcal{J}_B - \beta$ circuit to the intensity of $\mathcal{O}_C$ transmission. Thus, exciting A alone, after A-B-C, generates an excitation at C that is similar to the excitation which C received from both A and B on the first trial. The new circuits tend to <u>reproduce</u> the old input arrangement, modulo C. A similar remark holds for diagrams like



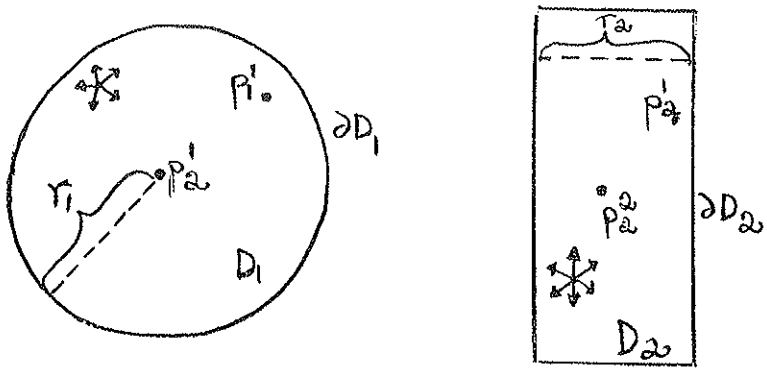
This linking of $\oplus_i R^i$ cells by $\mathcal{J}$ cells is topographically very specific and sensitive. By extrapolating from the A-B-C example, one can see quite clearly that specific <u>distributions</u> of input excitation to $\mathcal{F}(p)$ can be made to excite specific $\mathcal{F}(q)$ points with a well-determined spatio-temporal distribution, via $\mathcal{G}$. Moreover, we see that small collections of A points which are far from $\mathcal{F}(q)$ can come to control excitation distributions to $\mathcal{F}(q)$ which were originally controlled by many B and C points. When several sets of parallel lines , each

with different transmission time lags, occur, this sensitivity is still further increased. Such variable sets are introduced, for example, when various sets of comparable sets of horizontal fibers in P_1 exist. We shall discuss embedding problems of this type in a later work.

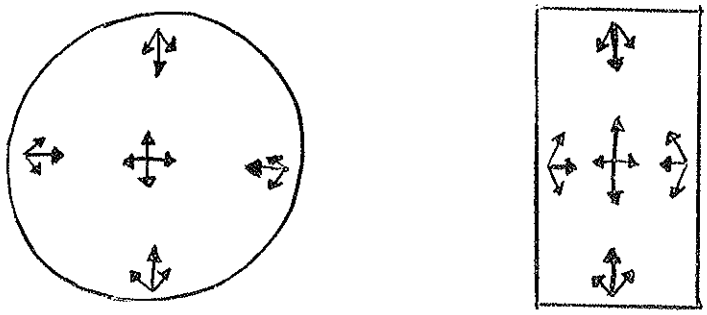
Transverse $\mathcal{J}$ cells can replace $\overline{\mathcal{J}}$ in the last diagram only when the length of α is roughly twice the length of β, but by changing the orientation of the $\mathcal{J}$ cells, we can build $\mathcal{J}$ bridges between γ's and β's whose lengths vary considerably.

151. Boundary Behavior

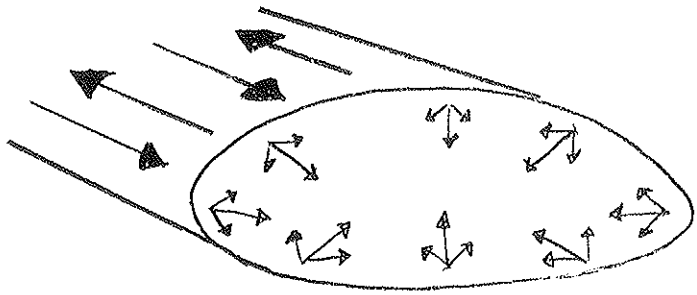
It has been supposed throughout that all of the various cell distributions exhibit some sort of symmetry property, say transverse symmetry. We have not, however, considered what happens to these cell distributions when the boundary of a collection of cells is reached. For example, consider the diagrams



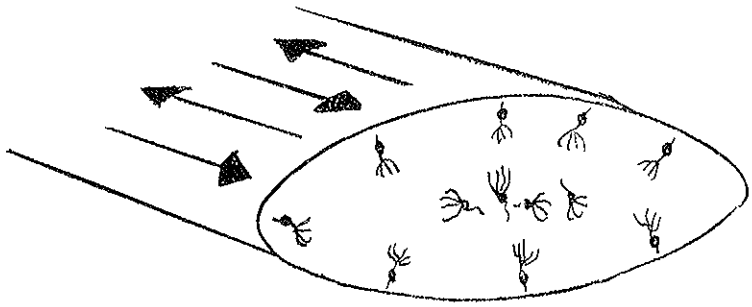
D_1 and D_2 are two planar regions with boundaries ∂D_1 and ∂D_2, respectively. Suppose that points are distributed uniformly in both regions. In both pictures, p_2^i is further from the boundary than p_1^i is, and the lengths of all the lines emitted by any p_j^i are much smaller than the field dimensions r_i, i=1,2. Suppose that all points emit lines with radially symmetric distributions. Then the total density of lines reaching p_1^i is strictly smaller than the total density of lines reaching p_2^i. Consequently, if we subject each of D_1 and D_2 to a randomly distributed set of inputs over a long time interval, the most highly facilitated lines will point away from the boundary:



Random inputs thus introduce an immediate bias into the line structure of boundary points. Since we have only a finite number of points, we are often confronted with a tendency of this kind, even when the boundary itself has no useful significance for the dynamics of the cells. In particular, when we are given a series of layers of parallel fibers, some fibers must lie at the periphery.



But these fibers will only be hindered in their task of accurately reflecting $\mathcal{F}(p) \to \mathfrak{L} \to \mathcal{F}(p \oplus q)$ interactions unless the boundary bias is overcome. We want to pack the parallel fibers as densely as possible, for increasing the number of fibers in a sensible fashion always sensitizes the interaction pattern. The distribution of $\oplus_i R^i$ cells thus cannot be expected to solve the boundary difficulty. We obviously need to postulate the existence of a cell that has locally distributed dendrites and which can be placed with a fixed orientation relative to the boundary:



When these cells occur near the boundary, their dendrites must be oriented away from the boundary. These dendrites collect excitation from the interior fibers and transfer it to the periphery, where it is needed to offset the unpleasant boundary bias. Deep within the interior of the parallel fibers, these cells should be oriented in random transverse directions to maintain the unbiased

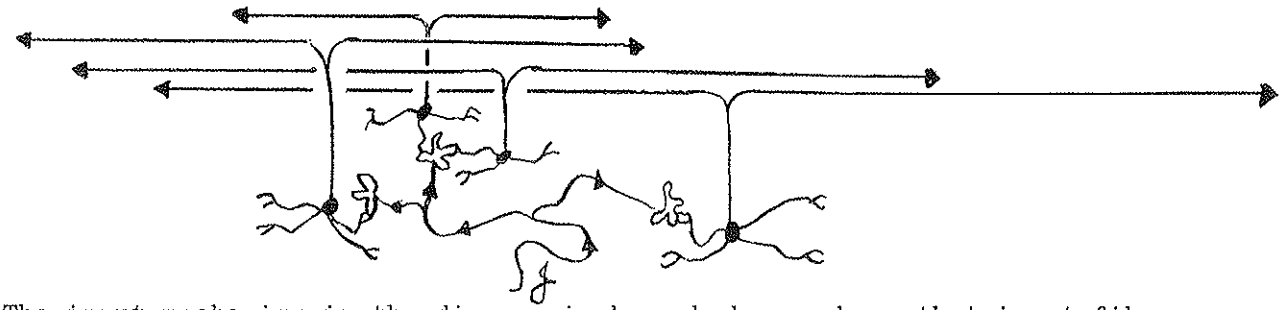
interaction pattern of the lines here. We already know of a cell of the proper construction: the $\mathcal{J}$ and $\bar{\mathcal{J}}$ cells. Thus, as part of their task of binding together parallel fibers that are derived from overlapping p_i cells, and joining parallel fiber endings to $\mathcal{O}$ cell dendrites, these cells also ensure that these fibers respond to the p_i excitations free from inessential local geometrical biases, such as those introduced by the boundaries of cellular collections. The introduction of specifically oriented $\mathcal{J}$ cells is based on considerations that are qualitatively similar to those leading to the introduction of parallel folia. In both cases, some inhomogeneous geometrical deformation within Euclidean space is required to ensure a homogeneity condition in the functional geometry of the cells.

152. Input Fibers to $\mathcal{G}$: $\mathcal{J}$ Cells

How do the input fibers--denoted by $\mathcal{J}$ --terminate in $\mathcal{G}$? They must terminate on the $\oplus_i R^i$ cells, so that the parallel fibers can be activated, and their distribution should be compatible with the partition of $\mathcal{G}$ cells into parallelopipeds. The parallel fibers which the inputs excite terminate on $\mathcal{O}$ dendrites that collect excitation in a nonvolumetric way. In order that the system: input fibers → parallel fibers → output fibers preserve this nonvolumetric dynamical feature, we expect each input fiber to break up into a collection of winding terminal fibers that distribute themselves to an appropriate region of juxtaposed parallelopipeds. These fibers must be capable of making contact with the large number of $\oplus_i R^i$ cells in this region. One way to do this is to branch the terminal fibers whenever a $\oplus_i R^i$ dendrite is encountered. Numerous branchings of the input axons will be created in this way. Each successive axonal branch is usually thinner than the last, and is unable to transmit the excitation from the last branch with complete efficiency. Thus, profusely branched axons will not distribute the full intensity of the input to the $\oplus_i R^i$ dendrites, and the total area of the axon-$\oplus_i R^i$ dendrite connection decreases as the degree of the branching increases. Since both the input intensity and the effective surface area of each connection decrease with increased branching, a profusely branched input distribution mechanism is inefficient. We need a way to correct these two deficiencies without denying any cells in the $\oplus_i R^i$ parallelopipeds their share of inputs.

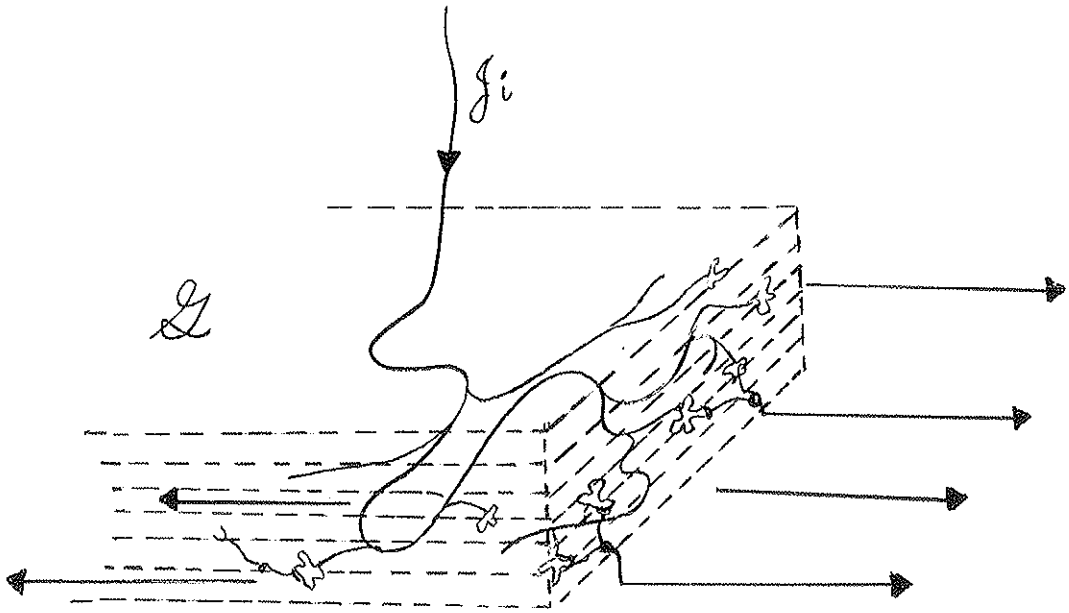
153. Rosettes

An obvious corrective measure is the following:



The input mechanism in the diagram is branched enough so that input fibers reach the general region of all appropriate $\oplus_i R^i$ cells. Instead of ending in profuse branches, however, each axon terminates on an extended cellular region which has a sufficiently large volume that it can accomodate several $\oplus_i R^i$ dendrites. Such cellular regions are called rosettes. The input to a rosette is distributed volumetrically throughout its effective surface area. Every $\tau \in \oplus_i R^i$ terminating on one rosette therefore receives approximately the same excitation density, which is not damped out by the existence of many terminal branchings and which is delivered to a large effective surface area. Rosettes permit the distribution of an input to large numbers of $\oplus_i R^i$ dendrites without serious decrement, and the collection of rosettes is dynamically split in much the same way as the segments of a dendritic cross-section. The use of rosettes brings about a large degree of convergence and divergence in the set of $\mathcal{J} \longrightarrow \oplus_i R^i$ connections. The $\mathcal{J}$ input functions of the $\oplus_i R^i$ embedding equations therefore have the form of space-time averages, and nearby $\oplus_i R^i$ cells have similar, but slightly shifted, $\mathcal{J}$ input terms. These shifts in space-time input averages are directly reflected in transverse shifts in the distribution of overlapping $\oplus_i \mathcal{L}^i$ inputs to the $\mathcal{O}$ cells.

On what parallelopipeds does a $\mathcal{J}$ fiber terminate? Since we constructed $\oplus_i R^i$ with stationary cross-sections, we can associate with every p_i a subset $\mathcal{R}_i \subset \oplus_i R^i$ lying vertically below p_i whose transverse cellular distribution is independent of i. $\mathcal{R}_i$ consists of cells from all $\mathcal{L}(k)$ layers. Letting $\mathcal{J}_i$ be an input from p_i to $\mathcal{R}_i$, the simplest assumption is that $\mathcal{J}_i$ distributes itself throughout $\mathcal{R}_i$, for the same reasons that guided us to let a single $\mathcal{O}$ cell's dendrites reach all $\mathcal{L}(k)$ layers. Thus, when p_i transmits an intensive $\mathcal{J}_i$ to $\mathcal{Y}$, practically all of $\mathcal{R}_i$ is excited, and excitations are emitted throughout the subset $\mathcal{L}_i$ of $\oplus_i \mathcal{L}^i$ which $\mathcal{R}_i$ controls:



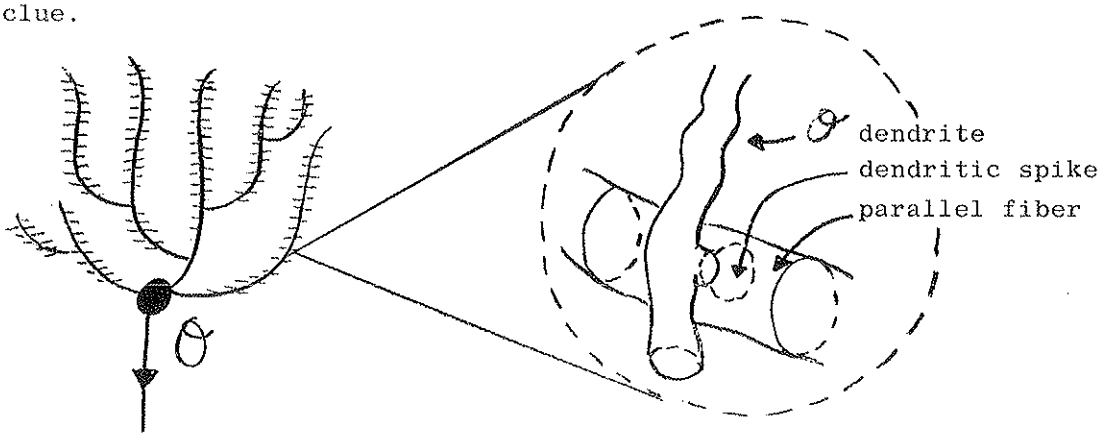
The total input to each parallel fiber as a functional of the $\mathcal{J}$ input can be computed if we know the distribution of input latencies and input fiber densities which reach every rosette on which an $\oplus_i R^i$ cell's dendrites terminate. The relation of a rosette's effective surface area to its volume must also be known. All of these quantities are purely geometrical. The total input to an $\oplus_i R^i$ cell if found by adding the easily computedcontributions of the individual dendrites, with the appropriate transmission time lags.

154. Dendritic Spikes

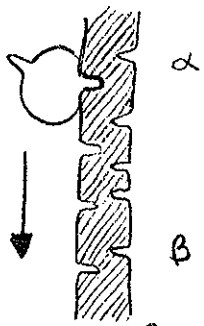
We want the total input from $\mathcal{R}_i$ to a longitudinally displaced $\mathcal{O}$ cell to vary inversely with the $\mathcal{O}$ cell's distance from $\mathcal{R}_i$. Otherwise, the radial distribution of $p_i \longrightarrow p_j$ connections, which decreases with increasing distance, will be violated. How can this condition on $\mathcal{L}_i \longrightarrow \mathcal{O}$ transmissions be fulfilled? One way is to let long fibers be less numerous than short fibers and to require that only the terminals of $\mathcal{L}_i$ axons make contact with an $\mathcal{O}$ cell, directly or via $\mathcal{J}$ cells. This is not the best way, however, for it wastes a great deal of effective surface area and possible binding between adjacent $\mathcal{L} \rightarrow \mathcal{O}$ systems. It permits extensive collections of $\mathcal{L}_i$ fibers to pass through many $\mathcal{O}$ dendrites without coming into functional contact with them. This wastefulness can be easily corrected: Connect the $\oplus_i \mathcal{L}^i$ fibers to all the $\mathcal{O}$ dendrites which they touch; envisage connections to $\mathcal{O}$ cells along the entire length of a given parallel fiber. If we do this, nearby $\mathcal{O}$ cells will communicate with more $\mathcal{L}_i$ fibers from a given $\oplus_i R^i$ parallelopiped than will distal $\mathcal{O}$ cells,

irrespective of the distribution of fiber lengths.

We are confronted by a difficulty in envisaging this possibility, however: The contact between parallel fiber and $\mathcal{O}$-dendrite has a very small effective surface area. How can we enlarge this area without disrupting the transmission of the fiber to more distal $\mathcal{O}$ dendrites? The rosettes provide the clue.



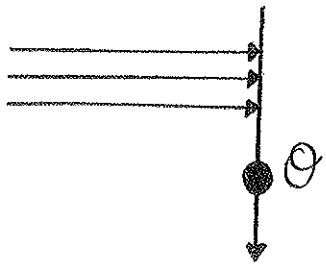
We let small protuberances grow with regular spacing on the $\mathcal{O}$ dendrites. Each such protuberance, or dendritic spike, invaginates a nearby parallel fiber. This spike obviously increases the effective surface area of each parallel fiber→$\mathcal{O}$-dendrite connection many-fold. Why do we not, instead, let spikes grow from the parallel fiber into the $\mathcal{O}$-dendrite? At least three reasons suggest themselves: (1) A spike lying within the parallel fiber shall be subjected to a more intense excitation when an input wave passes through the fiber than a spike growing from the parallel fiber into the $\mathcal{O}$-dendrite. Since the total breadth of both parallel fiber and dendrite is small, this efficiency is very important. (2) If spikes protruded into a dendrite, we would have a diagram like



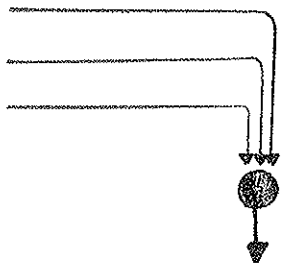
Cross-Section of an $\mathcal{O}$-Dendritic Branch

for the $\mathcal{O}$-dendrites. Excitation flowing down the dendrite from α will be impeded by the morass of β invaginations. Moreover, (3) the dynamical state of the β embedding spaces will be biased by prior α activation, and vice versa. Such biases are incompatible with transverse additivity and the desire

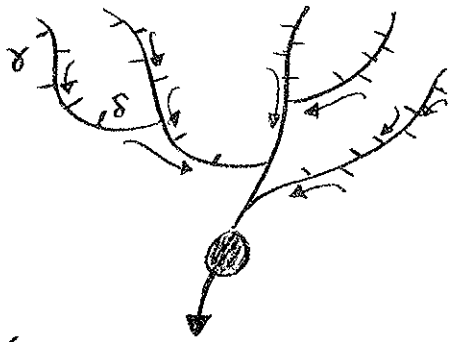
to realize a maximal independence of local dendritic activation. Indeed, when each local collection of spikes operates independently of all other collections, the diagram



can be replaced by



The spikes in the parallel fibers help to realize this latter situation by conducing to the following excitation flow diagram:

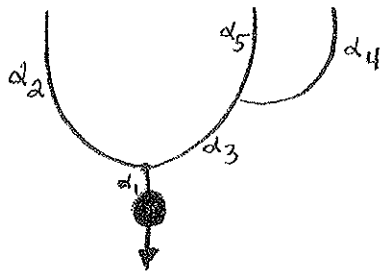


The flow down the γ dendrite will tend to bypass the protruding δ spike.

155. Dendritic Functionals of Input Functions

The simplest possible dendritic interactions arise when all spikes are independent of their adjacent dendrites and every dendrite passes on the excitation received from the spikes with a density that decreases decrementally as a simple function of distance traversed. For example, if each dendritic branch has a uniform breadth throughout its length, and impulses propagate electrotonically down the dendrites to B, we have the following situation. Let every

setting of the spatial variable w denote a fixed embedding space on some dendritic spike. To every w and time t, we can associate a point strength function s(w,t). By letting w stand for the entire embedding mechanism within a spike, we have coarse-grained our discussion from the very start. All further remarks depend on this coarse-graining for their validity. We suppose that s(w,t) is propagated at a linear rate down the dendrites and that it decays monotonely with increasing distance traversed. The rate of propagation and decay depend on geometrical properties of the dendrite considered; for example, its diameter. Given the dendritic tree



with branches α_i, i=1,2,3,4,5, we suppose that

τ_i = the velocity of s propagation through α_i,

$D(\lambda_{i1},\ldots,\lambda_{in};d)$ = the decrement in s after it is transmitted d units along α_i,

$L_i(w)$ = the distance of $w \in \alpha_i$ from the first dendritic intersection which is reached when flowing from w along α_i to B,

L_i = the length of α_i.

In this situation, the total input contribution from α_4 to B is

$$I(4,B) = \sum_{w \in \alpha_4} s(w, t - \textstyle\sum_{i=1}^{3} \tau_i L_i - \tau_4 L_4(w)) D(\lambda_{41},\ldots,\lambda_{4n};L_4(w)) \cdot$$

$$\cdot D(\lambda_{31},\ldots,\lambda_{3n};L_3) D(\lambda_{11},\ldots,\lambda_{1n};L_1).$$

The contribution from every α_i is of the same form, and the total contribution to B is the sum of the contributions of the individual α_i. For example, when the electrotonic decay is exponential,

$$D(\lambda_{i1},\ldots,\lambda_{in};d) = \lambda_{i1} e^{-\lambda_{i2} d}.$$

If the dendritic spikes are also uniformly spaced along α_4 with mean spacing h, we have

$$I(4,B) = \sum_k s(kh, t - \sum_{i=1}^{3} {}_iL_i - kh\tau_4)\lambda_{11}\lambda_{31}\lambda_{41} e^{-(\lambda_{12}L_1 + \lambda_{31}L_3 + kh\lambda_{41})}.$$

Thus, we find an induced $\mathcal{O}$-dendritic input to B of the form

$$\sum_{w \in \bigcup_i \alpha_i} s(w, t - T(w))p(w)$$

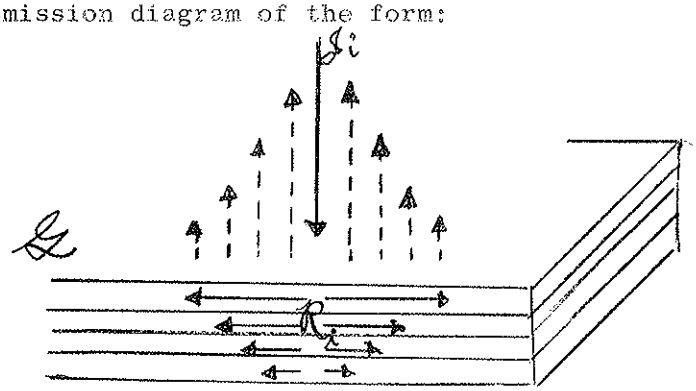
where $p(w)$ and $T(w)$ depend in a definite way on the dendritic geometry. We are already quite familiar with inputs of this type.

If the input mechanism is uniformly distributed to all $w \in \bigcup_i \alpha_i$, then the first w in $(w, t-T(w))$ can be dropped in $s(w, t-T(w))$, and we find the mapping

$$\Omega_{\mathcal{J}B} : I \longrightarrow \sum_{w \in \bigcup_i \alpha_i} s_I(\cdot - T(w))p(w)$$

where s_I is the s function determined at each w by the input function I. $\Omega_{\mathcal{J}B}$ is a functional mapping input functions to the dendritic spikes into input functions to B. In this special case, $\Omega_{\mathcal{J}B}$ is almost a linear functional: it is a linear functional of the locally determined embedding function s_I, and all global features of the dendritic bush are collapsed into the geometrical terms $T(w)$ and $p(w)$. When I is not uniformly distributed, $\Omega_{\mathcal{J}B}$ must be extended to take into account the axonal transmission to the rosettes, the volumetrical dispersion of excitation in each rosette, the transmission of excitation into the $\oplus_i R^i$ dendrites and thence to the $\oplus_i R^i$ cell bodies, followed by $\oplus_i \mathcal{A}^i$ transmission and finally by excitation of the $\mathcal{O}$-dendritic spikes. When all of these preliminary steps are also accounted for, $\Omega_{\mathcal{J}B}$ is a functional mapping $\mathcal{J}$ inputs into B inputs, and this functional is not, in general, linear even in the weak sense described above. Various special cases will be considered at a later time.

The existence of dendritic spikes implies that, under a fixed $\mathcal{J}_i$ input, we have a transmission diagram of the form:



The lengths of the dotted lines give the relative size of the $\mathcal{O}$ outputs determined by $\mathcal{J}_i$ when any outputs occur. The diminution of output with longitudinal distance from $\mathcal{J}_i$ depends entirely on geometrical factors of the type depicted above. When a complete set of antagonists is uniformly excited by a set of $\mathcal{J}$ inputs, it is possible than no $\mathcal{O}$ outputs will occur. Nonetheless, a gradient of $\mathcal{O}$ cell excitation will be established that decreases with distance, as depicted in the diagram. Even when the inhibition on the cell body of $\mathcal{O}$ is so great that no transmission occurs, facilitation of the c functions of the $\mathcal{L}_i \rightarrow \mathcal{O}$-dendritic spike connections will occur whenever inputs pass through them. The formation of such new connections within dendrites unaccompanied by actual output transmission underlies many examples in which new learning occurs without actual behavioral output. If, however, the individual spikes on a given $\mathcal{O}$-dendrite do act completely independently of one another, then letting several inputs from different $\oplus_i R^i$ sources converge on this dendrite simultaneously has no more direct facilitative effect on these connections than letting just the one input which excites the connections in question occur. Several indirect routes for registering the differential facilitation of cumulative inputs do exist; for example,

$$\oplus_i \mathcal{L}^i \longrightarrow \mathcal{J} \longrightarrow \mathcal{O}\text{-dendrite},$$

$$\oplus_i \mathcal{L}^i \longrightarrow \bar{\mathcal{J}} \longrightarrow \oplus_i R^i \longrightarrow \oplus_i \mathcal{L}^i \longrightarrow \mathcal{O}\text{-dendrite}.$$

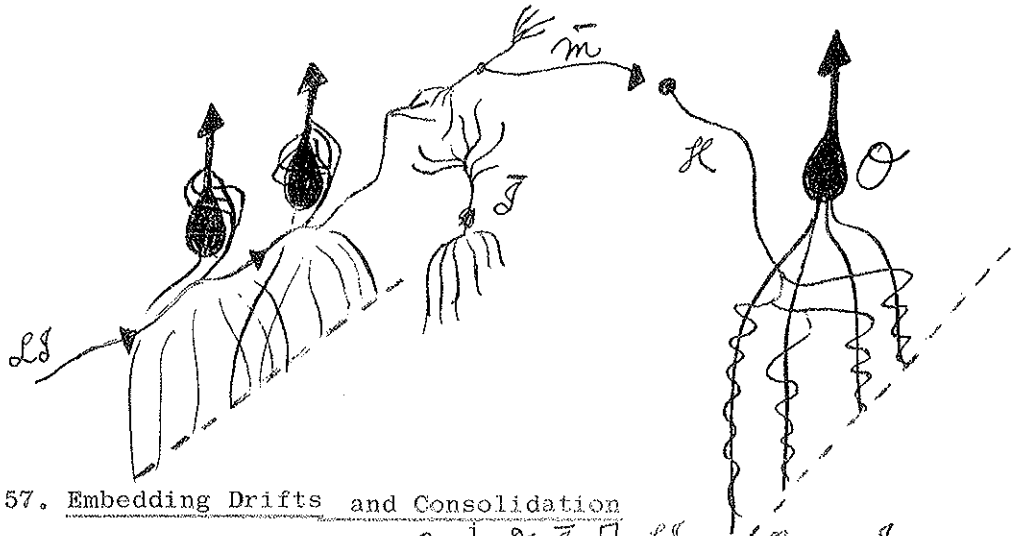
But these routes are only activated by the endings of $\oplus_i \mathcal{L}^i$ lines, unless dendritic spikes from the $\bar{\mathcal{J}}$ and $\mathcal{J}$ cells are also allowed to grow into the parallel fibers. Thus, when the $\mathcal{O}$-spikes do operate independently, even though an intense input to $\mathcal{J}_i$ always excites nearby $\mathcal{O}$ cell dendritic spikes, important new fiber circuits are formed primarily over fiber routes connecting parallel fiber terminals to their impinging cells. When the spikes do not operate with complete independence, we expect closely juxtaposed spikes to bias one another's activity, and antidromic inputs to a dendrite to bias all of its spikes. These spike-dendritic dependencies will decay at least as fast as $D(\lambda_{i1},\ldots,\lambda_{in};d)$, where d is the distance between source point and recipient spike. Further details will be provided in a later section.

This discussion of the local action of $\mathcal{O}$ dendrites is in substantial contrast with the fact that when muscle groups participate in motor acts which involve many automatic associated movements, the stability of these associations

can be assured by distributing $\mathcal{J}$ fibers across several folia. Such a distribution of $\mathcal{J}$ fibers is directly analogous to the distribution of closely juxtaposed input-output arrangements, substantially free from stellate intervention, found along the central fissure.

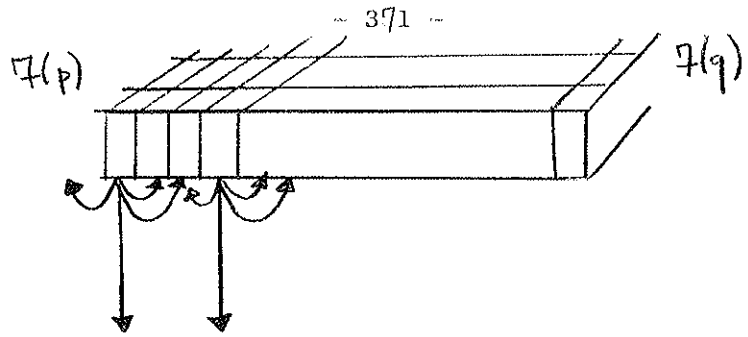
156. Subliminal Fibers for $\mathcal{G}$: $\mathcal{H}$ Cells and $\overline{\mathcal{M}}$ Cells

It is natural, in comparing $\mathcal{7}$ with $\mathcal{G}$, to ask whether an analog of a horizontal fiber exists in $\mathcal{G}$? Such a fiber--called an $\mathcal{H}$ fiber--must influence $\mathcal{O}$ through its dendrites. Parallel layers do not exist in $\mathcal{G}$, however, so that the separation of horizontal fibers and pyramid cells in $\mathcal{7}$ will be collapsed into an intimate union between $\mathcal{H}$ and $\mathcal{O}$-dendrites in $\mathcal{G}$. A natural way to establish such a union is to let an $\mathcal{H}$ fiber climb through an entire dendritic tree, wrapping itself about the $\mathcal{O}$-dendrites at every stage of the climbing process. We also need a cell--denoted by $\overline{\mathcal{M}}$--to join $\mathcal{J}$ cells to the $\mathcal{H}$ cells, just as the cells of Martinotti in $\mathcal{7}$ joined input lines to the pyramidal dendrites. As in $\mathcal{7}$, a convenient place for these $\overline{\mathcal{M}}$ cells to lie is in the layer where the $\mathcal{J}$ cells terminate. The $\overline{\mathcal{M}}$ cells will send out transverse dendrites and longitudinal axons in the layer of the $\mathcal{J}$ cells, for the usual reasons. Such an $\overline{\mathcal{M}}$-$\mathcal{H}$ fiber system subliminally excites the transversely oriented dendritic output system in $\mathcal{G}$ just as the $\mathcal{M}$-horizontal fiber system subliminally excites the radially oriented, pyramid dendritic output system in $\mathcal{7}$. When $\mathcal{J}$ cells distribute themselves across folia, the $\mathcal{J}$-$\overline{\mathcal{M}}$-$\mathcal{H}$ coupling is responsive to radially symmetric columns in $\mathcal{7}$. The reader can easily envisage particular fiber distributions for himself, all of which conform to this general pattern. The $\overline{\mathcal{M}}$ dendrites must be responsive to $\mathcal{LS}$ cells to prevent $\mathcal{O}$-dendritic excitations via $\overline{\mathcal{M}}$ cells from being generated in a region of $\mathcal{O}$ cell body inhibition. Thus, several $\mathcal{LS}$ axons must reach above the $\mathcal{O}$ cell bodies to the $\overline{\mathcal{M}}$ dendrites. The $\overline{\mathcal{M}}$ dendrites also make contact with $\mathcal{J}$ axons in the $\mathcal{J}$ cell region for similar reasons, and the $\overline{\mathcal{M}}$ axons make contact with the $\mathcal{H}$ cells. An analogy with the P_1 disk diagrams can easily be drawn for the $\overline{\mathcal{M}}$-$\mathcal{H}$ complex of $\mathcal{G}$.

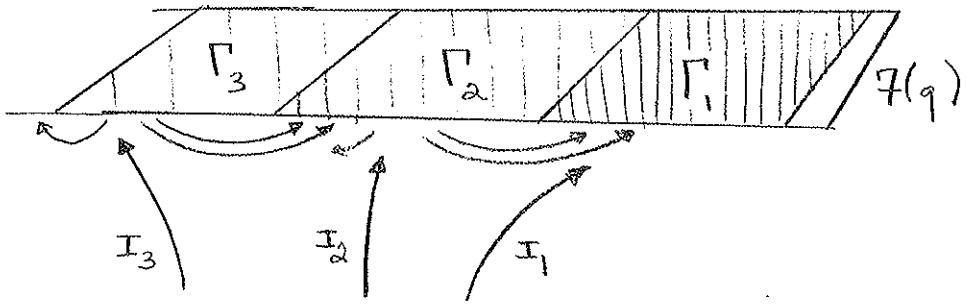


157. Embedding Drifts and Consolidation

This collection of $\oplus_i R^i$, $\mathcal{O}$, $\mathcal{I}$, $\mathcal{J}$, $\mathcal{L}\mathcal{S}$, $\bar{m}$, $\mathcal{K}$, and $\mathcal{J}$ cells is capable of rapid, massive, and very precise interactions over widely dispersed cell groups. We have already seen some examples of $\oplus_i \mathcal{L}^i \rightarrow \oplus_i R^i$ recircuitry in this system. It is easy to understand the qualitative features of local recircuitry between the other cell types from our remarks motivating their construction, particularly since all of these various local circuits are analogous to circuits in $\mathcal{7}$. The $\mathcal{G}$ which we have constructed satisfies our original aim of bringing widely separated points in $\mathcal{7}$ into intimate functional contact, and the range of this contact is increased many-fold by the possibility of establishing new $\oplus_i \mathcal{L}^i \rightarrow \oplus_i \mathcal{L}^i$ circuits. Nonetheless, $\mathcal{O}$ cells that lie very far from a particular $\mathcal{J}$ cell will not as readily enter into a strong $\mathcal{J} \rightarrow \mathcal{O}$ circuit as a nearby $\mathcal{O}$ cell. The distal $\mathcal{O}$ cell must depend on the longest $\oplus_i \mathcal{L}^i$ fibers and on multiple $\oplus_i \mathcal{L}^i$ circuits, whereas the nearby $\mathcal{O}$ cell can also form new connections which utilize parallel fibers of many different lengths. This restriction to $\mathcal{O}$ cells that can be reached in a small number of transmission steps is the counterpart of the expectation that embedded control forms in $\mathcal{7}$ which depend on the growth of new $\mathcal{J}$ chains will first be formed from p_i points near $\mathcal{7}(q)$, and will frequently include more distal p_i only after the shorter $\mathcal{7}(p) \rightarrow \mathcal{7}(q)$ chains are well-developed. We can transform this tendency into an efficient embedding mechanism in $\mathcal{7}$, without disrupting the long-range interactions within $\mathcal{G}$. Consider



Here we have included $\mathcal{7}(p) \longrightarrow \mathcal{7}(p)$ looped lines which are oriented primarily from $\mathcal{7}(p)$ towards $\mathcal{7}(q)$, although less profuse loops are allowed in the opposite direction. These loops accentuate a drift in the embedding dynamics towards $\mathcal{7}(q)$. Consider the diagram



In this diagram, at some fixed time, Γ_1 contains more $\mathcal{7}(q)$ control forms than Γ_2 does, and Γ_3 even fewer than Γ_2. Let input distributions I_i be delivered to Γ_i such that I_1 excites a set $\{\xi_j\}$ of Γ_1 control forms. Also let excitation waves from Γ_2 and Γ_3 reach $\{\xi_j\}$ while $\{\xi_j\}$ is still excited by I_1. New line connections from Γ_2 to $\{\xi_j\}$ will form more readily than connections from Γ_3 to $\{\xi_j\}$, and these connections from Γ_2 can be established with several $\{\xi_j\}$ simultaneously. Moreover, connections from Γ_3 to Γ_2 will be stronger than $\Gamma_3 \longrightarrow \{\xi_j\}$ connections. Partial Γ_3 control of $\{\xi_j\}$ does exist from the start, but is not as strong as Γ_2 control. Corresponding recircuiting will occur in $\mathcal{Q}$.

A later occurrence of I_2 will therefore indirectly activate many of the ξ_j control forms which I_1 directly activates. By the time the Γ_2 embeddings become better established, inputs to Γ_3 will have gradually come to exert increasing control over Γ_2, with the advantage that inputs to Γ_3 are capable of activating embeddings which regulate a still larger number of $\mathcal{7}(q)$ control forms than Γ_2 could control. By continuing in this fashion, we can construct Γ_i which control $\mathcal{7}(q)$ muscle sequences of the greatest intricacy. $\mathcal{Q}$ is of the greatest importance in binding these later control forms to the earlier forms in a cohesive manner, making full use of its excellent recircuiting equipment.

In order to ensure that this escalation of the complexity of $\mathcal{H}(q)$ control forms occurs automatically, we must imagine that inputs to the entire broad region $\oplus_i \Pi_i$ occur throughout the learning process. To see qualitatively how this input mechanism works, suppose that a Π_i band like $\Pi_i^{(-k)} = \cup\{\Pi_i, \Pi_{i-1}, \ldots, \Pi_{i-k}\}$ has become a well-structured $\mathcal{H}(q)$ control region. Inputs to $\Pi_{i+1}^{(m-1)} = \cup\{\Pi_{i+1}, \Pi_{i+2}, \ldots, \Pi_{i+m}\}$ have been occurring all along, but during the period when $\Pi_i^{(-k)}$ was quite homogeneous, behaviorally significant $\Pi_{i+1}^{(m-1)} \rightarrow \Pi_i^{(-k)}$ connections could not form, since $\Pi_i^{(-k)}$ was incapable of transmitting organized excitations from $\Pi_{i+1}^{(m-1)}$ to $\mathcal{H}(q)$. Once $\Pi_i^{(-k)}$ begins to exert significant control over $\mathcal{H}(q)$, the situation changes dramatically, though gradually. For the inputs to $\Pi_{i+1}^{(m-1)}$, which earlier were behaviorally quite ineffective, now induce $\Pi_{i+1}^{(m-1)} \rightarrow \Pi_i^{(-k)}$ connections between the lines excited by the input and the $\Pi_i^{(-k)}$ control forms that are active when the lines are. These connections have their repository in the c functions of the strongly coupled excitatory lines. When such an input distribution is delivered again to $\Pi_{i+1}^{(m-1)}$ at a later time, it excites the $\Pi_i^{(-k)}$ control forms selectively over precisely the facilitated $\Pi_{i+1}^{(m-1)} \rightarrow \Pi_i^{(-k)}$ lines. The $\Pi_i^{(-k)}$ control forms, in turn, propagate excitatory distributions towards $\mathcal{H}(q)$, until the appropriate $\mathcal{H}(q)$ muscle sequences occur. An input to $\Pi_i^{(-k)}$ which was able to set off $\Pi_i^{(-k)}$ control forms before $\Pi_{i+1}^{(m-1)}$ actively entered the picture will still be able to do so even after $\Pi_{i+1}^{(m-1)} \rightarrow \Pi_i^{(-k)}$ connections form. New control, of ever higher type, is achieved without losing any of the old, and more pristine, control mechanisms! It should be clear that this partition into Π_i bands is merely a conceptual convenience to emphasize that the centroid of the most intensive line formation shifts away from $\mathcal{H}(q)$ with increasing input experience. Another way of putting this is that control forms consolidate as close to the $\mathcal{H}(q)$ border as possible before new control forms are established. Broad regions of $\mathcal{H}(p)$ can participate in new embeddings at any one time, but not all regions will be equally effective. The breadth of these regions and their

embedded control forms is an underpinning of the so-called Laws of Mass Action and Equipotentiality, but such Laws cannot be fully understood without also considering the projections of $\mathcal{F}$ to renormalizing fields such as $\hat{\mathcal{F}}$. Inputs to the Γ_i can be delivered either from direct sensory-motor systems or from higher correlational fields. The inputs to Γ_i for i large are especially well-suited for the embedding of $\mathcal{F}(q)$ controls of the most intricate kind, and we therefore expect the highest correlational fields to project to these Γ_i more intensively than to the Γ_i bordering on $\mathcal{F}(q)$.

158. Spontaneous Hierarchical Inversion. Hull and Guthrie Revisited

Notice that before specific $\Gamma_{i+1}^{(m-1)} \rightarrow \Gamma_i^{(-k)}$ connections occur, $\Gamma_{i+1}^{(m-1)} \rightarrow \Gamma_i^{(-k)}$ transmissions will tend to be uniformly scattered by the undifferentiated sets of $\mathcal{J}$-chains. Since these chains exert the greatest influence on pyramid cell output, the complete sets of antagonists in $\mathcal{F}$ will tend to inhibit pyramidal output during this period. The pyramidal output from such a $\Gamma_i^{(-k)}$ region becomes increasingly intense as the $\Gamma_{i+1}^{(m-1)} \rightarrow \Gamma_i^{(-k)}$ $\mathcal{J}$-chains become more highly differentiated. This mechanism spares subcortical nuclei from the flood of behaviorally irrelevant $\mathcal{F}$ transmission that would otherwise occur in response to the inputs delivered from subcortical regions to undifferentiated $\mathcal{F}$ regions in preparation for new control form embeddings.

The automatic embedding within successive Γ_i regions thus depends on a process of inversion whereby new, ever more delicate control systems continually achieve mastery over old systems that preserve their integrity under the inversion process. We call this process spontaneous hierarchical inversion, or simply spontaneous inversion. We say that this inversion process is "spontaneous" because

the geometrical and input substructures which make it possible are available long before new control mechanisms "spontaneously" arise within the field. Another descriptive term for this all-important process is automatic (hierarchical) inversion. Spontaneous inversion is indispensable to the emergence of conscious behavior. More generally, an exacting study of the class of spontaneous inversions possible within any given set of embedding fields and input-output relations is coextensive with the study of learning on the cellular ensemble level. Indeed, all of our previous examples of learning may be viewed from such a standpoint. In the present example, the idea that the process is a kind of inversion within the line control structures of the actual macroscopic ordering of events is especially obvious because the drift in the $\gamma(p) \rightarrow \gamma(p)$ lines is so beautifully suited for the occurrence of such inversions.

It is interesting to observe that the concept of spontaneous hierarchical inversion includes, and explicates, the earlier concepts of the Hullian "trace" and Guthrie's notion of "contiguity". In the Hullian theory, the emphasis is on the fact that certain neural processes continue to propagate after the experimental stimulus has terminated, and that important interactions occur between these neural "traces" and later experimental stimuli. Thus, the Hullian theory suggested the possibility that the proper spaces $(\gamma \subset \gamma^*)$ in which to study learning are not merely spatio-temporal isomorphic copies of the perceived paradigm of experimental events. It was, of course, impossible for Hull to construct the proper spaces during his lifetime. Guthrie, on the other hand, emphasized the fact that, whatever these interpolated "learning spaces" are, we are fortunate that the transformations of experimental paradigms which they induce preserve many macroscopic ordering relations. The study of spontaneous hierarchical inversion in suitable embedding fields crystallizes both of these theoretical adumbrations and gives a unifying perspective to the study of the

special geometrical variations in interpolated fields which are suited to the input-output structures that these fields subserve. It will be of advantage to the reader already familiar with classical psychological theories to compare these theories with the embedding fields that arise as we proceed. The reader will see that each such theory partially grasped some interesting feature of the learning process, but that none of them succeeded in clearly visualizing the global field constructions and local dynamical laws which are a necessary precursor to penetrating insights.

159 . Neuroanatomical Identification of $\mathcal{7}$ and $\mathcal{8}$: Laminar Neocortex and Cerebellum

Having made many remarks about special types of cells, it is difficult to proceed without noticing that we can give the fields $\mathcal{7}$ and $\mathcal{8}$ a direct neurological interpretation. In $\mathcal{7}$, we have already identified pyramid cells, stellate cells, cells of Martinotti, horizontal fibers, spider cells, input inhibitory interneurones fibers, and their most striking arrangements. $\mathcal{7}(q)$ is an output field with a definite somatotopic ordering which forms a border for the field $\mathcal{7}(p)$. Such a somatotopically organized strip of muscle control cells occurs along the central fissure, and juxtaposed along the central fissure are broad laminar regions of neocortex that exhibit all of the cells which we have identified and in the general arrangements which we have found to be natural ones. Moreover, near the central fissure, the stellate cells are less abundant than deeper into the association cortex proper, and we have already suggested that such a minimization of stellates is advisable wherever temporally invariant distributions of output cells are deposited. We therefore identify $\mathcal{7}(q)$ with the somatotopically arranged primary motor area. That portion of $\mathcal{7}(p)$ bordering on $\mathcal{7}(q)$ we identify with a portion of Brodmann's Area 4, and the more distal Γ_i regions of $\mathcal{7}(p)$ are identified with the associational cortex of Area 6. The $\mathcal{7}(p) \rightarrow \mathcal{7}(p)$ loops from 6 towards 4 now become familiar corticicortical fibers, and the parallelopipeds become the well-known columnar cellular arrangements. The higher cortical correlational inputs to distal Γ_i sections become the inputs to 6 from such regions as prefrontal cortex (via Area 9, to 8, to 6 transmissions), the temporal Area 22, and the parietal Area 5. Projections from such regions as Area 3 are, of course, also necessary to provide a substrate of sensory information. Area 4 must receive inputs predominantly from regions which are less highly devoted to purely associational activities, such as from the primary sensory cortices of Areas 3,1, and 2. All of these connections are well-documented.

$\mathcal{Z}$'s distinctive geometry also suggests an unambiguous neurological interpretation. If we turn $\mathcal{Z}$ upside down in Euclidean space--which does not change any functional relationships, since we merely bend the input-output fibers without altering their transmission capacities or temporal relationships--it looks very much like a piece of the cerebellar hemispheres. Indeed, we can draw up the following dictionary relating abstract field objects to neuroanatomical objects.

Abstract Field Object	Neuroanatomical Object
$\mathcal{Z}$	a portion of the cerebellar hemispheres
$\oplus_i R^i$	granule cells
$\oplus_i \mathcal{L}^i$	parallel fibers
$\mathcal{O}$	Purkinje cells
$\mathcal{LS}$	basket cells
$\mathcal{J}$	stellate cells
$\bar{\mathcal{J}}$	Golgi cells
$\mathcal{K}$	climbing fibers
$\mathcal{J}$	mossy fibers
$\mathcal{M}$	Lugaro cells
folia	folia

Our description is, of course, much oversimplified and incomplete. What is significant is that we posed a natural embedding problem on a surface with a special distribution of output points along the boundary, found that a surface exhibited specific deficiencies, and, in a series of very simple and rational steps, extended this surface until we could distinguish neocortical and cerebellar structures, as well as some of their interrelationships. Moreover, in constructing these fields, we were at every step aware of their specific advantages and disadvantages. It is very gratifying that a series of considerations, whose meaning we understand and which are simple to follow, gives rise to structures which strikingly resemble several of the fields which Nature has herself wrought in profusion. Instead of proceeding ad infinitum by constructing more fields attached to these fields, and so on, the applied worker, having seen how one comes to such a neuroanatomical interpretation, can now proceed more rapidly by directly applying our dynamical principles to the vast quantities of data available on neuroanatomical fields to achieve penetrating qualitative insights into their ensemble physiology. An important feature of our construction which should always be kept in mind while doing this is that one cannot expect to understand the significance of the local geometry of a field until one also

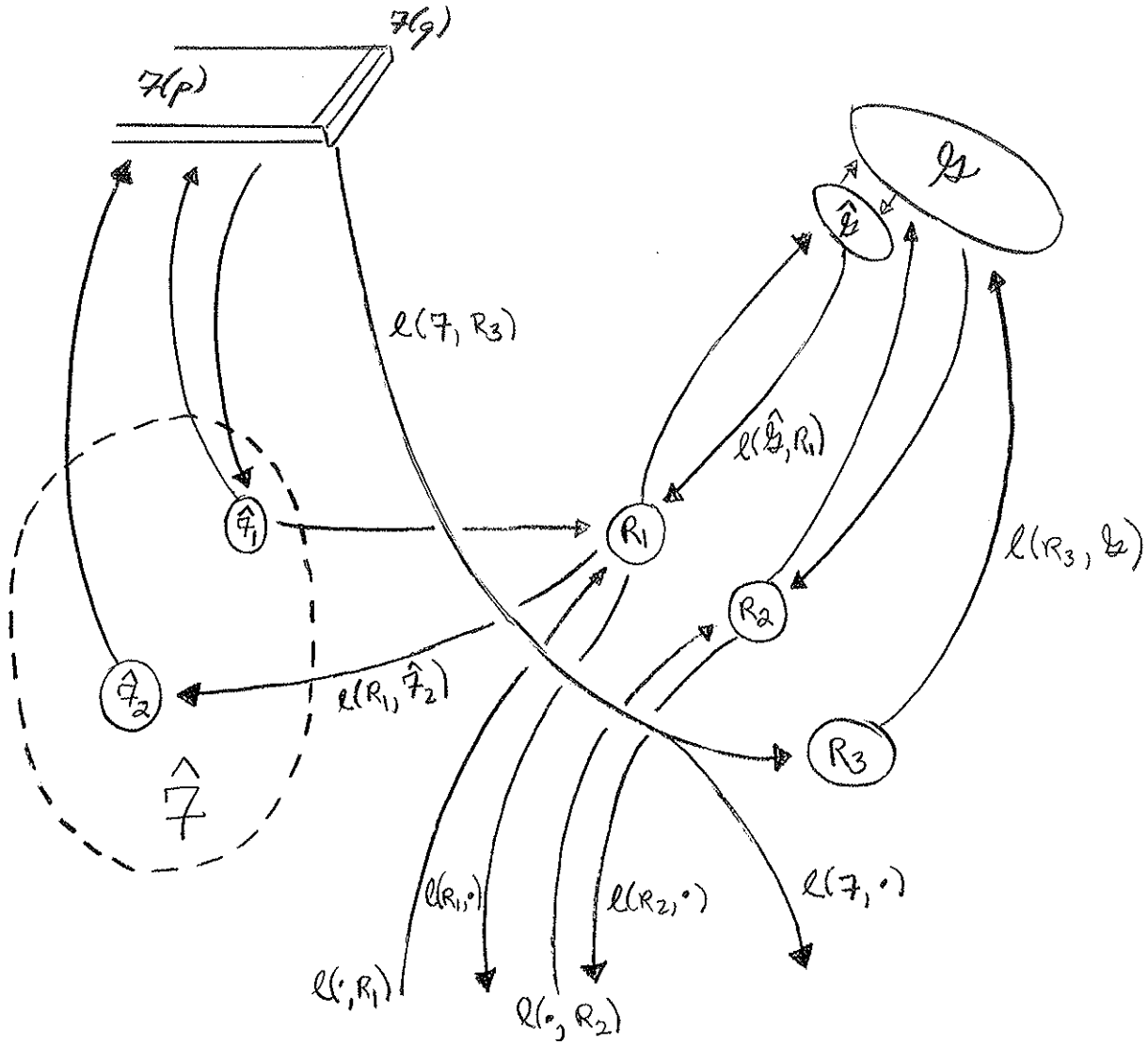
studies the local geometry of the fields to which it projects and from which it receives fibers. $\mathcal{T}$ and $\mathcal{Y}$ exhibit strikingly different geometrical arrangements. Yet we have seen that these differences complement one another beautifully and are manifestations of common underlying principles. In fact, the binding of spatially disparate $\mathcal{T}$ and $\mathcal{Y}$ points into well-coordinated series of points provides an excellent example of why a field of macroscopic control forms does not exhibit all microscopic cell distributions: Whole series of cells act as simple units, even before new embeddings bind privileged cell groups still closer together.

We must remark that several of our cellular identifications take the form of hypotheses about cell structure and function. The most speculative of these is the identification of $\mathcal{M}$ cells with Lugaro cells. Much research has suggested that the mossy fibers do transmit the greatest share of cerebellar inputs from distant nuclei while the climbing fibers do indeed seem to arise primarily from cerebellar sources. The Lugaro cells are distributed in a way that makes them look like candidates for $\mathcal{M}$ cells, but connections from Lugaro cells to climbing fibers have not, to my knowledge, ever been demonstrated. Even if the Lugaro cells are not the $\mathcal{M}$ cells which we seek, however, our discussion shows why the $\mathcal{J}$ fibers shoulder the primary burden of carrying inputs from distant nuclei, and that the $\mathcal{M}$ and $\mathcal{J}$ cells should be coupled most profusely somewhere within the cerebellum. Whether this coupling is mediated intimately by Lugaro cells, or even by a cell type in the dentate nucleus, is a matter to be settled neuroanatomically. In either case, we will know what the coupling signifies. It is also not clear to me whether the basket cells have actually been shown to inhibit the Purkinje cell bodies, although their uniquely qualified distribution for the task certainly suggests that they ought to behave like $\mathcal{LI}$ cells. Moreover, I have not seen a discussion, say, of the capacity of Golgi cells to form new excitatory $\mathcal{J} \longrightarrow \oplus_i R^i$ connections. A study of the distribution of excitatory transmitter in Golgi cell concentrations might clarify this matter. In general, the physiology of the cerebellum seems to be generally but vaguely understood, although our discussion points to clear and rationally derived hypotheses concerning cerebellar dynamics which involve all of the major neural cell types found in the cerebellar hemispheres. On the other hand, many of the neuroanatomical features of our abstract cells, even up to the rosettes, are well-known to be correct, especially as a result of Cajal's beautiful and exhaustive studies. An understanding of the primary structure of motor neocortex

and its cerebellar projections has thus been shown to be a consequence of a suitably posed boundary value problem and a general field extension procedure.

160. Neurological Renormalizing Fields

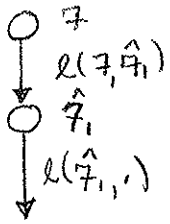
Apart from matters of detail, our qualitative discussion has omitted an extremely important collection of field structures. We need a renormalizing $\hat{7}$ for 7, and we must bind 7, $\hat{7}$, and $\mathcal{G}$ into a single dynamical unit which combines the advantages of a topographically well-ordered columnar arrangement, a broadly integrated renormalization field, and a field which permits ordered interactions between many 7 parallelopipeds. Now that we know what 7 and $\mathcal{G}$ represent neuroanatomically, we can look into an anatomy book to see what $\hat{7}$ looks like. Doing this, we find the following (much oversimplified and incomplete) diagram:



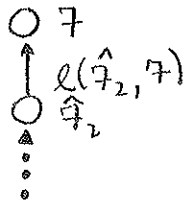
Here $l(Q_1, Q_2)$ refers to a fiber tract from field Q_1 to field Q_2. Accompanying this diagram is the dictionary:

Abstract Field Object	Neuroanatomical Object
$\mathcal{7}$	Areas 4 and 6
$\mathcal{Z}$	regions within cerebellar hemispheres
$\hat{\mathcal{Z}}$	dentate nucleus
R_1	red nucleus
R_2	inferior olive
R_3	pons
$\hat{\mathcal{7}}_1$	nucleus of the field of Forel
$\hat{\mathcal{7}}_2$	nucleus ventralis lateralis
$l(\hat{\mathcal{7}}_1, R_1)$	lenticular fasciculus
$l(R_1, \hat{\mathcal{7}}_2)$	rubrothalamic tract
$l(R_3, \mathcal{Z})$	brachium pontis
$l(R_1, \hat{\mathcal{Z}})$	rubrodentate tract
$l(\hat{\mathcal{Z}}, R_1)$	dentatorubral tract
$l(\hat{\mathcal{7}}_1, \cdot)$	ansa lenticularis
$l(R_1, \cdot)$	rubrospinal tract
$l(\cdot, R_1)$	spinorubral tract
$l(\mathcal{7}, R_3)$	corticopontine tract
$l(R_2, \cdot)$	olivospinal tract
$l(\mathcal{7}, \cdot)$	corticospinal tract
$l(\cdot, R_2)$	spino-olivary tract
$l(R_2, \mathcal{Z})$	olivocerebellar tract
$l(\mathcal{Z}, R_2)$	cerebello-olivary tract

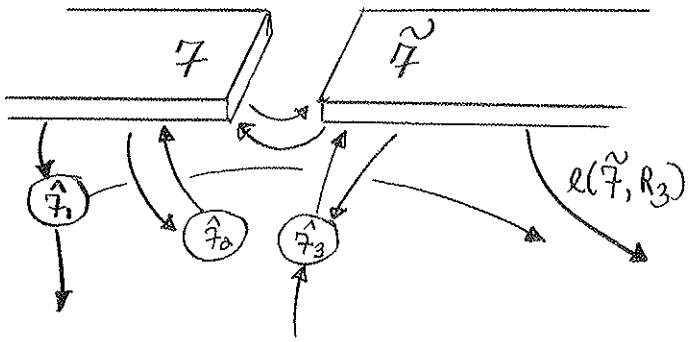
And so on. Several remarks can immediately be made about this diagram: (1) The dentate nucleus $\hat{\mathcal{Z}}$ acts like a somatotopically arranged dual field for $\mathcal{Z}$, just as $\hat{\mathcal{7}}$ is a dual for $\mathcal{7}$. (2) $\hat{\mathcal{7}}$ is decomposed into several individual nuclei which intimately interact with one another. $\hat{\mathcal{7}}_1$ and $\hat{\mathcal{7}}_2$ form only a small subset of these nuclei. The existence of another nucleus is immediately demanded by two asymmetries in the diagram: (i) $\mathcal{7}(p \oplus q)$ is primarily a motor output field; the points of $\mathcal{7}(p \oplus q)$ must be augmented by a sensory field of symmetrical construction. (ii) The output line $l(\hat{\mathcal{7}}_1, \cdot)$ in



has no input analog in the diagram

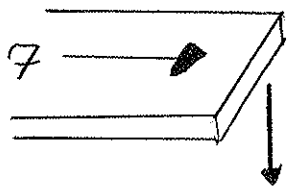


Both of these difficulties can be removed by the augmented diagram

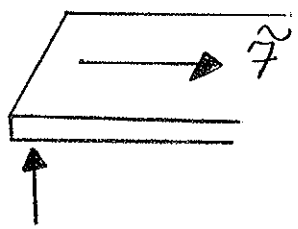


Abstract Field Object	Neuroanatomical Object
$\tilde{7}$	postcentral and parietal cortex (Areas 3,1,2,5,7)
$\hat{7}_3$	nucleus ventralis posterior
$\ell(\cdot, \hat{7}_3)$	medial lemniscus; ventral and lateral spinothalamic tracts
$\ell(\tilde{7}, R_3)$	parietopontine tract

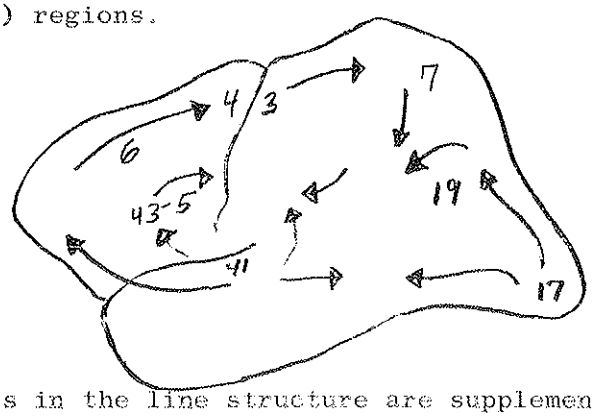
We obviously cannot attempt to discuss all of these pathways in this paper, and must refer the reader to the neuroanatomical literature for details. Certain qualitative conclusions can, however, be drawn. The embedding drift in 7 looks like



and flows towards to output points. Thus, the embedding drift in the primary sensory field $\tilde{7}$ looks like



and we expect that sensorimotor embeddings deep in the parietal cortex will be of higher associational type than embeddings bordering Area 3 itself, by a simple extrapolation of our discussion of the 6 to 4 drift. These hierarchical drifts within the line structure merge with drifts from the primary visual (17) and auditory (41) regions.



Such local drifts in the line structure are supplemented by profuse and delicately balanced corticicortical bundles of various lengths and distribution, and cortical → subcortical → cortical lines which help to bring disparate cortical regions rogether into systems that blend inputs from different modalities into control forms of higher type. $\mathcal{G}$ also helps in this task, since cortical fields that are far from one another can be made to have intimately related $\mathcal{G}$ representations. Indeed, we expect long $7 \to 7$ associational tracts to be mirrored in $\mathcal{G} \to \mathcal{G}$ connections. In general, differences in the geometrical relations of two 7 fields will be directly reflected in relations between their $\mathcal{G}$ projections. All cortical 7 fields possess dual $\hat{7}$ fields, which --as $\hat{7}_2$ and $\hat{7}_3$ show--can be the carriers of important input and output streams of excitation.

161. Comment on Sensory Nuclei, Neural Funnelling, Mach Bands, and Reaction Times

Even in our study of blocking in p_W, we saw that special regions of p_W could come into the service of specific p_i. In the present context, such a specificity of interaction means that interactions like $7 \leftrightarrow \hat{7}_2$ and $7 \leftrightarrow \hat{7}_3$ are somatotopically arranged. 7 ($\tilde{7}$) is a refinement of the structure of $\hat{7}_2$ ($\hat{7}_3$), and ordered lateral inhibitory structures can easily be envisaged in both the thalamic and cortical loci. A similar remark holds for the 7^* analog of $p_{(r)}$; lateral inhibitory fields resculpt potential distributions at practically every stage of transmission. A chain of subcortical nuclei leading to a specific neocortical termination has several advantages. First, the distinguishability of

inputs increases, as it does for any chain with local distributions of ordered converging and diverging nerve fibers as successive transmissions occur. Second, the successive application of lateral inhibitory inputs increasingly crispens the strength distribution, accentuating differential excitations and suppressing the spontaneous background effects. Third, if strong coupling mechanisms exist in every nucleus, then the embeddings representing a given external input are not localized. Rather, they are spread over all nuclei, and the nuclei which are the earliest to be excited contain the coarsest embeddings. New inputs are therefore gradually redirected over successive nuclei, with the finest details being left relatively intact until they reach the neocortical loci that can distinguish them. Fourth, each nucleus can act as a potential source of outputs to non-cortical terminals. The most peripheral nuclei, which distinguish best the coarsest input features, are ideally suited to activate the most broadly interacting renormalizing fields to signal the onset of new events to the higher cortical centers; e.g., the reticular formation. Fifth, chains of nuclei permit a much more sensitive staggering of input onsets through an interaction of input intensity with output thresholds than does a single nucleus. Such a staggering helps to lift a purely spatially distributed input into a space-time distribution of excitation for which temporal differences reflect input intensities as well as input onset times. In particular, intense inputs of later peripheral onset can overtake earlier weak inputs, and inhibit (or mask) them out of existence.

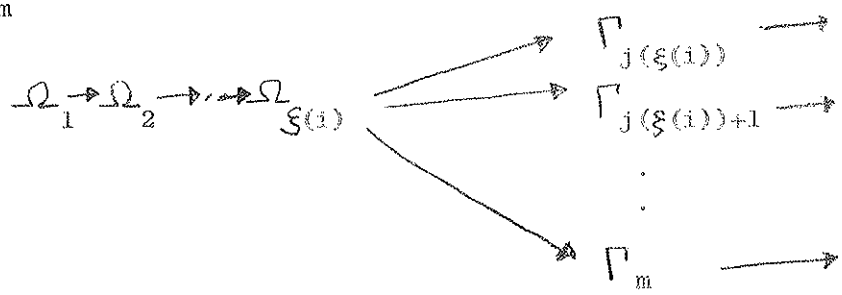
It is important to realize that the higher centers cannot possibly discriminate input details which are not at all present in the peripheral excitation distributions. All of the most refined sensory perceptions are present in the peripheral neural input--which is one important reason why the retina has made such extraordinary provisions for processing peripheral inputs in manifold ways in the first few steps of neural transformation. But they are joined by excitations which latently contain all of the features of the environment which the organism can perceive through the given modality. Here is where locally ordered, lateral inhibitory interactions in each nucleus play a critical role. In the more peripheral nuclei, the broadest features of the input are embedded and are gradually eliminated by a lateral inhibition. In the next nucleus, these broad features of the previous input will have been inhibited away, so that the more delicate input variations carry a greater total weight in this input. These more delicate input variations will therefore be able to induce relatively more substantial embeddings than they could in the

previous nucleus. After passing through many successive nuclei, the finest input details have achieved a relatively high weight, so that they, too, can induce nontrivial embeddings and exert a strong effect on some field structure. It is, indeed, a familiar fact that certain specific thalamic sensory nuclei are capable of reacting to lower sensory modalities in decorticate preparations, but that intricate neocortical structures are necessary to discriminate the finest details of the input. This process of successive refinement does not terminate when the cortex is reached. Such a phenomenon as color vision has, for example, been tentatively assigned to Area 18, which lies far from the periphery in terms of successive transmissions across sensory nuclei, and is primarily related to these nuclei via the neocortical Area 17. The higher associational fields lie still further from the peripheral input sources, and these fields subserve correspondingly more delicate interactions. We will discuss details of these sensory interactions in a paper that is presently being prepared. The important qualitative conclusion for the present is that the underlying principles guiding these successive discriminations are the very same which were applied in discussing the behavior, say, of output pyramid and Betz cells. In both situations, one cannot achieve a deep insight into the processes involved by studying only localized collections of cells. Rather, one must consider an entire input-associational-output system.

Von Bekesy's concept of neural funnelling is brought to mind in this context, since the lateral inhibitory mechanisms which we envisage for the specific thalamic nuclei are well suited to accentuate the contours of incoming input distributions, as are even such peripheral neural structures as the retina, where one manifestation of ordered "on"-"off" and "off"-"on" regions is the familiar phenomenon of Mach bands. Each of these effects refers to a partial recognition of the importance of inhibitory renormalizations. It is, however, possible to clearly envisage these renormalizations as a temporally ordered refinement of a space of equivalence classes of potential distributions only when an entire system of nuclei is considered as a single unit.

A somewhat more specialized example of these phenomena is the following. If the sensory nuclei send ordered lines to fields whichcontrol motor outputs of various sorts, then inputs can generate outputs after reaching the first few nuclei which can discriminate behaviorally relevant features of the input. We expect the lines sent out by a given nucleus to eventually lead to output lines which control motor centers whose complexity is comparable to the input complexity which the nucelus can discriminate. Since the various nuclei receive inputs in

different stages of the inhibitory sharpening process, we can immediately conclude that reaction times, with a fixed response measure, will depend both on specific characteristics of the input and on the "psychological set" of the output field which is formed by experimental instructions and other input precursors of renormalizing type, a fact which has not been clearly understood in the psychophysical literature. In particular, consider an input I with "stimulus characteristics" ξ. Suppose that the ξ are first distinguished in $\Omega_{i(\xi)}$, and let the entire truncated chain $\Omega_{i(\xi)}, \Omega_{i(\xi)+1}, \ldots, \Omega_n$ be capable of distinguishing ξ, for specificity. Let Ω_j lead to the output fields $\Gamma_{j(i)}, \Gamma_{j(i)+1}, \ldots, \Gamma_m$. The reaction time to the ξ characteristics of I is at least as great as the time required for transmission to occur over the system



The exact determination of this reaction time of course depends on how fast the input excites the appropriate strength functions to suprathreshold values, and this in turn depends on details of field structure as well as details of input distribution. Now suppose, moreover, that experimental instructions have pre-set the output field Γ_k, $k > j(\xi(i))$, and that Γ_k inhibits $\Gamma_i, i \neq k$. The reaction time to the ξ characteristics of I are here at least as great as the time required for transmission over the system

$$\Omega_1 \to \Omega_2 \to \cdots \to \Omega_{\xi(i)} \to \Omega_{\xi(i)+1} \to \cdots \to \Omega_{m(k)} \to \Gamma_k \to ,$$

where $\Omega_{m(k)}$ is the first Ω_i which discriminates ξ and which leads to Γ_k. This reaction time is, other things equal, an increasing function of $m(k)$. Our next paper will discuss psychophysical dependencies such as these more explicitly.

The $\hat{\mathcal{F}}_i$, i=1,2,3, by no means exhaust $\hat{\mathcal{F}}$. This can be seen directly by observing that the projection range of the lateral inhibitory mechanisms in $\hat{\mathcal{F}}_2$ and $\hat{\mathcal{F}}_3$ is limited by the requirement that these nuclei be able to transform somatotopically arranged inputs into somatotopically arranged outputs. The task of renormalizing extensive cortical regions must therefore be left

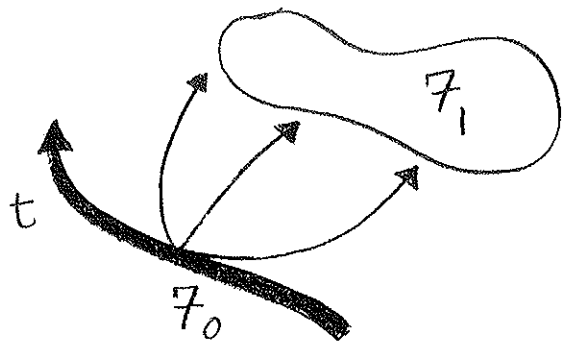
to other nuclei. Indeed, since many interacting $\mathcal{F}\oplus\hat{\mathcal{F}}\oplus\mathcal{G}$ fields must all be simultaneously renormalizable when new behavioral inputs begin to arrive at the peripheral receptors, we must envisage dual fields $\hat{\hat{\mathcal{F}}}$ which tie together the $\hat{\mathcal{F}}$ duals, and possibly duals to these. The space which eventually develops by such extensions is one in which renormalizing fields are hierarchically organized relative to the peripheral input stations and the behavioral output stations. The neural processing stations which lie nearest to the periphery--in terms of the minimal number of transmission steps from the periphery--must control broadly projecting renormalizing fields, for these stations must quickly signal the onset of new events, as we already observed in our remarks on EEG suppression. Similarly, the fields which subserve the last few steps of processing overt behavioral patterns must exert a strong control on broadly projecting suppressive renormalizing fields in order to prevent competitition between incompatible behavioral emissions. The reticular formation provides an excellent example of a broadly projecting renormalizing field. Intermediate stages of neural transformation require renormalizing fields which exhibit the more local projection domains appropriate for the processing of local strength waves and eddies.

162. Higher Associational Fields

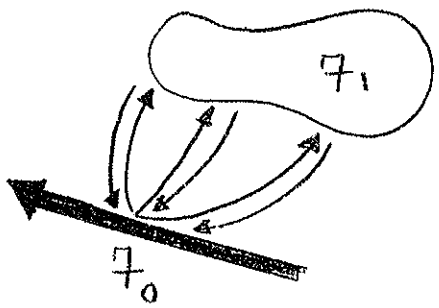
Fields which receive direct inputs and emit behavioral outputs cannot sustain the complicated new embeddings and spontaneous inversions necessary for higher learning. Their proper functioning, indeed, depends on the existence of line structures which remain essentially invariant through time. If the input-output line connections fluctuated wildly, the interpolated embeddings would lose all behavioral significance, for they would at one moment be activated by one input and generate one output, and at the next moment be activated by a different input and generate a different output. In the highest mammals, the associational fields are therefore only indirectly connected with input and output fields, and the most indirect connections conduce to the embedding of the most subtle and sensitive control forms. The simplest motor associational fields, as we have seen, must be able to control output fields like $\mathcal{F}(q)$, whose regular somatotopic arrangement supports a relatively simple spontaneous inversion mechanism. This simplicity is somewhat deceptive, for even $\mathcal{F}(q)$ projects with great delicacy to subcortical nuclei and spinal destinations. Nonetheless, viewed

as a set of given control forms, the (approximately) one-dimensional symmetry of $\mathcal{T}(q)$ provides us with as simple an output field as we can hope for. The associational fields $\mathcal{T}_o$ which control integrated sensory memories, on the other hand, exhibit much more subtle arrangements, even when we consider them as control forms. Such $\mathcal{T}_o$ are found, for example, within the temporal lobes. An $\mathcal{T}_o$ must project to many sensory fields simultaneously in order to be able to reproduce the divers concomitants of a single past experience. Yet it is quite pointless for $\mathcal{T}_o$ to send its most profuse connections to primary sensory regions such as 3, for these regions do not possess rich enough stellate cell collections to render delicate new line connections possible. $\mathcal{T}_o$ must project in greatest profusion to those sensory fields which are endowed with line restructuring mechanisms in their own right. Such sensory fields are precisely those whose lines have been continually restructuring themselves in response to inputs from the more primary sensory receptive areas, like 3. Examples of such associational sensory areas, the $\mathcal{T}_1$ fields generically, are the somesthetic and visual association areas. These $\mathcal{T}_1$ associational regions are closely juxtaposed along the speech areas of the angular and supramarginal gyri, and are thus well-located for the formation of spontaneous inversions which eventually lead to such behavioral evidence of sensory familiarity as the evokation of the name of an object whose input distribution has just flowed through the proper $\mathcal{T}_1$ representational channels. An important part of the task of an $\mathcal{T}_o$ is to reproduce on the various $\mathcal{T}_1$ large numbers of the control forms which were re-excited, or newly embedded, during a given multi-sensory "experience." This is achieved by exciting a control form within $\mathcal{T}_o$ which leads to all the $\mathcal{T}_1$ control forms that were activated during the experience, many of these $\mathcal{T}_1$ forms belonging to different modalities. One result of such reactivation is the organism's capacity to emit many overt behavioral signs of the recollection. In this sense, the sequence $\mathcal{T}_o \rightarrow \mathcal{T}_1 \rightarrow$ overt behavior (e.g., naming) is similar to the $\Gamma_{i+1}^{(m-1)} \rightarrow \Gamma_i^{(m-k)} \rightarrow \mathcal{T}(q)$ sequence, although the "overt behavior" term in this sequence is a multi-stage process itself. An equally important result of such reactivation is the replication of the interpreted sensory distributions, within $\mathcal{T}_1$, which are the embedded mirroring of the experience itself. It is here that the simple one-dimensional symmetry of $\mathcal{T}(q)$ becomes especially inadequate. For many sensory distributions on an $\mathcal{T}_1$ will be spread broadly, and intricately, over a large cortical surface. One need only consider the visual system to realize that the symmetries of $\mathcal{T}_1$ distributions will be of a higher dimensionality than one. Yet $\mathcal{T}_o$ must be able

to reproduce such higher dimensional distributions, and several of them at once. Summarizing, an $\mathcal{T}_o$ must (1) project broadly to many interpretive sensory cortices, and must (2) project with such a highly specific pattern that complicated excitation forms on each of these cortices can be fairly well reproduced by exciting the appropriate $\mathcal{T}_o$ control form. On the other hand, (3) the control forms in $\mathcal{T}_o$ must be so simply ordered that we can form a concept of <u>time</u>, by which memories can be judged as "old" or as "new". If this natural perception of a temporal order is carried to its limit, the temporal ordering of $\mathcal{T}_o$'s control forms means that a one-dimensional "time" variable can be homeomorphically mapped onto the collection of $\mathcal{T}_o$ control forms. (1)-(3) together thus demand that a space of $\mathcal{T}_o$ control forms, related to one another by a low dimensional symmetry, control a space of $\mathcal{T}_1$ forms of much higher dimensionality. In the space of forms, this situation looks like



Moreover, it is a familiar fact of daily life that a highly "distinctive" stimulus from a past event can call to mind many other sensory concomitants of that event. In terms of $\mathcal{T}_o$, $\mathcal{T}_1$ interactions, this means that certain $\mathcal{T}_1$ control forms can excite other, simultaneously embedded forms, via $\mathcal{T}_o$; or that a "subspace" of control forms of an experience can activate the "entire representation space" of that experience. In terms of our diagram, this means



We also know from daily experience, however, that many events occur in varying contexts. Such events are often free from specific contextual recollections. We have already studied the essential background for understanding this fact in terms of multiple crossings and renormalization patterns. Qualitatively, when the line structure is very rich, lines joining the representation of a given event to a certain context representation may differ for different contexts. Yet, when this event reoccurs alone, all such contextual representations will be simultaneously activated, and will tend to inhibit one another before any one contextual representation becomes sufficiently intense to enter consciousness.

Moreover, sensory experiences are delivered as inputs to sensory associational fields in which no natural temporal ordering apart from the ordering induced by the lines themselves exists, and this ordering is in general far from being a one-dimensional temporal ordering. Yet it is precisely these sensory associational fields which most often are the source of inputs leading to experiential memories, let alone experiences.

163. Dimensionality. Time and Minimal Fields

The eventual outcome of such considerations is the realization that a global time variable does not exist in our experience. Simultaneity is measured entirely in terms of the relative timing of the arrival of excitatory and inhibitory inputs at particular cellular loci. Our various input, output, and associational fields, with their special geometrical symmetries and hierarchical arrangements, continually play upon one another. The dimensionality of our experience is the dimensionality of the global minimal field which exists at any moment. In this sense, our experience is a succession of global minimal fields. Notwithstanding the great diversity of these global minimal fields, the local laws that generate them are of a universal character, as we have tried to show through various examples.

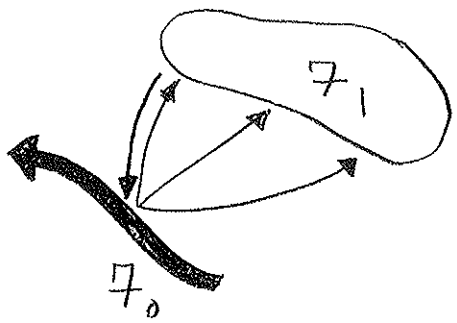
164. Interaction Between Sets of Associational Fields and Inputs

This discussion also points to another clear need for global renormalizations shortly after a sufficiently intense input reaches the peripheral receptors. For our General Principle on the delivery of external inputs show that an $\mathcal{7}_o$ must be activated before inputs are delivered profusely throughout the $\mathcal{7}_1$ specific sensory associational areas. Otherwise, inputs from $\mathcal{7}_o$ could not possibly reach $\mathcal{7}_1$ in time for a spontaneous $\mathcal{7}_o \rightarrow \mathcal{7}_1$ inversion to occur, whence the input could not possibly induce an integrated experiential record in the lines. $\mathcal{7}_o$ must therefore be activated during the early stages of the processing of a new input. This activation cannot be accomplished by a specific sensory or associational field, since these fields have not themselves yet been excited. It must therefore be accomplished by a broadly distributed, nonspecific field, as we just claimed.

Notice that $\mathcal{7}_1$ must be provided with a mechanism to prevent $\mathcal{7}_o$'s broadly distributed input from flooding $\mathcal{7}_1$, and $\mathcal{7}_1$-connected fields, with excitation. Lateral inhibitory mechanisms are indispensable here in permitting nonspecific inputs without generating behaviorally chaotic outputs. $\mathcal{7}_1$ fields must be covered by a collection of local weighted sets of antagonists which do not permit massive outputs in response to nonspecific $\mathcal{7}_o$ inputs. The appropriate weighting is determined by the particular $\mathcal{7}_o \rightarrow \mathcal{7}_1$ distribution of lines. A simple inhibitory mechanism is given by sets of inhibitory interneurones which send their axons to the $\breve{1}$ regions of nearby output cells. Such neurons prevent $\mathcal{7}_o$-induced output transmission from $\mathcal{7}_1$ without preventing the formation of new line connections in cellular regions that are nonvolumetrically bound to the $\breve{1}$ regions; for example profuse dendritic trees. This is essentially the same mechanism which we postulated for the prevention of massive outputs from $\Gamma_{i+1}^{(m-1)}$ in response to inputs delivered before $\Gamma_i^{(-k)}$ had achieved indirect control of $\mathcal{7}(q)$. We now see that it is a general mechanism which permits intracortical input processing while sparing regions to which the cortex projects from massive cortically induced inputs which are not associated with a nonrandom cortical embedding. It also now appears that the sets of cortical antagonists which accomplish this inhibition of cortical output must have a joint distribution which prevents outputs from being induced by any of the neural fields sending lines to the cortex when they are randomly excited.

Since $\mathcal{7}_o$'s initial activation precedes full $\mathcal{7}_1$ activation, the spatial

locus of initial 7_o activation by an isolated input must be independent of the particular 7_1 excitation distribution induced by the input. Nonetheless, the final locus of 7_o activation is strongly dependent on the particular 7_1 distribution; for a random choice of terminal 7_o loci implies that a single 7_o control form can come to control both "old" and "new," and behaviorally incompatible, 7_1 control forms. This means that as the 7_1 distribution evolves, it must project excitation to the 7_o field, which renormalizes the $7_o \rightarrow 7_1$ transmissions so that they gradually come to be compatible with the evolving 7_1 excitation form. But some 7_1 forms already exist when a new input to 7_1 arrives. Thus some "subspace" to "total space" $7_1 \rightarrow 7_o \rightarrow 7_1$ connections like



also exist. Consequently, as the externally induced 7_1 input is being delivered, this input gradually causes 7_o, as well, to spray 7_1 with specific input distributions; namely, those inputs which correspond to globally embedded experiences that occurred when the externally induced 7_1 input did in the past. It is important to realize that this $7_1 \rightarrow 7_o \rightarrow 7_1$ influence develops gradually in time; the $7_1 \rightarrow 7_o \rightarrow 7_1$ interaction occurs before the external input to 7_1 is fully processed. Thus, the $7_o \rightarrow 7_1$ component of the input never really responds to a complete representation of an 7_1 form. Rather, it receives 7_1 input segments at every stage of the 7_1 excitation process, and responds to these segments reciprocally with $7_o \rightarrow 7_1$ inputs. The $7_o \rightarrow 7_1$ inputs, in turn, alter the background excitation which greets new external 7_1 input segments. The line embedding within 7_1 for these new external input segments is correspondingly altered. We thus have successive, reciprocally-influenced contractions within the 7_o and 7_1 fields just as we had within individual cortical columns.

If during this complex $7_o \leftrightarrow 7_1$ interaction, 7_1 transmissions satisfy some "salient" response criterion--where response criteria are understood in terms of complex, embedded excitatory and inhibitory interactions which finally generate an output to a terminal behavioral motor form--, we will be convinced that the subject recognizes the input. A frequent precursor to such overt behavioral evidence of recognition, and one which can easily exist <u>without</u> overt behavior by simply globally suppressing the later stages of motor processing, is the intense reproduction of an embedded form on some thalamo-cortical sensory field. With this reproduction comes conscious sensory awareness of the input.

165. <u>What Is An Object?</u>

This simple reasoning has important epistemological consequences. "When" does the "experience" occur? "Where" do the "objects" of the experience lie in the brain? To the naive view, the "experience" occurs "when" the input "reaches the brain," and "where" this input projects. But we have just seen that the input activates wholly nonspecific excitations before it really gets near to a sensory center that subserves consciousness, and thereafter follows an intense interaction between many fields that strongly deforms the trajectory which the external input would have followed in vacuo. This intense interaction is the competitive process which we discussed when introducing vector input functions, and the trajectory which the external input would carve out in vacuo is the "virtual trajectory" of the input. We now see that, by virtue of interactions like $7_o \rightarrow 7_1$, an external input which has been delivered many times in the recent past will be differentially facilitated by the $7_o \rightarrow 7_1$ input, because a set of 7_o control forms for the 7_1 control form(s) representing this input has already been embedded and selectively facilitate this form. The excitation delivered to such an 7_1 form will therefore grow very rapidly. In particular, motor consequences of such a form will be rapidly realized; for example, reaction time for naming the "object" represented by the form will be small.

An external 7_1 input is thus never embedded in vacuo. The new $7_o \leftrightarrow 7_o \leftrightarrow 7_1 \leftrightarrow 7_1$ lines that form under the impetus of this input depend on what $7_o \leftrightarrow 7_o \leftrightarrow 7_1 \leftrightarrow 7_1$ lines already exist, and each such interaction involves a corresponding $\hat{7}\oplus\ell$ interaction. For example, $7_o \rightarrow 7_1$ inputs

inputs help to automatically tie the new $\mathcal{T}_1$ input to all of the contextual innuendoes with which previous occurrences of the input have been associated. This rich associational complex is activated in stages. If the embedding field is quiescent when the external input reaches the periphery, the field will be globally excited. Then, gradually, as further stages of the space-time form of the input occur, the embedding space gradually contracts its global excitation distribution on the basis of specific features of the input. This successive contraction of the excitation distribution can be viewed as a temporally ordered, ever refining, reconditioning of the embedding space in response to the increase of "information" about the input as time goes by. Indeed, we have already discussed the close relation of the information functional to conditioning processes through the mechanism of inhibitory renormalizations. Gradually, the increasingly specific line embeddings representing old inputs with space-time distributions similar to that of the current $\mathcal{T}_1$ input begin to interact with the $\mathcal{T}_1$ input. All of these intricate interactions occur before we recognize the input consciously, and before any object-specific outputs can occur. The foci of new line embeddings, and the embeddings themselves, are thus considerably determined before we are even fully aware that the input has occurred. This rapid focalization of the input distribution made possible the conceptual delivery of an input "to" a point in our macroscopic verbal fields. The conceivable flexibility of $\mathcal{T}_0 \leftrightarrow \mathcal{T}_1$ interactions shows, however, how careful one must be in deciding "where" to deliver such an input in the space of forms, as we saw in invoking vector input functions with functional coefficients. After the input actually excites an embedded form or forms in $\mathcal{T}_1$ sufficiently to excite $\mathcal{T}_1$-controlled behaviorally significant forms, still new inputs will occur, either by sensory- or by neural-feedback, and the process continues. It is this part of the process to which our original discussion of interactions between macroscopic forms was addressed. If the field is already focally activated when a new external input arrives, the field renormalization will be altered in a manner that the reader can now easily qualitatively envisage on the basis of our previous remarks. In particular, new inputs can be inhibited out of existence before reaching cortical centers if they are incompatible with presently activated control forms.

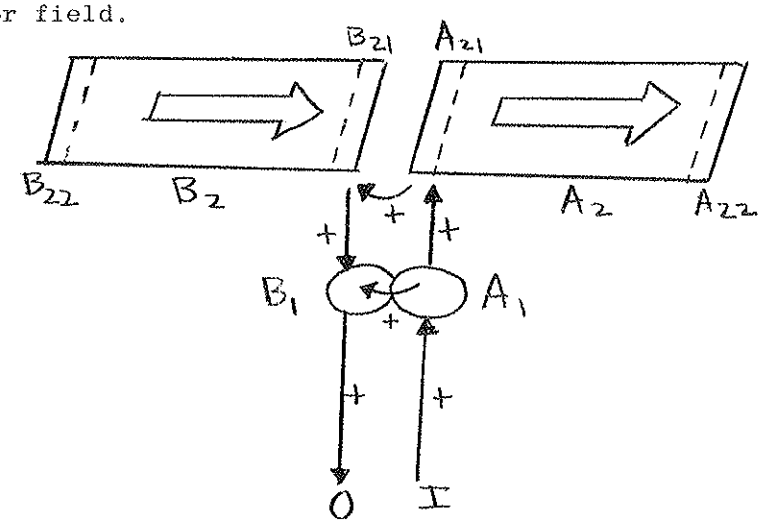
Thus, we do not merely recognize an input at some time or in some place. Rather, there gradually emerges to a behaviorally significant level an asymptotic excitation distribution that is produced only after the entire

7^* embedding space is involved in an excitation contraction process which calls upon all previous embeddings, although some embeddings are more highly favored than others are under different inputs, and different regions of 7^* exhibit various projection ranges and selectivities during this contraction process. An "object" thus cannot be conceived of as an entity which is definable locally either in space or in time. Every object is a partial realization of every other object of our past experience. No object exists in a relational vacuum. It is a direct consequence of this fact that, in the strictest sense, no operation can be performed on an object by a human being in a single instant of time. Indeed, we have already criticized the view that a "decision" is a locally definable concept. It is the control forms which underlie a decision which are relatively localized, but even these are really spread out in space-time. Every mathematical operation which has psychological consequences must therefore be viewed as a time-bound process. Does there exist a mathematical operation without such consequences? The apparent discreteness of many such processes is largely due to a sensitive stabilizing interaction between the actuated embedded representation forms of the relevant objects and the forms that control eye movements and the other regulatory feedback devices which localize our primary perceptual fields. These stabilizing properties will be studied in forthcoming papers. Another consequence of these observations is that the processes of "perception" and of "learning" cannot be naturally distinguished in a functional way. For during the very process of coming to "perceive" an object, the inputs representing this object interact with the lines remaining from earlier "learning" experiences and form new lines based on the residues of these experiences. Without such "learned" embeddings "perception" would be impossible, while "perceptions" are a particular manifestation of the general embedding pattern.

All of these remarks possess a rigorous sense in every specific embedding field. A particularly revealing way to view the recognition process in such a field is to deal with a minimal field representation throughout. When this is done, all successive contractions of the excitatory distribution are seen, in a vivid way, as contractions of the dynamical space which immediately reveal all of the geometrical symmetries underlying the contraction and their consequences for the output mechanisms.

166. Pyramidal and Extrapyramidal Systems

The ultimate quantification of these remarks will require many specific field constructions. A certain amount of additional insight can nonetheless be rapidly acquired by considering the following idealized diagrams, in which the broad arrows represent the direction of embedding drifts in a charicature of a sensorimotor field.

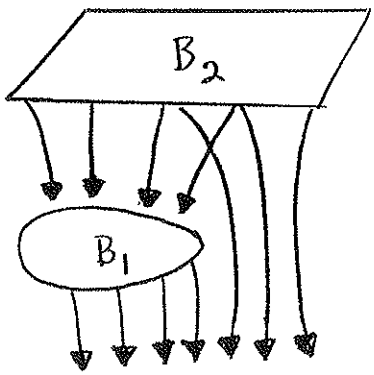


A_1 is a spacific (thalamic) sensory nucleus delivering inputs to an idealized A_2 (neocortical; e.g., 3) sensory field. Each sensory A_i field is paired with a motor B_i field which is capable of controlling motor output forms of the same level of complexity as the sensory field from which it receives excitation. Each A_i and B_i field possesses lateral inhibitory structures whose lines are sufficiently short to preserve the general somatotopic structure of their inputs in the outputs they generate. Inputs I to A_1 that are easily distinguished within A_1 activate the $I \longrightarrow A_1 \longrightarrow B_1 \longrightarrow O$ transmission system, to which the system $A_1 \longrightarrow A_2 \longrightarrow B_2 \longrightarrow B_1$ contributes in a secondary fashion. Inputs which are well distinguished only in A_2 are, by contrast, primarily processed through the system $I \longrightarrow A_1 \longrightarrow A_2 \longrightarrow B_2 \longrightarrow B_1 \longrightarrow O$; the $A_1 \longrightarrow B_1$ link is dynamically relevant only insofar as lateral inhibitions and other standard dynamical transformations in A_1 accentuate the input features which are well discriminated by A_2.

In this diagram, if B_2 projects only to the cells in B_1 with which A_1 interacts, then the capacity of A_2 to distinguish input types must be comparable to that of A_1's capacity. For inputs that are discriminated by A_2 reach behavioral significance as O outputs only after they are transferred, via B_2, through B_1, and B_1 is constructed to distinguish inputs discriminable by A_1. If A_2's input-discriminatory capacity far surpasses that of A_1, then either of two general

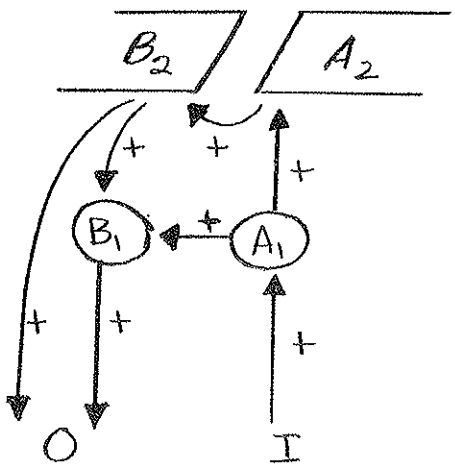
alternatives must hold. (1) B_1 must contain phylogenetically newer cell groups to which B_2 primarily projects, while A_1 projects primarily to phylogenetically older B_1 regions. Since the cellular grain in $A_2 \longrightarrow B_2$ is often finer than that of the phylogenetically older regions of B_1, we expect A_1 to send lines primarily to a macrocellular B_1 region, while B_2 sends lines to a primarily microcellular B_1 region. To the extent that A_1 and A_2 can both discriminate the same input features, the cellular structures of the B_1 regions to which they send lines (perhaps in several steps) are also similar. Cellular gradations in B_1 thus reflect differences in the discriminatory capacities of the systems $A_1 \longrightarrow B_1$ and $A_1 \longrightarrow A_2 \longrightarrow B_2 \longrightarrow B_1$. Or (2): B_2 outputs bypass B_1 entirely. The Betz cell corticospinal tracts are representatives of this latter alternative. Here the neocortical columns entirely replace the intermediate B_1 stations as output processing stations. Such a bypassing of B_1 cannot mean, however, that B_1 and B_2 operate as entirely independent systems, for they are indirectly coupled by the close binding between A_1 and A_2 that is necessary to insure that inputs which are not annihilated by A_1 are not wastefully annihilated when they pass on to A_2. Nonetheless, inputs to B_2 from the B_{22} fringe or other input sources not involving A_1 can, indeed, partially bypass the B_1 system. Such inputs are often from phylogenetically recent fields, such as those governing socialized or intellectual behavior. This example thus illustrates the important fact that the degree of dependence of neural subsystems depends critically on which inputs are perturbing them at any time.

Also observe that it is less probable for Betz cell corticospinal tracts to emerge from regions near B_{22} than from regions near B_{21}. For the B_{21} region is the receiver of the most stable and direct source of sensory inputs, while the B_{22} region comes to embed forms which control B_{21} only indirectly. These B_{22} forms therefore emit excitation whose effects on B_{21} are realized in successive stages. Among such effects are the effects of long B_{22} outputs lines on long B_{21} output lines. The long pyramids of B_{22} will thus often send lines to cellular way-stations which in turn send lines to B_{21} terminal regions. We have a qualitative diagram like



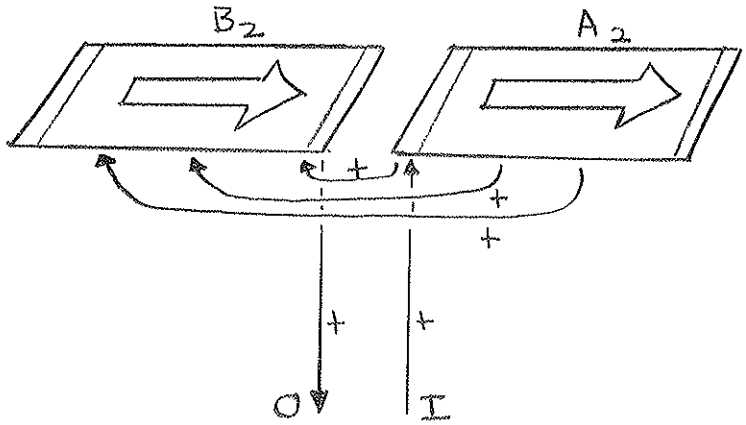
One can extend this diagram, though in several stages, to achieve a quite general understanding of the functional differences between pyramidal and extrapyramidal systems. Several of the crucial questions in so doing are: (1) How many cellular way-stations are interpolated between a neocortical source and its spinal destination? (2) How localized are the cell distributions in the interpolated nuclei? (3) What input modalities, and in what order, converge directly or indirectly on these interpolated nuclei? and (4) How do such local variables as transmission thresholds vary with the density of fibers converging from each input source? Since the possible systems to study are so numerous, it is impractical for us to try to itemize them here, and it will probably be more profitable to the reader to select a system of special interest to him and to follow it through, guided by the equations, principles and examples which have already been presented. A cautionary remark should be observed. The partition of long neocortical output lines into two classes--pyramidal and extrapyramidal--is much too restrictive. If a given localized cortical region sends direct lines to spinal regions and indirect lines to thalamic or other regions rostral to spinal centers, then one cannot understand all the preparatory secondary, and multisystemic effects controlled by this region unless both types of lines are simultaneously studied as a unified system, and their relative importance to the functioning of this system assessed. A neocortical center cannot operate successfully by sending lines exclusively to a single output destination. Rather, it must, in a graded fashion, modulate all of the various cellular systems over which it has been superimposed through gradual evolutionary changes.

To illustrate these remarks in a simple case, consider the diagram



Suppose that A_1 inhibitory lines inhibit excitations beyond a range R of their excitatory source in a somatotopic fashion. Then A_2 excitatory lines will lie predominantly within the range R', which is the diameter R as it is projected by $\mathcal{L}(A_1, A_2)$ onto A_2. In this sense, A_1 responds to coarse I input features, but restricts A_1 to the finer range ($\ll$ R'). An input feature that is processed in A_1 passes directly over the system $I \longrightarrow A_1 \longrightarrow B_1 \longrightarrow O$, while finer input features must pass over $I \longrightarrow A_1 \longrightarrow A_2 \longrightarrow B_2$ ($\longrightarrow B_1$) $\longrightarrow O$. Since the finer input features are processed over a longer chain of nuclei than the coarse features, their outputs tend to arrive at terminal destinations later than the coarse input features do. In fact, if transmission velocity is proportional to line length, then one can arrange that the finer input features arrive m time steps later than the coarse input features, where m is the number of additional interpolated nuclei that the finer input features must pass through before generating an output. In this way, each step in the input processing system creates a time step in the distribution of relative output onset times, so that the total output is graded in time with the coarsest output features arriving at the output destinations first. The output fields themselves are thus subject to a contraction process within the space of equivalent potential distributions, and this contraction process is made possible by the arrangement of nuclei in hierarchical systems, relative to the peripheral input-output stations, which themselves arise gradually during the evolutionary process, each refined addition an offshoot from its precursor nuclei. One therefore sees that, whereas a very simple input can be adequately processed in the lowest rungs of the input-output hierarchy, most of the inputs which are common in the daily experience of an organism utilize practically all levels of this hierarchy in a nontrivial way. The partition of output lines into pyramidal and extrapyramidal systems becomes correspondingly impossible as a meaningful theoretical device.

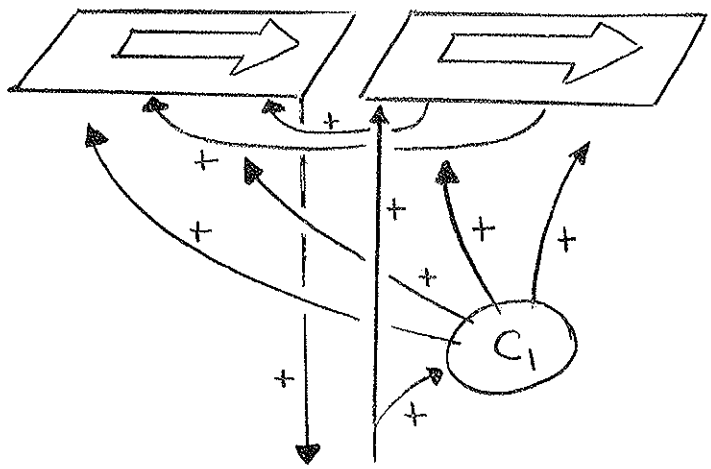
Now consider the diagram



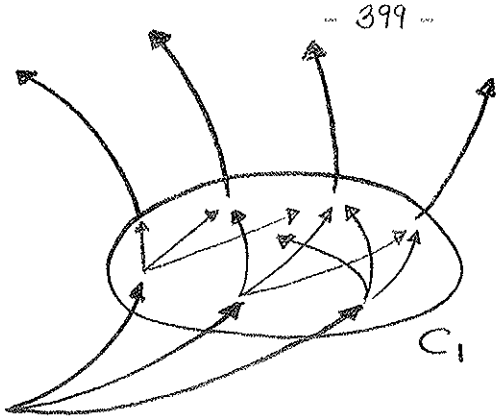
Here A_1 and B_1 have been eliminated, for pictorial simplicity, but the $A_2 \longrightarrow B_2$ connections have been extended. The $I \longrightarrow A_{21} \longrightarrow B_{21} \longrightarrow O$ system of interactions is again the most direct input-output system. Yet gradually, superimposed upon this direct system, are interactions which involve A_2 and B_2 regions which fall nearer the A_{22} and B_{22} borders. These interactions become more pronounced as new embeddings are introduced by spontaneous inversions that are compatible with the $A_2 \longrightarrow B_2$ embedding drifts.

167. Interactions Between Specific and Nonspecific Fields

We now recall that it is necessary for nonspecific inputs to globally activate $A_2 \oplus B_2$ before specific input features reach it. We therefore augment the field with a global activating nucleus C_1:

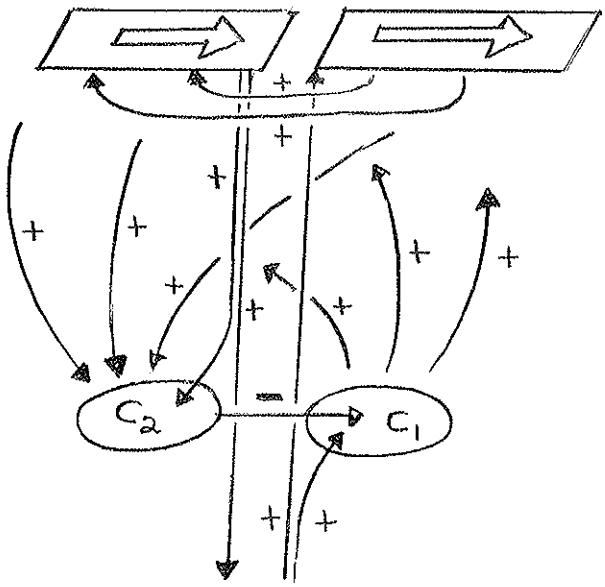


An input from the I system to C_1 globally excites $A_2 \oplus B_2$ through a system of interactions that can be schematically represented by



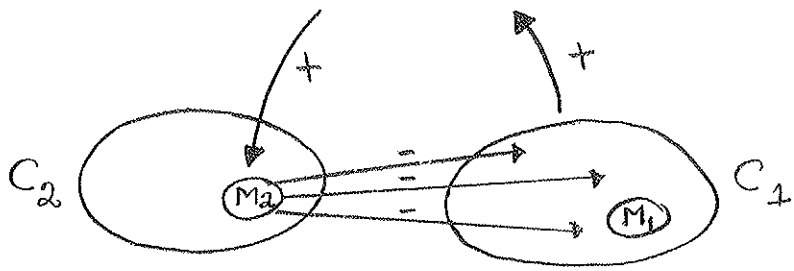
One can think of C_1 as an idealized sector of the midbrain or thalamic reticular formation. It might be expected that C_1 project with least intensity to A_{21}, for A_{21}'s total embedding spaces can thereby come strongly under the control of specific I features. The A_{22} and B_{22} regions can receive a more intense nonspecific C_1 input to prepare these fields for ready response to A_{21} outputs. Indeed, the input from A_{21} must be able to activate control forms that lie ever closer to the $A_{22} \oplus B_{22}$ borders without being seriously hampered by spontaneous strength decay and transmission loses, which must occur as A_{21} excitation spreads over $A_2 \oplus B_2$. The nonspecific C_1 input helps to offset these accumulating strength deficits, which are inevitable when many successive transmissions are required. C_1 thus quite literally has, in this respect, an energizing effect on $A_2 \oplus B_2$. B_{21} can also profit from large C_1 transmissions, both for the reasons cited above and because the very large Betz cells of B_{21} can effectively use large subliminal excitations, without losing their capacity to respond to finely graded input variations.

We must augment this field still further, as follows.



C_2 receives inputs from $A_2 \oplus B_2$ which, in turn, inhibit C_1. These inputs are most profuse from B_{21}, less profuse from A_{22} and B_{22}, and least profuse from A_{21}. Thus, when B_{21} is strongly activated and O outputs are being realized, the nonspecific C_1 inputs are strongly inhibited, since these inputs, though useful in subliminally preparing $A_2 \oplus B_2$ for as yet unknown inputs, must be eliminated if they are not to interfere with the refinements in the space of forms that must precede stable B_{21} outputs. This $A_2 \oplus B_2 \longrightarrow C_2 \longrightarrow C_1$ inhibition process takes place gradually and with increasing intensity as the B_{22} forms are increasingly activated, and the most profuse $\mathcal{L}(C_1, A_2 \oplus B_2)$ terminal loci in $A_2 \oplus B_2$ are the sources of the most profuse $\mathcal{L}(A_2 \oplus B_2, C_2)$ lines.

How "nonspecific" are the $C_1 \oplus C_2$ interactions with $A_2 \oplus B_2$? It is clear that a diagram of the following sort holds:

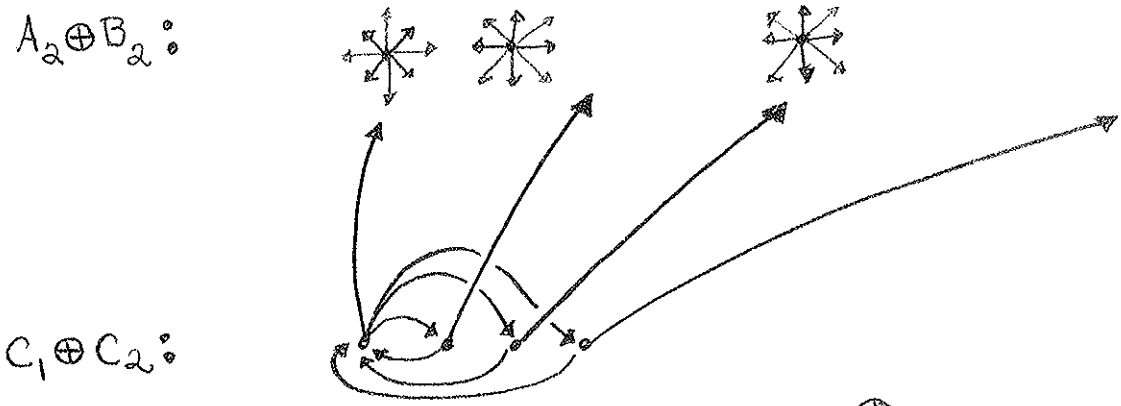


That is, the region M_2 in C_2 most intensely inhibits the complementary region $C_1 \setminus M_1$ in C_1. This complementary inhibition means that as a form in $A_2 \oplus B_2$ becomes increasingly differentially excited, it will more effectively suppress the background excitation in the surrounding regions, and thereby even further differentially peak the local excitatory distribution. This diagram of C_1 and C_2 interactions separates the C_1 and C_2 regions into two disjoint nuclei. Yet the same functional relations can obviously be made to hold if M_1 and its reciprocal M_2 region are closely juxtaposed in Euclidean space. Thus $C_1 \oplus C_2$ can be realized as a set of cellular patches for which an input to one part of a patch can excite a large region of $A_2 \oplus B_2$ cortex, while an input to another, closely juxtaposed part of a patch can terminate this excitation. When nonspecific $C_1 \longrightarrow A_2 \oplus B_2$ excitation is inhibited, it leaves the cells in each cortical cell type in a (statistically) quite uniform state of excitation or inhibition, which is often followed by broad cortical rhythms that depend critically on the far-reaching symmetries that exist in the spatial distribution of cortical cell types.

Notice that the distribution of C_1 and C_2 fibers for $A_2 \oplus B_2$ preserves the symmetry of cortical rows:

Indeed, if the line distributions in $A_2 \oplus B_2$ were entirely radially symmetric and shift invariant, then layers near B_{22} would exert a stronger lateral inhibitory effect on layers near B_{21} than vice versa, and a similar remark holds for A_{22} and A_{21} inhibitions. The embedding drifts counterbalance this inhibitory drift, and the two drifts keep $A_2 \oplus B_2$ rows in a state of tense reciprocal inhibition. Thus, the local embedding drifts of $A_2 \oplus B_2$ are perfectly counterpoised by a drift in the density of "nonspecific" $\mathcal{L}(C_1 \oplus C_2, A_2 \oplus B_2)$ lines. The nonspecific drift is gradually inhibited as specific forms emerge in flows that follow the local $A_2 \oplus B_2$ drift.

It is very important to realize that the distinction between "nonspecific" and "specific" inputs is entirely a relative matter. The C_1 inputs are nonspecific only in the sense depicted by the following diagram:



Thus, points which are strongly coupled by mutual lines within $C_1 \oplus C_2$ often project to points whose interactions within $A_2 \oplus B_2$ are either entirely absent or are only slightly coupled. The strength distributions carried from C_1 to $A_2 \oplus B_2$ are therefore slowly varying relative to the strength distributions which can be achieved in $A_2 \oplus B_2$ alone. It is only in this sense that inputs from C_1 to $A_2 \oplus B_2$ can be said to provide "background excitation" in $A_2 \oplus B_2$. If the range of the $A_2 \oplus B_2$ lines were comparable to that of $C_1 \oplus C_2$ lines, it would be more proper to consider $C_1 \oplus C_2$ as a dynamical double of $A_2 \oplus B_2$ than as a nonspecific source. The similarity of $C_1 \oplus C_2$ and $A_2 \oplus B_2$ structure is further emphasized by the existence of ordered lateral inhibitory lines in both

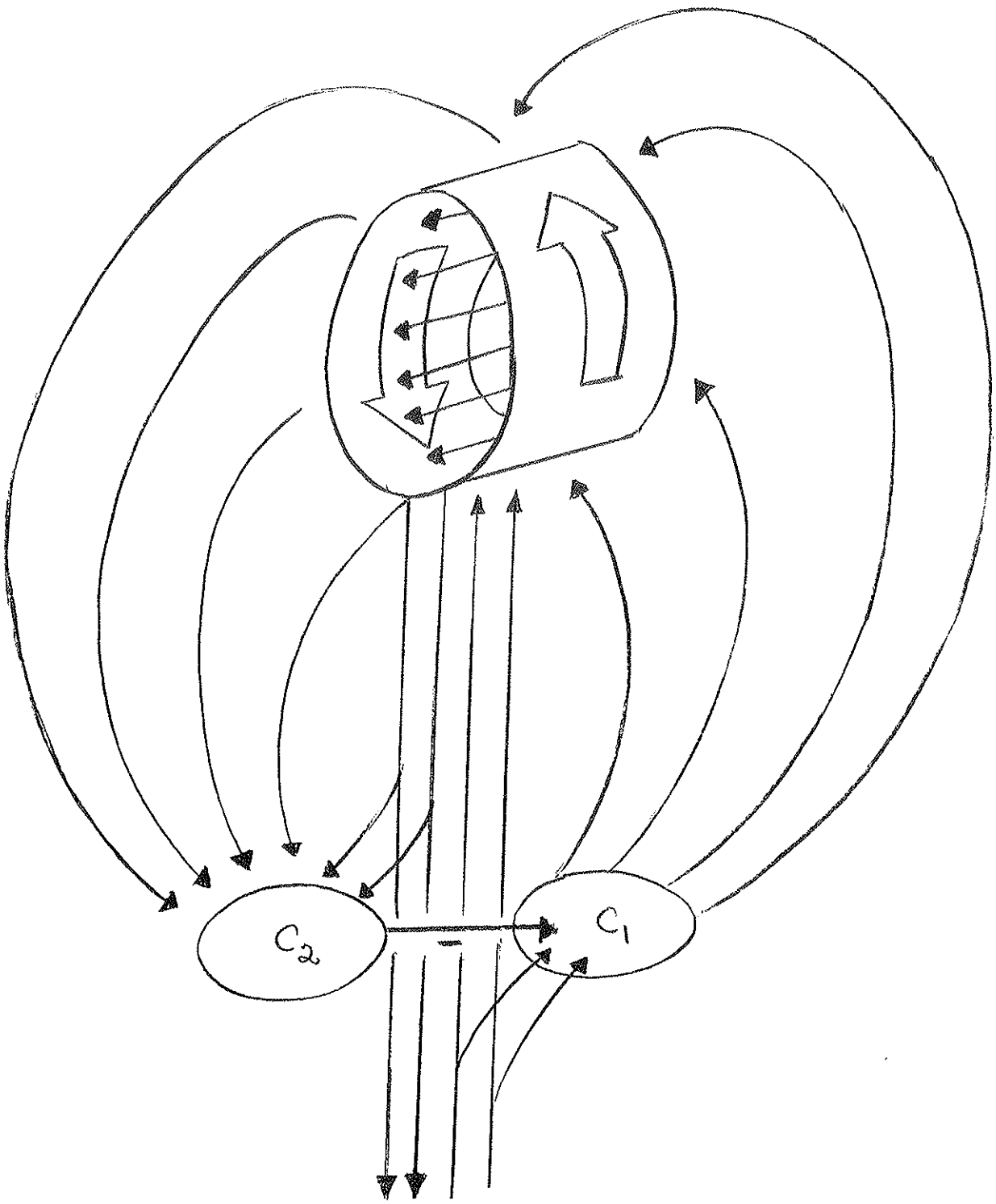
structures. The extension from "on"-"off" to "on"-"off" and "off"-"on" regions is made obvious by field-antifield duality.

The specific subcortical sensory nuclei may be viewed as an intermediate structural type, whose internal interactions are more localized than those of $C_1 \oplus C_2$, modulo projections to $A_2 \oplus B_2$, but are still not usually as highly localized as those of $A_2 \oplus B_2$. This hierarchical evolution of cellular structures with increasingly localized and refined interactions, superimposed upon older and more coarsely grained nuclei, is a geometrical substrate of the ordered dynamical contraction that occurs when any input is processed. To say that a relatively coarsely grained nucleus provides background excitation for a nucleus with more localized lines to which it projects is merely to say that the more localized distribution of lines can distinguish fluctuations within the input which vary more rapidly in space. The ordered lateral inhibitions which occur at successive way stations of ever more finely grained nuclei are ideally suited for the inhibition of precisely those spatially slowly varying input components which can only interfere with the direct processing of the spatially rapidly varying components of the input that fall within the range of the more localized line distributions. Each nucleus thus simultaneously forms an embedded residues of the input fluctuations which can be distinguished by the range of lines which comprise it and eliminates the spatially slowly varying components to provide an output whose spatial range of fluctuation is the same as the range of the lines of the sensory nucleus which will receive this output. In particular, one can envisage a chain of nuclei $I \rightarrow \Omega_1 \rightarrow \Omega_2 \rightarrow \Omega_3 \rightarrow \cdots \rightarrow \Omega_n$ such that Ω_m is particularly responsive to the m^{th} spatial partial derivatives of I. Yet this is but a special case. By properly choosing the distribution of lines, one can construct systems that are sensitive to practically any conceivable human percept or concept. This fact is not surprising once one comes to actually believe that all human percepts and concepts are realized by some process evolving in a suitable field, although the grandeur of our neural endowment makes this belief hard for a modest person to sustain at first, even in principle.

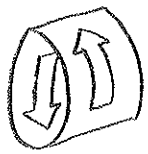
In summary, just as the $C_1 \oplus C_2$ fields "nonspecifically" excite $A_2 \oplus B_2$ as inputs begin to arrive, $A_2 \oplus B_2$ "nonspecifically" inhibits $C_1 \oplus C_2$ as outputs begin to leave. In the interim periods, local $A_2 \oplus B_2$ cell groups interact in delicately balanced excitatory and inhibitory ways which are superimposed on the background potential field induced by $C_1 \oplus C_2$. Yet eventually, the wavelets

exert a strong reciprocal control upon the waves. In more linear terms, a broad spectrum of wavelengths are coupled in $A_2 \oplus B_2$. When one measures these wavelengths in the $A_2 \oplus B_2$ strength field alone, this spectrum is at first restricted to the largest wavelengths, but gradually ever shorter wavelengths, coupled to the longer wavelengths, appear with greater intensity. The long wavelengths are gradually inhibited and one receives the impression of the emergence of a spectrum of wavelengths first sweeping from large to small values and finally peaking on the range of the smallest values that appear. Thereafter, the long wavelengths reappear and all wavelengths are coupled to them as a slow cortical rhythm re-emerged. One can, of course, attempt to associate these wavelengths with suitable eigenvalue problems, but the fact of the matter is that one obviously loses an enormous amount of information in doing so, for one considerably obscures local nonlinearities of the field structure and the structure of the input sources in defining the operators of the problem. One can also do a correlational analysis in the space of wavelengths, where the strength densities become contracted densities in wavelength space. Such an analysis will yield results which have some theoretical relevance, as a comparison of the strong coupling c equations with a correlation function shows, but again, the dynamical mechanisms will not be obvious from such a study, since wavelength space both "flattens" and introduces a nonlocal chaotization principle into the underlying embedding space.

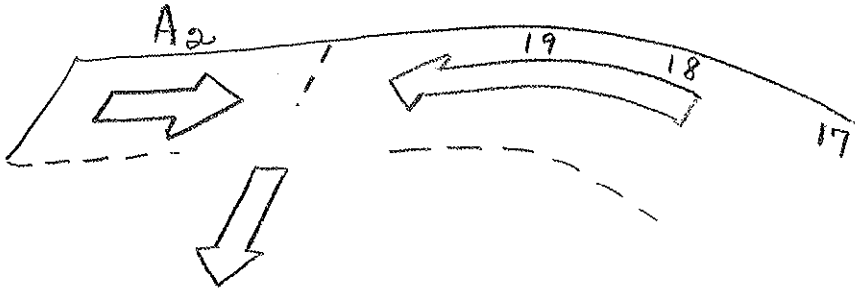
One can see the beautiful symmetry of the $A_2 \oplus B_2$ system by joining A_{2i} with B_{2i}, $i=1,2$. One then has the schematic diagram


C2
C1

It is immediately obvious that one finds the mirror-image picture if all arrows, including the embedding drift arrows, are reversed in this picture; that is, if the input-output relations are exchanged. In this sense, this system is invariant under parity transformations. This invariance depends critically on the fact that the inputs and outputs of this field serve essentially the same muscle groups. That is, parity invariance of the total field and input-output reciprocity go hand in hand. For most input-associational-output systems, it is not true that the muscle groups associated with the inputs and the outputs are the same, and for these systems we cannot expect parity invariance of the above type. We will see many examples of this violation of parity in later sections. Now we merely observe that the oriented tubular field



of the above diagram does not hold even in the idealized sense here considered, precisely because $A_2 \oplus B_2$ interacts with fields that violate parity, and $A_2 \oplus B_2$'s local tubular structure is deformed by these violations. Indeed, sugmenting A_2 is the set of visual associational fields, and these fields possess an embedding drift that has an orientation opposite to that of A_2:



Details of these asymmetries will be provided in later work.

The above observations can be applied wherever pairs of fields interact with varying degrees of line specificity. In particular, we now combine these observations with our remarks on $7_0 \leftrightarrow 7_1$ interactions to conclude that the presentation of inputs representing well-embedded 7_1 forms will rapidly cause a suppression of nonspecific centers to permit the asymptotic processing of specific embedded forms. When no strongly embedded 7_1 forms are activated by the input, nonspecific influences can persist over a longer time interval. Similarly, one sees that broad Euclidean sectors of even somewhat more specific field regions will be either actively suppressed or freed from nonspecific

excitation due to the reciprocal inhibition which differentially excited specific cellular regions exert on appropriate nonspecific sources.

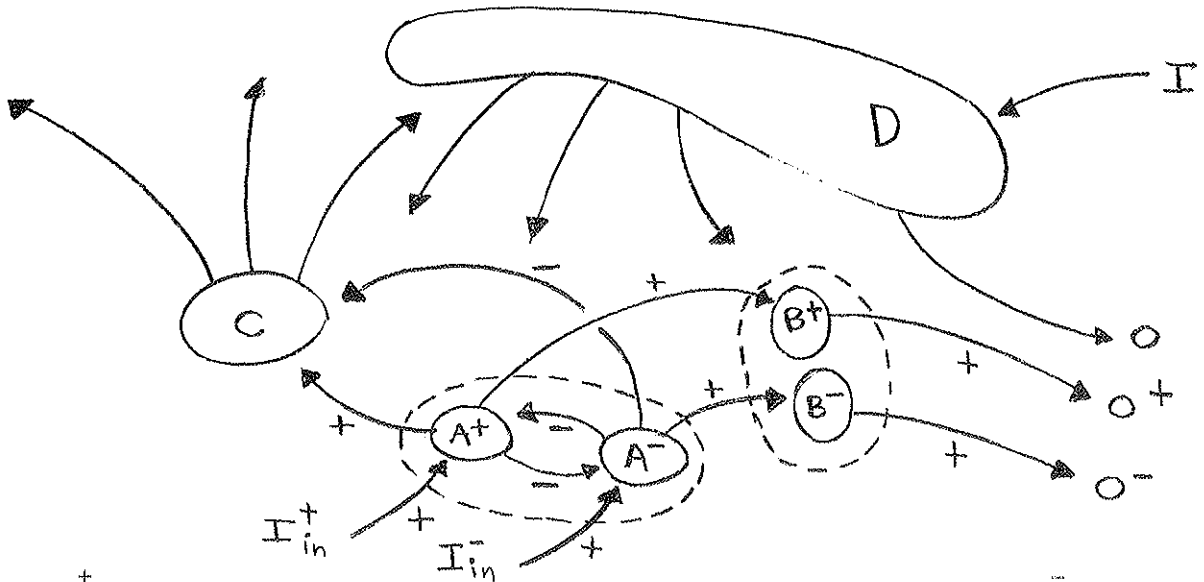
It is important in discussing such specific-nonspecific interactions to always keep in mind that the total hierarchical arrangement of the nervous system is organized relative to the nonneural peripheral input-output centers, which form the only natural boundaries of 7.* Physiological inputs to a higher center must usually pass through phylogenetic precursors of this center, and many of the most important output centers lie in the phylogenetically old regions, whence strong couplings from even the highest centers must often form links--perhaps in several synaptic stages--with the older regions. In particular, it is naive to expect the removal of a neocortical region of moderate size to always render impossible new embeddings within phylogenetically old behavioral modalities. The entire spectrum of ongoing inputs which survive inhibitory renormalizations simultaneously form associational couplings whenever their trajectories overlap. Since the highest neocortical fields, such as the 7_o fields, were superimposed upon thalamic fields through evolution, there must exist subcortical analogs of the 7_o fields, in addition to specific sensory nuclei. The reasons for this are essentially the same as those which motivated the construction of the 7_o fields themselves; loci from which many specific modalities can be activated must exist if experiences are to be bound into an integrated unity. Thus, it is partly through thalamo-cortical interactions, say with the pulvinar, that such 7_o fields as the temporal fields come to interact with many 7_1 fields, such as the visual associational fields, and without the time biases that are inevitable when the surface topology of neocortex is not violated by action at a distance. Similarly, such subcortical 7_o analogs provide associational routes by which neocortical regions establish directed coupled paths to hypothalamic and other centers of primary importance to the regulation of the substrates of behavior. Finally, as we have already observed, some inputs bypass neocortex almost entirely and are almost completely processed at thalamic or even lower centers. An immediate consequence of these remarks is that the contracted locus of a neocortical embedding must also very often involve thalamic centers in a direct way. A neocortical embedding is useless and impossible to establish unless it is coupled to some source of prior embeddings which, even though by a series of very indirect steps, was constructed over a skeleton of quite direct input-output systems. It is, in particular, easy to understand from this perspective how a (nonspecific) subcortically induced DC

potential shift on the neocortex can be strongly coupled with a specific, properly polarized, electrical input to the neocortex itself. Indeed, one can understand hundreds of the published reports on EEG and direct electrical activation studies on animals by a direct application of these ideas.

168. Operant Conditioning, Motivation, and Homeostasis

One particularly important aspect of the control by phylogenetically old centers of even neocortical structures involves the study of positive and negative "reinforcements," or rewards and punishments. Many of our previous remarks have tacitly assumed that an input was either rewarding or, at least, neutral in its reinforcing properties. For we have usually permitted our inputs to reach the field destinations imagined in our thought experiments, and have supposed that nonspecific inputs are excitatory. In effect, therefore, we have usually assigned a "+" sign to our inputs. In discussing negative reinforcers, we must assign a "-" sign to our inputs in an appropriate way. The distinction between positive and negative reinforcers will thus involves some kind of field-antifield duality. Our discussion here will be little more than a sketch of how to subsume the operant conditioning situation under previously developed ideas. The details involve many intricate field constructions. Since so much has been written about positive and negative reinforcers, we assume that the distinction is reasonably well-known to the reader, at least in a macroscopic operational sense, and proceed to mention several neural mechanisms that have the major properties of these operational distinctions. Along the way, we will briefly discuss such matters as the concept of "motivated states," and of the manner in which (roughly speaking) positive reinforcers increase the probability of an ongoing behavioral sequence while negative reinforcers decrease its probability. After all of our previous preparation, we can pose these matters as questions whose form immediately suggests the appropriate mechanisms. Briefly put: How can we assure that directed systems of strong couplings are established between behavioral sequences and sources of positive reinforcers when the "drives" underlying the positive reinforcers are not satiated? How does the strength of such a coupling vary with the intensity of the drive state? How can we assure that such systems are not established between behavioral sequences that intensify sources of negative reward and centers of positive reinforcement?

A primitive mechanism that has many of the desired properties is

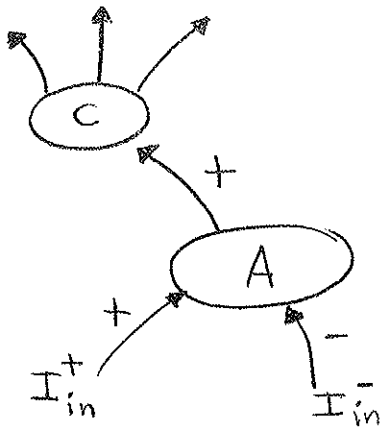


Here I^+_{in} represents a source of positively rewarding inputs while I^-_{in} is a source of negatively rewarding inputs. It might seem somewhat unsatisfying at first, in discussing what positive and negative rewards are, that one is given inputs which one labels from the beginning as positively or negatively rewarding. Such a labeling is, however, inevitable. Although we are accustomed to speaking of positive and negative rewards as being separate entities, in the nervous system one can only speak of systems of cell groups, what inputs reach them, and how they interact to produce outputs. A splitting of certain of these cell groups into positively and negatively rewarding systems exists from the beginning of an animal's life, as part of its fundamental preparations for survival, and is accompanied by a comparable splitting of inputs and outputs. Such a splitting is a "given" of the situation. Our present task reduces to: given such a system, how do new control forms become coupled to it? The positive-negative distinction itself can be further analysed only by studying the biochemical specificities that generate it.

We must translate notions of "drive state" or "motivation" into neural terms. It is incorrect to believe that the concept of "drive" underpins a universal neural process. "Drive" is merely a rubric for the realization that certain behavioral systems become deactivated after appropriate energizing correlates of these systems are satiated by the activation of suitable behavioral and/or chemical sequences. Every such system exhibits special specificities and structural arrangements. For example, in the diagram $A=A^+ \oplus A^-$ is an associational field receiving the internal inputs I^+_{in} and I^-_{in}. I^+_{in} and I^-_{in}

both represent particular aspects of a certain homeostatic mechanism within the organism. For specificity, suppose that blood vessels in the region of A break up into glomeruli of thin-walled capillaries that permit diffusion of blood chemicals associated with this homeostatic mechanism directly to A, and that the A cells are sensitive to the density of these chemicals in the vicinity of their cell membranes. I^{+}_{in} (I^{-}_{in}) measures the input to A cells of a chemical which exists in the bloodstream in greatest profusion when a deficit (surfeit) of the homeostatic mechanism exists. One can think of these inputs in terms of a chemical correlate of a foodstuff which forms a coupled system with a bodily produced chemical, for specificity. A^{+} and A^{-} laterally inhibit one another, so that the output from A to C measures the net imbalance of I^{+}_{in} and I^{-}_{in} in the bloodstream.

In the diagram, we have split the I^{+}_{in} and I^{-}_{in} sensitive cells into A^{+} and A^{-} cell groups. We could just as easily have envisaged the diagram



in which every A cell is sensitive to both I^{+}_{in} and I^{-}_{in}. Here, I^{+}_{in} and I^{-}_{in} have reciprocally polarized effects on the A cell membranes, so that all lateral inhibitory effects occur, not between cells, but within individual cell membranes and the interior cell systems that are coenzymatically coupled to the membrane polarity and permeability.

When the (foodstuff-derived) chemical deficit exists, A^{+} is activated and sends excitatory inputs to B^{+} and to C. C can be thought of as a nonspecific activating nucelus, whereas B^{+} is a nucleus carrying control forms of primitive behavioral acts whose performance enhances the animal's opportunity to reduce the homeostatic deficit. For example, in an absence of food, B^{+} might control peripatetic approach and sniffing outputs. C's activation excites fields which subserve behavior that also conduces to the reduction of the I^{-}_{in} deficit and inhibits nuclei subserving incompatible behavior. As I^{+}_{in} increases, with no

corresponding increase in I^{-}_{in}, this C inhibition increases and the animal's behavior is more completely channeled into behavioral outputs which tend to equalize the (I^{+}_{in}, I^{-}_{in}) imbalance. Such a competition for the animal's attention and behavioral output is simultaneously ongoing for many systems of internal and external inputs, each system being particularly responsive to a special class of inputs and their imbalances. As usual, the animal is spared from a massive flooding of his neural fields with behaviorally chaotic excitation by reciprocal lateral inhibitory structures--such as those connecting A^{+} and A^{-}--which permit only the most intensely excited nuclei to reach a behaviorally significant level.

The animal's focalized behavior induces new external inputs. For example, visual inputs (of food) representing an indirect source of I^{-}_{in} will project to B^{+} and associated nuclei and intensify the animal's running behavior to the goal object at the same time that nonspecific mechanisms compatible with the visual inputs, and specific interpretative embeddings, are also excited. These various excitatory loci of compatible behavioral outputs and internal inputs in $B \oplus C \oplus D$ will all be excited as the goal object is attained. The situation is therefore perfectly suited for the formation of strong couplings between all of the excitatory loci that project to one another and which have been relevant to the attainment of the goal object. After I^{-}_{in} and other goal specific inputs increase, the "drive" induced by a superfluous I^{+}_{in} input is laterally inhibited and other, more "salient", drives emerge into behavioral relevance. On a later occasion, the differential excitation of A^{+} by I^{+}_{in} will induce behavioral sequences that tend to reproduce the sequences which have successfully led to an improvement of (I^{+}_{in}, I^{-}_{in}) balance in the past. Successive trials will contract the forms leading to successive I^{+}_{in} "drive reduction" relative to one another, so that the animal's behavior will appear more "purposive" and efficient with increased training.

Thus, the situation of positive operant conditioning is here made possible by the establishment of spontaneous hierarchical inversions which are initially activated by homeostatic mechanisms necessary to the survival of the organism and which are ultimately suppressed only after the arousal of overt behavioral sequences that make homeostatic supplements available to the organism. After new embeddings of this type form and project to the homeostatic regions, one can, in the usual way, establish still further embeddings which control these new embeddings. Such multiple control hierarchies usually go under the rubric of secondary reinforcers. In short, the most highly "reinforcing" inputs will be

those that arrive when the strongest couplings with the nuclei subserving the "reinforcing" modality are possible. The vital point is that both respondant conditioning and positive operant conditioning depend on the formation of strong couplings which underlies spontaneous hierarchical inversions. The two processes differ only in terms of the special fields that are activated, including the motor fields that actively carry the organism to the completion of input-output circuits which are mediated by internal homeostats as well as by fluctuations in peripheral inputs.

How does negative operant conditioning work? We must obviously seek an antifield analog of positive operant conditioning. In particular, an "aversive" input should inhibit the nonspecific fields which a "rewarding" input excites. For example, in the above diagram, increasing I_{in}^{-} inhibits the C activating system, at the same time that activating A^{-} generates B^{-}-controlled behavioral outputs that are well suited to re-establish homeostatic equilibrium. If B^{+} controlled running towards a goal object, B^{-} would most likely control running away from it. Inputs to D that occur when A^{-} and B^{-} are active will form strong couplings with these nuclei, and not their associated reciprocal A^{+} and B^{+} nuclei. In particular, the sight of an environmental situation which has become coupled to A^{-} or B^{-} will initiate a fleeing, or avoidance, response whose intensity varies directly with the strength of the coupling. It is important to realize that a new input type cannot become coupled to either A^{+} or A^{-} unless these nuclei are differentially activated by an independent input source. One cannot build any differential affective responses in an (external and internal) environment for which all inputs have a neutral affective status. Unfamiliar inputs achieve affective status only by being "compared," though strong couplings, with nonuniform distributions of reactivated, familiar inputs with a nontrivial affective projection. If some input to D is associated with an increase in an "aversive" input, say I_{in}^{-}, then the D input will become more strongly coupled to nuclei, such as B^{-}, which subserve avoidance and other negative behavior, while C even further inhibits the systems which are usually coupled to $A^{+} \oplus B^{+}$. On the other hand, if a particular set of D inputs is associated with a rapid decrease in I_{in}^{-}, A^{+} will undergo an inhibitory rebound and the dynamical stage will be once again set for strong couplings of this input with systems of $A^{+} \oplus B^{+}$ type. Such a deactivation of aversive inputs is therefore also a form of rewarding drive reduction. The term "drive reduction" is, however, quite misleading, for

the strong coupling process can only be clearly understood if one considers a delicate balance between antagonistic "drives," and when one such "drive" is reduced, a complementary "drive" is increased.

As the animal's behavior changes the relative position of his peripheral receptors with respect to particular environmental features, the distribution of inputs will constantly change their locus in a compensatory way. Whereas a particularly primitive A^+ nucleus might initiate a pattern of diffuse exploratory behavior, the effects of this exploratory behavior on new inputs can activate more specific fields--say those controlling salivation and other preparations for eating behavior--which, in turn, alter the pattern of behavioral outputs. Every integrated behavioral act of an animal thus involves the ordered activation of a large number of fields which differ in their specificity, complexity, and phylogenetic recency. The analogy with the processing of inputs along chains of specific sensory nuclei is obvious, yet such processing is but a small subsystem of the animal's total behavior. The extraordinary complexity of the total field activity is one reason why it is often useful to study spaces of contracted control forms, rather than 7* distributions, when considering the freely responding organism from a psychological viewpoint.

It is hoped that these remarks suffice to qualitatively illustrate the basic fact that both respondant and operant conditioning, of all kinds, are subsumed under a study of the field renormalizations which permit the formation of directed systems of strongly coupled hierarchical inversions, while the study of "motivation" reduces to an investigation of fields which are selectively responsive to homeostatic mechanisms and are often equipped with delicately balanced reciprocal inhibitions whose special features vary with the homeostatic mechanism that they favor. Details of field structure will be discussed somewhat further in the following pages, and in later works.

In spite of its necessity, at first broach the representation of painful or rewarding inputs as inputs to conveniently placed nuclei might seem to be a somewhat disappointing solution to the problem of representing emotional behavior. One can only observe that such a representation is made possible by special properties of the theorist's cognitive fields which derive part of their flexible symbolic power from their very independence of strong interactions with emotive fields and the relatively automatic response patterns that emotive fields activate. All is not lost, however, for the highly motivated reader can

train himself to feel, in small measure, the affective experience which is represented by inputs such as I_{in}^{+} and I_{in}^{-}. To do this, the symbols "I_{in}^{+}" and "I_{in}^{-}" become the inputs, received by the reader's eyes, and the reader can try to establish strong couplings between these symbols and those fields of his which subserve the appropriate affective response. Although such an attempt at self-training must be viewed as slightly fanciful, it is indeed true that the formation of such couplings constitutes one method of establishing intuition into the workings of emotive fields. Here is therefore one of those rare opportunities for which, in principle, the reduction of an abstract variable to a sensual level actually enriches the direct meaning of the variable, and is now merely an itemization of special cases.

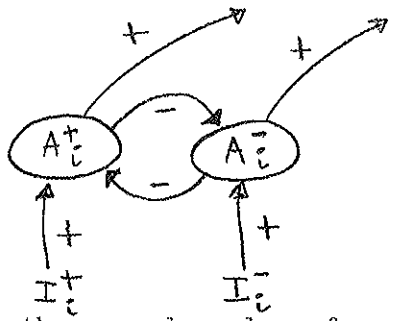
169. Stimulus Sampling Theory

How does stimulus sampling theory fit into this picture? The sampling theory provides a highly limited representation of what is really happening neurally. Indeed, one cannot find "stimulus elements" in the brain. The nearest that one comes to the idea of a "stimulus element" are the control forms that represent contractions of excised dynamical subtrajectories. Moreover, no "sampling" operation occurs which links the uninterpreted "stimulus points" to one another. Rather, external or internal inputs excite appropriate control forms which gradually establish broadly distributed strong couplings in a manner that depends on the particular input sequence and on the initial state of 7^{*}. A major contribution of stimulus sampling theory to psychological thought, in addition to its initial success as a mathematical model of a biological phenomenon, is its effort to represent quantitatively, through its postulates for the rearrangement of sets of stimulus elements, the fact that one cannot merely fill psychological time with blanks. The absence of an experimental manipulation is as essential to determining the state of 7^{*} as is a veritable barrage of experimental inputs. Nonetheless, the point space and the operations of stimulus sampling theory simply do not provide an interpretation of what is really happening when learning occurs. Its axioms do not embrace any of the truly interesting phenomenological substrates of adaptive behavior. It is true that instead of the formally very pleasant homogeneous set of "stimulus points," we must, in the context of the theory of embedding fields, study highly inhomogeneous field constructions. Since geometry and dynamics are strictly dual

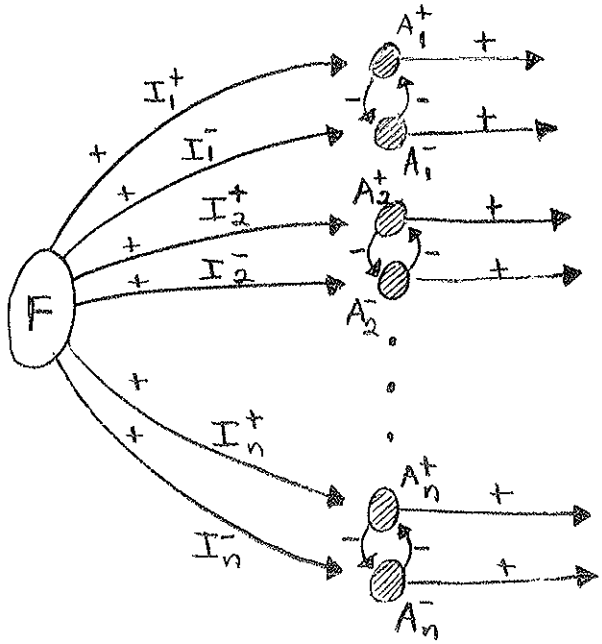
in neural fields, such constructions are, nonetheless, an inevitable precursor to deep insight. Moreover, we may be consoled in contemplating these constructions by having already seen a glimpse of the very beautiful symmetries and dualities which underpin embedding fields on every level of their organization.

170. Scales, Temporal Masking, and the Law of the Excluded Middle

One of the most pervasive features of the embedding field situation is the symmetry of excitatory and inhibitory processes. This symmetry exists not only on the local cellular level, say in the equations for an idealized volume conductor, but also in the construction of cellular ensembles according to a principle of field-antifield duality. In particular, we envisaged homeostatic mechanisms whose readiness to offset momentary chemical imbalances is counterpoised by mechanisms of reciprocal inhibition that prevent the emergence of floods of accumulating background excitation. The implications of such widespread symmetries between excitatory and inhibitory processes are profound, and are in ready evidence in a broad variety of our daily experiences. For example, suppose that a homeostat is realized as in the diagram

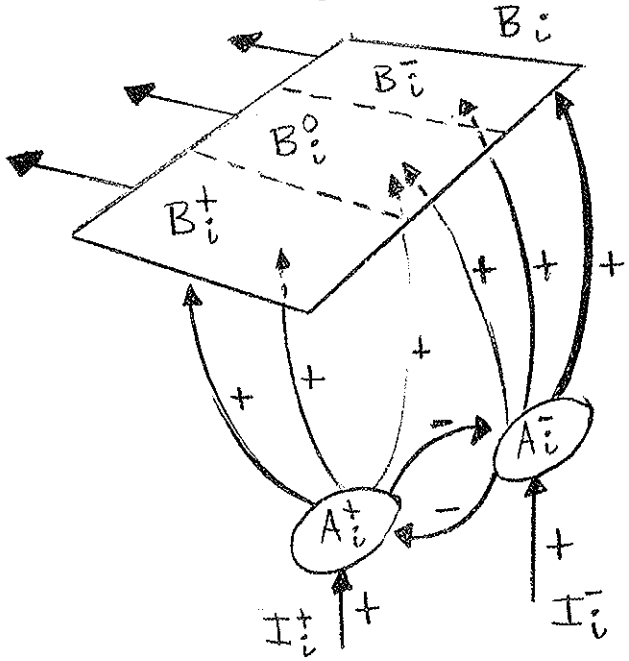


The system $A_i^+ \oplus A_i^-$ induces the neural analog of a one-dimensional scale by which to measure the system (I_i^+, I_i^-). Similarly, given a nucleus F for which



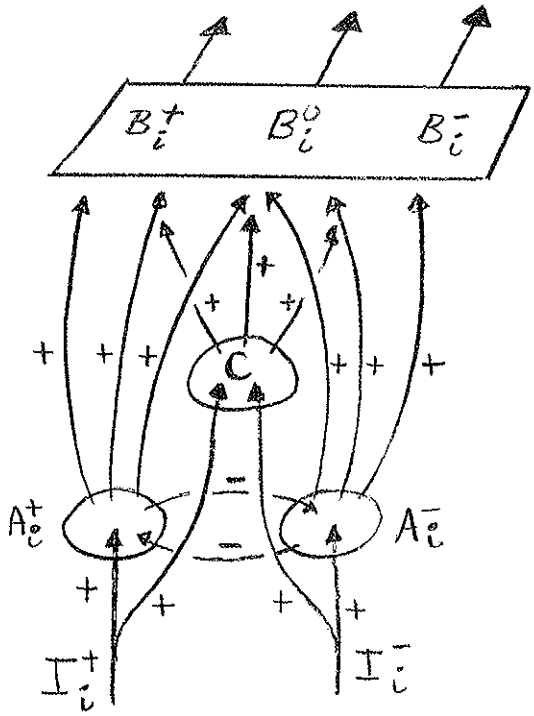
where the $A_i^+ \oplus A_i^-$ are pairwise disjoint and disconnected, then $\oplus_i (A_i^+ \oplus A_i^-)$ is an n-dimensional scale for F. The significance of the dimensionality of a scale is obvious from the diagram.

It is, in the strictest sense, somewhat meaningless to discuss a scale separate from the entire set of fields to which it projects, especially the output fields. For the output fields which are capable of discriminating fine variations in scaling fields often have nontrivial feedback effects on their sensitivity, as we shall presently see. Although the dimensionality of a scale sets the upper bound on the fineness of the structure of the fields of distinguishable forms activated by the scale, the lower bound can be effectively zero in degenerate cases. To illustrate the situation in a simple case, consider the idealized diagram



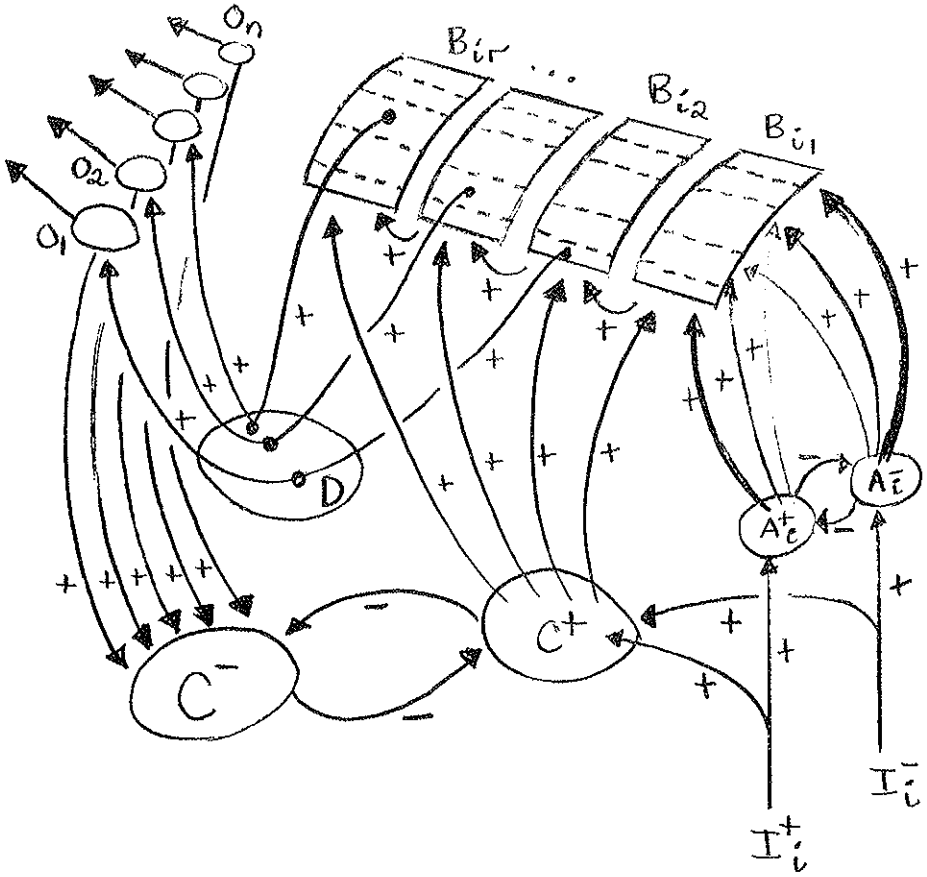
Here $A_i = A_i^+ \oplus A_i^-$ projects to the associational field $B_i = B_i^+ \oplus B_i^o \oplus B_i^-$. A_i^+ (A_i^-) projects primarily to B_i^+ (B_i^-), while both A_i^+ and A_i^- send some lines to the B_i^o region. As usual, we fix the $A_i \longrightarrow B_i$ transmission thresholds to assure that stable $\mathcal{J}(A_i)$ values are realized before intensive transmission occurs. When A_i^+ (A_i^-) is strongly activated and A_i^- (A_i^+) suppressed, B_i^+ (B_i^-) is strongly activated. When the excitation delivered to A_i^+ and A_i^- is suprathreshold and approximately equal, transmission to B_i will be reduced, but field constants can be set to permit some $A_i \longrightarrow B_i$ transmission at this time. In this case, it is easy to ensure that B_i^o is excited more than either of B_i^+ or B_i^-. Thus, the locus of maximal B_i excitation moves across B_i from B_i^+ to B_i^- as the ratio $I_i^-/(I_i^+ + I_i^-)$ increases from 0 to 1. Local lateral inhibitory lines within B_i help to peak this locus of maximal excitability and to suppress the excitatory surround.

We have, however, run into a difficulty, for although B_i^o is maximally excited when both A_i^+ and A_i^- sustain equal excitations, the absolute magnitude of $\mathcal{J}(B_i^o)$ is relatively small. It is easy to partially offset this difficulty. We merely augment the field with a nonspecific excitatory field C_i for B_i:



Now the absolute magnitude of the inputs I_i^+ and I_i^- sets the excitatory background magnitude of B_i so that the A_i inputs can induce B_i outputs with less difficulty, even in B_i°. The localized maxima of B_i excitation thus generate outputs which discriminate between various relative settings of $\mathcal{L}(A_i)$ values. This output mechanism can be very much improved. In particular, fields B_{ik} can be attached in series to $B_i = B_{il}$ $(B_i = B_{il} \rightarrow B_{i2} \rightarrow \cdots \rightarrow B_{ir})$ to accentuate these local maxima without disrupting their relative position on successive B_{ik}. As an application of our construction of one-many fields, one sees that a B_{in} field can be approximated, taking advantage of an admixture of nonspecific excitatory inputs, localized lateral inhibitions, and position preserving $B_{ij} \rightarrow B_{i,j+1}$ transmissions, for which all supraliminal $\mathcal{L}(A_i)$ settings generate large localized $\mathcal{L}(\oplus_j B_{ij})$ values.

Actually, one can partition B_i into more than three parallel rows; the number of rows depends on the spatial gradient of the $A_i \rightarrow B_i$ lines relative to the coarseness of the B_i internal line grain in an obvious way. Such an $A_i \oplus B_{il} \oplus C_{il} \oplus B_{i2} \oplus \cdots$ field can now be used to deliver stable inputs to an output field. Consider, for example, the simplified diagram



Here $B_{ij} \longrightarrow B_{i,j+1}$ transmissions are directed along parallel rows. All of $\oplus_j B_{ij}$ projects to the (subcortical) associational nucleus D, which sends lines to the language control forms $O_1, O_2, \ldots, O_{n_k}$. Denote the row in B_{ij} associated with O_k by this transmission path by B^k_{ij}. The total projection domain to O_k from $\oplus_j B_{ij}$ is therefore $\oplus_j B^k_{ij}$. Successive $B_{ij} \longrightarrow B_{i,j+1}$ transmissions tend to peak the excitation in the intermediate rows which are excited by $\mathcal{J}(A_i^+) \approx \mathcal{J}(A_i^-)$ values; namely $\left\{\oplus_j B^k_{ij} : k \approx n/2\right\}$. The extremal rows $\left\{\oplus_j B^k_{ij} : k \approx 1, n\right\}$ are also peaked by such transmissions. When these peaking effects reach sufficiently high supraliminal values, the excitation is conveyed to suitable O_k forms. The O_k, in turn, both generate behavioral outputs and project to a nonspecific C^- nucleus which, in the usual manner, inhibits C^+. One can, for example, think of O_1 as a control form for the word "hot," and O_n as a control form for the word "cold." Intermediate $\oplus_j B^k_{ij}$ rows map onto control forms for intermediate sensations of warmth or coolness. The particular output words chosen are not critical in asymptotic studies.

Notice that the extremal rows can achieve high strength values after a relatively small number of successive $B^k_{ij} \longrightarrow B^k_{i,j+1}$ transmissions. Thus the O_i, $i \approx 1, n$, can be effectively excited shortly after I^+ and I^- occur. Once a $B^k_{ij} \longrightarrow D \longrightarrow O_k$, $k \approx 1, n$, path is excited, C^- is also activated, and the ensuing inhibition of C^+ renders further transmissions in $\oplus_j B_{ij}$ difficult, especially for the sectors $\left\{B^m_{iw} : m \approx n/2,\ w \gg 1\right\}$. In this sense, A_i-proximal, extremal B^k_{ij} sectors temporally mask A_i-distal, intermediate B^m_{iw} sectors. Before effective $B^m_{iw} \longrightarrow D \longrightarrow O_k$ transmissions can occur for $m \approx n/2$, a larger number of successive $B^m_{ij} \longrightarrow B^m_{i,j+1}$ transmissions must ordinarily occur than are necessary when $m \approx 1, n$. The $\oplus_j B^k_{ij} \longrightarrow D \longrightarrow O_k$ transmissions for $k \approx n/2$ are therefore harder to establish and have a longer reaction time than the $k \approx 1, n$ transmissions.

Thus, (1) the relative imbalances in a simple system of reciprocally inhibiting cell groups can be made to induce localized excitatory loci, (2) these excitatory loci can be transformed successively to large excitatory values that preserve their relative local position while offsetting energetic deficiencies in the intermediate positions, and (3) the indistinguishable packets modulo A_i, namely $\oplus_j B^k_{ij} \oplus O_k$, can be mapped in an order-preserving way on a one-dimensional continuum. (3) underlies the overt behavioral expression of the latent one-dimensionality of the "scale" A_i. Neurological scales are, as usual, more subtle, but one can go far towards understanding them using the ideas of this example.

One pervasive feature of our construction is (4): The intermediate values of the scale--those associated with the $\{\oplus_j B^k_{ij} : k \approx n/2\}$ rows--pose particular difficulties. Since they are less easily discriminable, modulo $\oplus_i O_i$, than are the extremal rows, they require more $\oplus_j B_{ij}$ processing than do the extremal rows. A familiar behavioral manifestation of this difficulty is that children often learn to distinguish the extremes of a scale--the "hot" and "cold" ends--before they can accurately apply intermediate words--"warm" and "cool." To the extent that a scale exhibits one-dimensional symmetry of the above type, we thus expect the extremal positions of the output field to be most easily discriminated and most readily exhibited in overt behavior.

The "hot"-"cold" scale is not the only one which has a latent one-dimensional character. One need only consider the scales

1) hot--warm--cool--cold
2) yes--maybe--no,
3) happy--sad
4) good--bad
5) true--false

to realize this. Each scale, of course, varies in complexity and in the structure of its underlying field and input sources. What is important, however, is the clear tendency for extremal values to predominate, sometimes to the exclusion of intermediate values. It is particularly significant that some of the socially most important scales--such as (4) and (5)--have often been restricted to extremal values for long periods of time. Many overlapping reasons underly the primitive structure of these social scales, but perhaps the most unifying reason is that, when intermediate values are difficult to discriminate, restricting the behavioral

outputs to extremal scale values stabilizes the social interactions which are relevant to the scales in question. In (4) and (5), where the social consequences of poor discrimination are of great importance, one can intuitively see without difficulty the value of initially restricting overt behavioral outputs--which are the primary social data--to easily discriminated extremal values. All of these remarks on stability can be interpreted in terms of the simple fact that when a given behavioral discrimination must be propagated by many individuals to many other individuals, discriminatory nuances tend to be averaged out. Moreover, in scales such as (2), the extremal values are coupled to control forms which activate an entire behavioral sequence in many cases, and whether or not this entire sequence will be activated depends wholly on which extremal value is excited. The intermediate values for such a scale actually depict an oscillation between the two extremes until one of them becomes stabilized and the appropriate behavioral sequence, or its firm absence, becomes evident. We will also shortly see--in the next two sections--another mechanism whereby "important" social distinctions--those which relate to basic individual "drives"--can be restricted to a small set of output alternatives. On a societal level, the number of overt behavioral signs of a given scale may thus be roughly viewed as a joint measure of the ease with which scale inputs can be discriminated by individuals, and of the value to an individual, in a particular environmental and social context, of being able to emit overt behavioral signs of the discrimination. The study of socially stable scales--both cognitive and emotive--requires a study of strong couplings between individuals in a particular environmental and social (input) context.

An immediate consequence of these remarks is that a multi-dimensional scale can usually discriminate many more features of the total input array than a one-dimensional scale can. Although in a primitive social context the restriction of outputs to low dimensional scale sources increases societal stability, it also limits the capacity of the individual organism to make subtle discriminations of his total input manifold, unless the individual frees himself from the reward system underlying the socialized discriminatory pattern. The tendency for this to happen varies directly with the individual's capacity to make discriminations which are socially unacceptable but nonetheless, for one reason or another, clearly visible to him. Moreover, in a society for which many

discriminations in a given scale are required, an individual who cannot make these discriminations effectively contracts many socially inequivalent signs into equivalent (indistinguishable) packets of neural forms. Outputs based upon these socially unrecognized equivalences can easily increase the social instability of the poorly discriminating individual, especially when certain of these signs are considered socially acceptable while others are viewed with contempt. One can extend these remarks in a natural fashion to achieve a rather general discussion of social interactions and mobility patterns. We leave the details to a later work.

The present remarks on social interactions are concluded with the observation that the choice of a particular social scale can neither be said to be natural or unnatural in a social and environmental vacuum. Nonetheless, such choices are not matters which need properly be relegated to mere whimsy. For every individual scale has a natural embedding in a particular neural frame, which is a given of life. It is only in terms of the consequences for the total neural endowment of every individual, in a given context, that the choice of social scales can be evaluated.

171. Behavioral Regression and Anxiety

Two further remarks of importance to the psychology of individuals can be read from the above diagrams. The first hinges upon our observation that extremal B_{ij}^{k} sectors with small j values can temporally mask intermediate B_{iw}^{m} sectors, $w >> 1$, via $D \rightarrow O_k \rightarrow C^- \longrightarrow C^+$ transmissions. Since the B_{ij}^{k} sectors induce easily discriminable and relatively primitive outputs compared to the B_{iw}^{m} sectors, the $B_{ij}^{k} \longrightarrow C^+$ suppressive sequence can be viewed as a latent source of regressive behavior. Indeed, suppose that the organism is subjected to a high level of generalized anxiety, which induces a strong nonspecific $C^+ \longrightarrow \oplus_j B_{ij}$ input. It is clear that the B_{ij}^{k} sectors will be rapidly activated and will temporally mask the B_{iw}^{m} sectors. Since the B_{iw}^{m} sectors gained control over overt behavior later than the B_{ij}^{k} sectors did, one receives the impression that older behavioral types have precluded the occurrance of more recently emerging and subtle behavior, as indeed they have. Hence, the appropriate term behavioral regression. We can immediately see that a similar mechanism underlies all regressive behavior. Whenever nonspecific or coarsely grained cellular systems

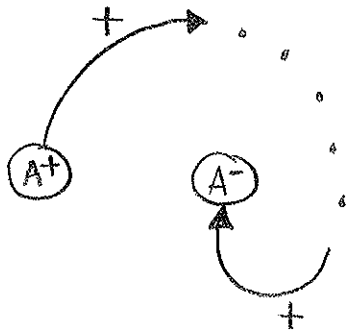
are very strongly activated, inputs arriving from the periphery will be channeled--as they are in the above example--through the phylogenetically older, and more stable, fields that subserve the more primitive and autistic behavioral types. The stronger is this nonspecific barrage, the stronger is the truncation of the input flow to direct input processing systems. When the internal sources of nonspecific inputs become very intense, practically all higher input modalities can be dynamically masked, and the organism retires into a state of highly primitive stereotyped behavior. For example, in our study of the (charicature of a)sensorimotor field, the frontal inputs will be increasingly masked as the effective inputs are contracted to the $I \longrightarrow A_1 \longrightarrow A_{21} \longrightarrow B_{21} \longrightarrow B_1 \longrightarrow O$ system, then to the $I \longrightarrow A_1 \longrightarrow B_1 \longrightarrow O$ system, and so on. To the student of the evolution of species, the introduction of intense nonspecific inputs has the fascinating, though sobering, aspect of a regression, not only in terms of individual behavior, but also to an almost complete dependence on the integrity of neural structures which are shared with man, in substantial measure, by many of the phylogenetically older mammals.

Moderate nonspecific inputs are, on the other hand, necessary to the normal functioning of phylogenetically recent fields, as we have earlier seen in general, and as this example suggests in the $C^{+} \longrightarrow E^{m}_{iw}$, $m \approx n/2$, $w >> 1$, transmissions. One hereby finds the neural underpinning of the familiar fact that slight anxiety, or other sources of slight nonspecific inputs, sometimes actually improve academic performance, whereas high anxiety induces a decomposition of behavior in general. It should not, however, be overlooked that even slight anxiety often increases autistic behavior in qualitatively the same way that high anxiety does. The important point is that neocortical functioning is not completely stifled and that, regretably, academic success for even college student subjects often depends in overwhelming measure on autistic memorization and obedience. This is especially unfortunate since the dynamical contractions induced by anxiety force a bypassing of the highest associational fields, which subserve the most delightful forms of human creativity, and produce a motivational system based on short-run stabilizations of a homeostatic anxiety-reduction mechanism, which limits the capacity of the individual to tolerate the prolonged periods of ambiguity that often precede the emergence of the best new ideas. When academic pressure is carried to a fever pitch, the academic system ridicules its own ostensible purposes by being the most effective source of the student's inability to achieve fulfilling insights and a mature

emotional disposition. Great immitative stability is, to be sure, achieved, but the best imitators often cannot tolerate the pressure of the behavioral paradigm which academic pursuits have offered, leaving academic studies permanently at the earliest possible date with but an autistic memory, rapidly fading, of their academic years, and little real conceptual understanding of their world. And the brilliant immitators who stay on must often be fed short, well-defined problems to survive their own academic training. Here is, therefore, an example of the regression to extremal values in a massive social context at our very doorstep. Let us hope for the day when academic institutions, on a broad scale, can present more subtle scales of acceptable behavioral alternatives to their students.

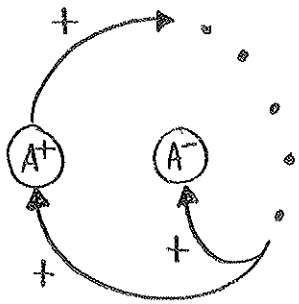
172. Experimental Neurosis, Ambivalence, and Goals

The second remark on psychological implications of the above diagram is the psychological analog of the unpleasant social consequences which can ensue when an individual cannot discriminate between behavioral acts which are classified in the social reward structure in markedly different ways. Our remarks on operant conditioning emphasized the initiation of behavioral sequences by I^+ inputs to A^+ nuclei with the natural end result of inducing I^- inputs to reciprocal A^- nuclei, thereby bringing the A^+ A^- relevant systems into equilibrium. It sometimes happens that an error is committed somewhere between the delivery of I^+ and the asymptotically sought I^- input, with the effect that an I^+, rather than an I^-, input is the end result of the interpolated behavioral sequence. If the behavioral sequence leading from an I^+ input to another I^+ input has not already been strongly embedded, then the "error" is coupled with "negative reinforcers," as we earlier observed. In this sense, no "error of expectancy" has been committed, since the behavior was not guided by a stable embedded sequence of forms. The situation is quite different when forms which have become strongly coupled to A^- induce an increase in the I^+ input. This is an "error of expectancy" in the sense that the organism incorrectly identifies a goal object $\tilde{G}$ with the goal object G whose neural representation had previously been coupled to the A^- system. As a result, A^- will be activated by some external inputs (e.g., visual inputs) which are induced by the mis-identified $\tilde{G}$, but A^+ might well be activated by other external inputs that are unmistakably associated with $\tilde{G}$ itself (e.g., gustatory inputs from a quinine coated food pellet). Thus, instead of the "expected" cycle



(I)

we have



(II)

Since A^+ and A^- are coupled to incompatible behavioral sequences in many instances, the animal's behavior becomes disorganized. Moreover, whereas (I) tends to strengthen the embeddings which realize it, (II) tends both to strengthen and to weaken them. Situations of this type have been studied under the heading of experimental neurosis, which becomes increasingly pronounced as the $G, \bar{G}$ distinction becomes increasingly difficult to make and the A^+ "drive" is high. The critical feature of such neuroses is thus that embedded forms strongly activate one type of nucleus during operant behavior, while the goal object itself simultaneously activates an antagonistic nucleus along a different input route. The embeddings thus lose their "significance" to the organism as sources of A^- inputs, and a period of "ambivalence" and behavioral confusion follows. Since the activation of A^+ "drives" often is a cyclic, metabolically controlled affair over which the organism has little or no conscious control, the total 7^* field is necessarily frequently contracted to the $A^+ \oplus A^-$ system, yet the organism cannot distinguish a behavioral sequence which promises to bring $A^+ \oplus A^-$ into equilibrium. If the difficulty in discriminating a consummatory behavior sequence is particularly great, it is easy to see that the organism's behavioral output can be almost completely paralyzed. For the 7^* contraction becomes more pronounced as the homeostatic balance is increasingly upset, so that the organism must find a behavioral path to re-establish equilibrium before other behavioral systems can be released from $(A^+ \oplus A^-)$-induced inhibition. Yet as the homeostatic imbalance increases,

nonspecific nuclei also come increasingly into play, with the end result that only the most autistic behavioral outputs can be activated. If none of these outputs is suitable to remove the ever-increasing imbalance, the organism completely loses its capacity to deal with the problem. One also sees why, under high drives, the organism's efforts to solve the problem become increasingly maladaptive; his capacity to discriminate environmental details rapidly sinks to a low value.

From these remarks, the meaning of a behavioral expectancy or goal becomes quite clear. For many neural systems can be brought into equilibrium, or "behavioral completion", only if particular environmental input sources are made available at particular times in the realization of embedded control sequences. Such environmental input sources become "goals" for the organism when these embedded control sequences are activated. The $A^+ \oplus A^-$ system provides an example of homeostatic and affective equilibrium, but it should also be realized that even nonspecific $C^+ \oplus C^-$ nuclei can be coupled to particular behavioral subsequences, and we have already seen many examples in which only properly ordered inputs can bring a nonspecific system into equilibrium. With this introduction to the matter, the reader can now easily construct many examples of systems whose behavioral completion requires special environmental inputs at well-determined space-time loci of their activation cycle.

An obvious conclusion of these remarks is that an organism which has suffered from particularly crippling (II) paradigms might try to ensure that such a paradigm never occurs again. A simple way to ensure this is to entirely avoid the situations in which the (II) paradigm was realized. It is qualitatively easy to see how such avoidance behavior arises, for whenever the control forms associated with input signs of the situation are activated, A^+ and A^- will again be jointly activated, behavior will be gradually disorganized, and the behavioral act will never be completed. To the extent that an autistic fleeing response can be activated in such "conflict'filled" situations, the organism will be able to terminate the source of the upsetting inputs by running away. One can understand many behavioral disordered in a similar fashion. The reader might, for example, try to apply embedding methods to a discussion of psychic repression. One can even discuss the neural substrate of such somewhat metaphysical concepts as Id, Ego, and Superego in these terms, with the great advantage that one can now construct fields which exhibit these effects as natural manifestations of neural interactions.

173. Bertrand Russell's Number "2"

From the present perspective, we see that Bertrand Russell's logical definition of the number Two as the set of all pairs of objects in our experience is psychologically partially correct. For our "understanding" of the (auditory or visual) input "2" requires the excitation of many pairs of object representations from our experience in order to deform the global potential distributions in such a way that an embedded residue is formed, excited jointly by all of these representational pairs, which has none of the specific characteristics of these representations apart from their "2"-ness. Even this "2"-ness has no unitary aspect. For one can make the "2" sound, or can make formal arithmetical computations with the "2" symbol, without having jointly embedded any of the experiential pairs required to have a denotative concept of "2." Indeed, one can even be trained to raises two fingers whenever the symbol "2" is presented by a specific modality without having a denotative concept of "2." It is only when a "2"-like minimal field extension arises in complex interaction with many "pairs of objects" that the abstract meaning of "2" begins to emerge, and this meaning is manifest in the complex contractions of the global minimal field which occur whenever the input "2" is delivered through a modality that has been denotatively trained to receive it.

It should be perfectly clear that most conceivable embedding fields cannot sustain such a joint control form extension, as reflection upon the limited capacity for abstraction shown by most phylogenetic forms immediately shows. To see how such extensions spontaneously occur in man, we will therefore expect to be led into a study of some very complicated minimal field constructions. Such constructions presuppose the construction of a reasonably sensitive $\mathcal{T}_0$ field, and the various $\mathcal{T}_1$ fields with which it interacts. These constructions would take us deeper into lengthy neuroanatomical details than the present length of this paper permits.

174. A Remark on Psychophysics: Power Laws and Critical Durations

The embedding field dynamical laws and geometrical arrangements provide a theoretical description of the workings of the physiological "black box" which is interpolated between physical inputs and behavioral outputs. A subject of particular interest in psychophysical studies of this "black box" has involved the demonstration, say in the work of S.S. Stevens, and Mountcastle and Poggio,

that various input modalities, in their steady state behavior, are transformed by approximately a power function of the input intensity, up to a shift by a scaling parameter. To understand this question in detail, one must construct specific minimal fields for every experimental paradigm, based on the particular symmetries of the modalities involved, and study the ensemble behavior thereof. We will turn to aspects of this task in later works. To motivate these forthcoming studies, we here call attention to central local dynamical features that underlie the ensemble picture.

In the simplest possible case, one envisages a single point strength equation of the form

$$ds/dt = a(M-s)I-bs,$$

where I is taken as a known function of time. Thus,

$$s(t) = aM \int_0^t I(w)\exp(-\left[a \int_w^t I(u)du+b(t-w)\right])dw$$

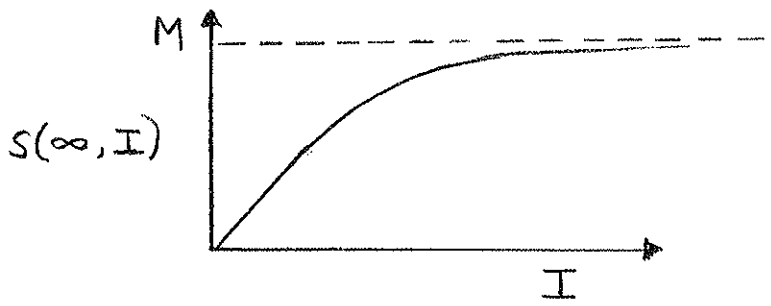
whenever $s(0)=0$. Since we are interested only in steady state behavior, we let

$$I(t) = \begin{cases} I & t \geq 0 \\ 0 & t < 0 \end{cases}$$

where I is a positive constant, for simplicity. Then

$$s(\infty) = \lim_{t\to\infty} s(t) = aIM/(aI + b).$$

Considering $s(\infty)$ as a function of I, denoted by $s(\infty,I)$, we have the graph



of the asymptotic $s(\infty,I)$ level as a function of I. Notice that for I small compared to b/a, $s(\infty,I) \approx (aM/b)I$, so that if, in particular, $I \approx \gamma J^{\delta}$, $\delta > 0$, for small I, $s(\infty,I) = S(\infty,J) \approx (a\gamma M/b)J^{\delta}$, and the asymptotic s value is approximately a power function of J. Nonetheless, for large I values, the asymptotic s value approaches a finite asymptote M, and does not approach infinity as

a power function does. Similarly, if I is given asymptotically by $I = \gamma(1-e^{-kJ})$, where J measures the steady state intensity of an input parameter, then

$$s(\infty,I)=S(\infty,J)=\frac{a\gamma M(1-e^{-kJ})}{a\gamma(1-e^{-kJ})+b},$$

which can also easily be mistaken for a power law for small J values, but which again has a physically desirable finite asymptote.

Thus, even for the most trivial strength function of a single cellular unit, we find asymptotic strength values that, for small input values, behave as a power function, or approximately so, of the input without exhibiting the physically unpleasant property, shared by power functions, of approaching infinite values for large I. What is measured in any given experiment is, however, not the behavior of any one cell, but rather the resultant behavior of interacting clusters of cells in specific arrangements. Even a peripheral physiological input cannot be viewed as an input to a single cell, for from the periphery to the most central neural stations, cells are organized to provide mutual inhibitory and synchronizing effects at every successive transmission, and many of these cells will have disparate thresholds, transmission latencies, local coupling types, and the like. Yet all such cells will tend to produce asymptotic $s(\infty,I)$ values which are convex as a function of I and approach finite asymptotic values that depend on the total embedding spaces of the cell and on dynamical rate constants.

How does the above transformation of asymptotic s values behave in a transmission chain $I \rightarrow p_1 \longrightarrow p_2 \longrightarrow \cdots \longrightarrow p_n$? Here we have the equations

$$ds_i/dt = a(m-s_i)rs_{i-1}(t-v) - bs_i$$

in the simplest case, where $s_o = I$ and we have identified all constants between various points. Also for simplicity, suppose that $r=1$ and that M, a and b are so chosen that with each successive transformation in the chain, the new input may be viewed, in first approximation, as a step function. Then

$$s_1(\infty,I) = aMI/(aI+b),$$

$$s_2(\infty,s_1(\infty,I)) = aMs_1(\infty,I)/(as_1(\infty,I) + b) = a^2MI/\left[(a^2 + ab)I + b^2\right]$$

$$\vdots$$

$$s_n(\infty,s_{n-1}(\infty,\ldots)) = aMs_{n-1}(\infty,\ldots)/(as_{n-1}(\infty,\ldots) + b)$$

$$= \frac{a^nMI}{(\sum_{k=0}^{n-1} a^{n-k}b^k)I + b^n}$$

$$= \frac{aMI}{(\sum_{k=0}^{n-1}(b/a)^k)I + (b/a)^{n-1}b}$$

$$= \frac{aMI}{\left[\frac{1-(b/a)^n}{1-(b/a)}\right]I + (b/a)^{n-1}b},$$

whence for $b<a$ and n large,

$$s_n(\infty,I) \approx (1-(b/a))aM = (a-b)M,$$

or

$$s_n(\infty,I) \approx \begin{cases} (a-b)M, & I > 0 \\ 0 & I = 0 \end{cases}$$

which is independent of $I>0$. Thus, even in the absence of lateral inhibitions and nonspecific inputs, one finds a tendency for peripheral energy control to diminish over a chain of successive transmissions of the most trivial type. Even more important is the fact that the form of the asymptotic s_m values, namely

$$s_m(\infty,\ldots) = aMI/(a_mI + b_mb),$$

where a_m and b_m are functions of a and b, is preserved for all $m=1,2,\ldots,n$. In particular, if $I=\gamma J^{\delta}$, for small J, then the successive s_m asymptotes will all reflect this dependence on δ, mixed with the tendency for all positive energetic differences to be eliminated. A similar remark holds when $I=\gamma(1-e^{-kJ})$. It must be recalled in this regard that long transmission chains of the above

type are rarely exactly realized; an enormous degree of convergence and divergence of neurons, including nonspecific energizing effects, are constantly in play, whence the invariance of the asymptotic expression over many transmissions exerts a strong effect.

Observe that local alterations in the dynamical laws significantly alter the $s(\infty,I)$ asymptotes. For example, given the equation

$$ds/dt = a(m-s)(r^+s + I^+ + 1) - bs(r^-s + I^- + 1)$$

where I^+ and I^- are again taken as known nonnegative constants, we find, setting $ds/dt = 0$, that

$$s(\infty;I^+, I^-) = Y + \sqrt{Y^2 + 4M(1+I^+)}$$

where

$$Y = \left[Mr^+ - (1+b+I^+ + bI^-\right] / (r^+ + br^-).$$

Yet once again $s(\infty,...)$ is a combination of sums and powers of the input asymptotes. All of these asymptotic s values may, of course, be directly translated into statements about steady state frequency modulation according to our remarks on spiking invariance principles. Ultimately, computations of $s(\infty,...)$ values must be given as functionals of the steady state behavior of the local dynamical systems which directly mediate between external inputs and neural systems.

In these two examples, all thresholds were taken equal to zero. In the presence of nontrivial thresholds, inputs to a cell do not generate transmission unless the transmission threshold is exceeded. Since many successive transmissions are required in the passage from any peripheral input to a peripheral output, the psychophysicist receives the impression that there exist "critical durations" during which, it has been thought, energy contributions summate and after which an asymptotic value is reached and a more central part of the neural processing system is activated. It has also sometimes been erroneously inferred from the existence of these critical durations that local psychological time is quantized. In reality, there exist many staggered "critical durations" which are, in local terms, coextensive with the time necessary for each cell to reach its transmission threshold, and even after the transmission threshold has been realized, strength continuously accumulates to modulate the frequency of transmission. Since, however, psychophysical experiments usually measure on only one or two kinds of output behavior scales, these various local summations may, in first approximation, be lumped into a single

waiting time which measures the cumulative transmission lags over successive transmissions. An important difficulty with this view, apart from its insensitivity to the actual dynamical evoluation of the system, is that as the input and output scales are taken ever finer, and more complete, the time scale of "critical durations" that appears in the data becomes increasingly fragmented into ever smaller, overlapping, and inhomogeneously weighted pieces. This follows directly from our remarks on the existence of hierarchically staggered input-associational-output systems. Nor can it be overemphasized that the process whereby strength values are augmented to exceed transmission threshold is not a simple additive one. Even apart from exponential volume conductor terms, an excitation in one part of the system is usually coupled to an inhibition in another, as one so clearly sees in phenomena relating to critical bands in hearing.

In summary, the tools for understanding the general form of the psychophysical transformations are now in our hands. In particular, the universality of this form is a direct consequence of the universality of the general structure of embedding equations. Armed with these equations and a superstructure of embedding principles, research can now turn to exhaustive theoretical studies of particular systems.

175. Applications to Child Development and Therapeutic Methods

The interactions envisaged above are pertinent to familiar facts about child development. In the earliest years of life, many associational field interactions are interpolated between input-output lines that are directly relevant to the satisfaction of the child's primary needs, and are embedded in the closest possible juxtaposition to the sensorimotor centers that control the satisfaction of these needs. These needs are often provided for by such multiple sources of reward and punishment as the child's parents. Our remarks show that an integrated associational complex will be embedded in the child's brain to represent every such multiple source. Such an associational complex intimately interacts with the mechanisms subserving the primary needs of life whenever these needs arise. In particular, all future inputs that are relevant to these needs will activate the parental associational complex and will be embedded in close harmony with this complex. In this way, the earliest multiple sources of reward for the child strongly determine the child's perception

of the many related facets of his experience which occur thereafter, and these newer experiences are actually embedded in the child's brain in a functionally close juxtaposition to the early multiple source representations. The childhood years thus leave the strongest imprint on the interpretation of later experience, as Freud incisively observed on many occasions. All future embeddings that involve those sensorimotor systems which have acquired a distinctive behavioral mode during childhood will be embedded in such a way that the earlier mode is maximally sustained.

The interpolation of early associational forms between sensorimotor systems and later embeddings shows that a non-gradual therapeutic attempt to alter these early structures will be accompanied by great instability of behavior and the possible collapse of all related associational systems, for the associational bonds linking these systems to the "real world" of sensorimotor interactions will have been shattered before new bonds can be formed. In the same way, a patient will quite automatically exhibit resistance to symbolic therapy, for embedding fields are constructed to maximally sustain their control forms once they are successfully embedded, whatever their denotative content might be. Such resistance is a natural manifestation of the therapeutic process of embedding new forms whose $7_o \leftrightarrow 7_1$ interactions differ considerably from those of the old forms.

When a behavioral difficulty is induced by a particular cognitive associational complex, wholly noncognitive therapeutic methods can often do more harm than good. For unless the therapeutic method takes advantage of the specialized structure of the sensorimotor systems through which the troublesome control forms were originally embedded, it is very difficult to change these forms without changing practically all of the forms in the field. A patient who has lost all of his associational forms, however, has lost all of his potentialities for coping with, and enjoying, the world once he is "cured" of his malady. Nor can it naïvely be thought that such a patient can be retrained to a fulfilling life. His brain is no longer well-suited to global retraining, even if the therapeutic method does not irreversibly destroy large masses of brain tissue. In particular, many new lines have grown and/or become myelinated since the patient's childhood. The line structures of his fields have become increasingly rigid to permit the complex, stable interactions of maturity. Once these stable structures are rendered unfunctional, little remains to replace them.

We recapitulate this line of reasoning in somewhat more general terms because of its importance, and the apparent lack of understanding that is so widely prevalent in certain therapeutic circles. A particular complex of associational forms cannot cause profound behavioral difficulties unless it is strongly embedded and renormalizes many input forms to fit its contours. The input forms which are affected are those which are delivered to the systems that are closely integrated with the associational fields containing the troublesome complex. If one envisages a therapeutic method which is not at least partially realized by delivering inputs in the physiologically natural manner to the afflicted systems, one can only alter these systems in a global way. Any such alteration that succeeds in removing the influence of the troublesome complex must also, perforce, render all less highly embedded forms unfunctional. Since the troublesome complex would not require therapeutic attention were it not so strongly embedded that it can induce broad, maladaptive field renormalizations, this means that very many useful control forms will be indiscriminately obliterated by the global method. Moreover, such a global obliteration leaves the field incapable of developing new adaptive forms in the future. Although the patient might possibly lose his problem under such therapy, he will surely lose many of his most treasured capabilities.

A global therapeutic method that falls squarely under the above criticisms is electroshock therapy. Shock cannot remove behavioral difficulties engendered by a particular associational complex without first indiscriminately destroying large regions of the brain on the microscopic level. The only situations where the useful effects of shock can even begin to hope to offset its noxious concomitants are those cases in which the behavioral problem is not linked to a particular associational complex, but is rather tied to a difficulty that is itself induced by a global renormalization mechanism. Abject withdrawal cases are sometimes of this type. Prefrontal lobotomies are also global in the noxious sense. Drugs which merely tranquilize a patient do not alter a single embedded form. If and when such patients reawaken, they will be as sick as ever. Drugs that reversibly influence particular brain nuclei can be used effectively, if they are supplemented by modality-specific inputs while the drug is still in effect. For example, a reversible drug that inhibits nuclei which give direct expression to fear reactions without producing an unconscious thalamo-cortical system can be used in conjunction with verbal therapy. Customarily, when cognitive forms projecting strongly to the fear centers are

activated, the stimulation of the fear centers will induce widespread activation of defensive mechanisms. In particular, new cognitive inputs will be renormalized out of existence, rendering the effective embedding of new, adaptive forms difficult or impossible. This is essentially the paradigm of generalized avoidance behavior. When the fear centers are specifically inhibited, on the other hand, the defense mechanisms are never activated, so that new forms can gradually be embedded over the old fear-inducing forms. Notice that these new forms might not be able to eliminate the old forms. But it might well be possible to embed new forms that reciprocally inhibit the old forms, or which project to centers that inhibit the fear reaction induced by the old forms. The general idea underlying therapy of this type is: although it is sometimes very difficult to embed new forms within the entire representation space $\mathcal{7}^*$, if we functionally inactivate a subspace $\mathcal{7} \subset \mathcal{7}^*$ we can consider the problem of embedding in $\mathcal{7}^* \setminus \mathcal{7}$, which might be much easier to accomplish if $\mathcal{7}$ is properly chosen. Moreover, if we are skillful, after embedding in has been completed, the new embeddings, considered now in $\mathcal{7}^*$, inhibit control forms which have caused the patient discomfort in the past. In abstract form, our problem is: Given an associational complex ξ, activated by inputs I_ξ which induce the undesirable effects $z_1(\xi, I_\xi), z_2(\xi, I_\xi), \ldots, z_n(\xi, I_\xi)$, find a sequence of subspaces $\mathcal{7}_1(\xi, I_\xi), \mathcal{7}_2(\xi, I_\xi), \ldots, \mathcal{7}_m(\xi, I_\xi) \subset \mathcal{7}^*$, and input clusters $I_1(\xi, I_\xi), \ldots, I_m(\xi, I_\xi)$, such that after I_i is delivered to $\mathcal{7}^* \setminus \mathcal{7}_i$ in a specified ordering, for all i, the undesirable effects $z_1, \ldots, z_n$ no longer occur when the I_ξ are thereafter delivered to ξ, now considered in $\mathcal{7}^*$. The side condition $\xi \not\subset \bigcup_i \mathcal{7}_i$ is usually imposed. And, of course, we wish to accomplish this in "the best possible way, under the circumstances." For example, $\mathcal{7}_{2i} = \phi$, $I_{2i} = ?$ corresponds to leaving the patient to his own devices between regular therapeutic sessions.

Generally speaking, one must always employ a therapeutic technique whose inputs are capable of selectively altering the behaviorally objectionable forms. Such inputs must be delivered on a time-scale for which the relevant maladaptive line structures can be made to change without inducing generalized behavioral instability. To deliver an abrupt global shock in an effort to erase embeddings which have gradually, and specifically, accumulated over many years is to admit profound ignorance of even the rudiments of memory storage. A careful application of the general principles and examples of this paper can be made to suggest reasonable therapeutic procedures, or at least to suggest which procedures are unreasonable, before many lives are irreversibly mangled in experimental

blunders. We make particular reference to our discussion of cognitive-emotive interactions and of conditioning paradigms of various types, as well as to the various uses of lateral inhibitory structures, which these interactions helped to motivate. In a later work, we will concentrate on such problems in detail, but we must first study the structure of the underlying fields more thoroughly.

176. General Remarks

The theory of embedding fields associates with every geometrical object w in a structural carrier one or more dynamical functions f which describe the time dependent behavior of the object. The set of all geometrical objects, paired with their respective dynamical functions: (w,f), defines a free embedding field. No free field exists in Nature, for the very emergence of the field geometry is guided by input arrays which are themselves partially determined by geometrical precursors, as we saw in discussing field rigidity and plasticity. Thus, to completely define a field $\mathcal{F}=(w,f)$, we must be given an explicit input paradigm $\mathcal{I}$ and the output paradigm $\mathcal{O}$ generated from $\mathcal{F}$ by $\mathcal{I}$. The $\mathcal{I}$ which are admissible as inputs to $\mathcal{F}$ depend on $\mathcal{F}$'s local structure, as we saw in discussing field extensions from the viewpoint of the set of stationary $C(\mathcal{F})$ which $\mathcal{F}$ can carry. $\mathcal{O}$ also influences the field extensions of $\mathcal{F}$ and thereby indirectly determines $\mathcal{I}$, which of course reciprocally determines $\mathcal{O}$. A field is therefore completely given by a triple $(\mathcal{F},\mathcal{I},\mathcal{O})$. The inputs and outputs are, in turn, themselves determined as dynamical processes over appropriate embedding fields, whence every function in the field, whether in $\mathcal{F}$, or $\mathcal{I}$, or $\mathcal{O}$ may be viewed as an intrinsic measuring device of the field activity which it can distinguish. Were it true--which it is not--that we had not been able to determined the local dynamical structure of $\mathcal{F}$, how could we have achieved more partial and inferential insight about this structure? Our only alternative would have been to study the $\mathcal{I}$ and $\mathcal{O}$ as functions measured by physical measuring devices interpolated within the input and output lines. These physical measuring devices must, in a thorough analysis, be studied as dynamical augmentations of the original system to gain intrinsic insights. Since, however, we are supposing that the local $\mathcal{F}$ structure was impossible to ascertain by these devices, we can be content to think of the numbers which these devices provide as givens of the situation, not subject to further analysis. Doing this, we see that to every input function $I\in\mathcal{I}$--as I is seen through the numbers in our measuring device--gives rise to a specific output function $O(I)\in\mathcal{O}$. That is, we have a mapping $S:\mathcal{I}\rightarrow\mathcal{O}$, and that is all

that we have, for $\mathcal{F}$'s structure is unknown. Each of $\mathcal{I}$ and $\mathcal{O}$ is a collection of functions, whence S: $\mathcal{I} \to \mathcal{O}$ becomes an operator between two function spaces. Ignorance of the local structure of $\mathcal{F}$ hereby forces us into a functional analytic study of operators on function spaces which represent asymptotic input-output properties of the total system $(\mathcal{F}; \mathcal{I}, \mathcal{O})$ relative to $\mathcal{F}$. Corresponding to the function space approach is the need to replace dynamical statements by kinematical arguments which facilitate the study of field symmetries, but which thoroughly obscure the underlying dynamical meaning of these symmetries.

Several remarks about the asymptotic S operators immediately follow from our previous investigations: (1) Whenever the $\mathcal{F}$ line structure activated by I exhibits profuse convergence and divergence of lines relative to the activated O(I) lines, we are placed in a situation that is formally identical to that governing dendritic cross-sections: the input to every O(I) line is determined by many small, relatively independent dynamical contributions, whence an approximation to a random process with independent increments given as averages in space-time becomes possible, and S is an approximately linear operator. Increasing the convergence and divergence of lines, on the other hand, increases the field's capacity to distinguish inputs and thereby stabilizes field dynamics. (2) As in the case of Purkinje outputs due to localized $\mathcal{I}_i$ inputs in the cerebellum, the output from $\mathcal{F}$ is graded in space-time. Thus, the behavior of local regions in space-time will be highly correlated, so that it will seem appropriate to study integrals of O(I) relative to measures which properly weight the $\mathcal{F}$ grading. Hence, we are not only led to study asymptotic linear operators; we must also consider our function space from the purview of the theory of distributions, with our test functions chosen to replace the intrinsic description of the distribution of excitation by $\mathcal{F}$ lines. Such functionals as the information functional, product-difference functionals, and exponential polynomials with functional coefficients, which are replaced by intrinsic deynamics in $\mathcal{F}$, reappear as functionals in the contracted S description. (3) A further consequence of asymptotic S studies is that, instead of providing a description in terms of the interaction of local strength and line functions, we must talk about the distribution of I functions relative to the distribution of O(I) functions. That is, we are forced to consider S probabilistically. In particular, dynamical conditioning and the propagation of symmetries in $\mathcal{F}$ becomes a discussion of probabilistic conditional distributions to S. Moreover, since the strength field of $\mathcal{F}$ is invisible to S, the binding of hierarchies of line functions to this field becomes obscured and all that is visible are hierarchies of seemingly recursively defined correlation functions, which are the asymptotic analog of the line functions themselves, the highest order correlation functions corresponding to the most multiple

lines. (4) Field-antifield duality and stability conditions---realized by intrinsic field extensions---in 7 appear as CP and T symmetries, relativistic contractions, and wave motions to S. (5) Parity invariance in I → O(I) is preserved only if I and O(I) subserve the same muscle groups; that is, only if input-output reciprocity holds. (6) A superposition principle for admissible inputs of different S wavelength holds, in first approximation, because the lines of fixed length cannot distinguish wavelengths of different non-multiple length, and these wavelengths are inhibited out within the local weighted complete sets of antagonists of the lines, whence only wavelengths of the order of these lines affect their dynamical evolution. (7) When 7's local structure is spatio-temporally self-similar, quantization of 7's dynamical behavior, including masses and average energies, reduces essentially to the fact that the number of objects in 7 is finite. Admissible inputs to 7 are then also spatio-temporally self-similar, and, by (6), M values of a fixed range in S - 7 - O interactions will resonate with one another, while M values of a different---and non-multiple?---range will be inhibited out of existence within the restricted M system. Spatio-temporal self-similarity is itself a stability condition which assures a sensitive reciprocity between local control and global interactions. It is a reply to the intuitively appealing question: how can outputs from a given system be constructed to ensure that when they reach another system as inputs, the integrity of the recipient system will not be violated---given that when the output arises, it cannot know precisely what systems will eventually feel it as an input. (8) Consider a given localized output line L from 7 as an intrinsic measuring device of 7's behavior. When a wave of excitation passes over L, L measures the contracted field induced by the wave, which takes the form of virtual self-inputs with both advanced and retarded potentials in many cases. Only one type of potential occurs when the line structure in the field carries as induced directionality, but even here one finds an ordered sequence of excitatory and inhibitory potentials in "on"-"off" and "off"-"on" fields. When L is not localized in 7, the advanced and retarded potentials can be obscured, and the time variable which is attached to L is no longer the natural time variable of local events in 7. All measuring devices measure a minimal field which is a contraction to a virtual system of the total field. All field structures are measuring devices. The implantation of new measuring devices therefore alters the distribution of field objects according to field laws. When the measuring device operates on the same level of graining as the original system, the act of measurement alters the distribution of field quantities even as the measurement is being made. Yet the field laws do not change. We are merely in a different

field. Whereas it is impossible to measure a system without a measuring device that can perturb the theorist's macroscopic cognitive fields in an "understandable" way, it is nonetheless possible to discuss the behavior of such systems theoretically, in principle. By restricting oneself to asymptotic S considerations, however, one loses so much dynamical information that what is possible in principle becomes uninformative in practice. A theoretical local study of 7 is necessary to acquire significant new insights beyond those that are directly measurable. Without such local studies, at least for neurological 7's, one almost completely misses the interesting phenomenological points, as the customary Markov models have. (9) Although local dynamical laws are completely symmetric, an irreversibility in dynamical time is achieved by coupling a collection of fairly homogeneous units of fixed interaction range with a system whose interaction range is sufficiently broad, rapid, and massive to renormalize all of the other units when one of them is differentially excited, as we saw in Sections 5 and 6. Thus, irreversibility in ensembles of units reduces to a discussion of field renormalizations determined by admissible field extensions, which is a question about the local stability of individual units. (10) Indistinguishable packets of points are sometimes necessary to provide properties unrealizable by individual units. These packets are indistinguishable only modulo specific admissible inputs and outputs. In S studies, the class of inputs and outputs does not include any internal 7 inputs and outputs, and indistinguishable asymptotic inputs and outputs are properly lumped into the maximal equivalence classes for which elements from two different classes are distinguishable. (11) Local dynamical laws fall along several mutually exclusive continua. Consider a single object. If the local law is volumetrical, the object is contracted to a single point with a single function. A superposition law is the antithesis of this contraction: all units remain independent. Consider two or more objects. If the local subsystems of one object affect the second object's subsystems by transport processes with relatively great local fluctuations, the two objects are chaotically coupled. Otherwise, the local tubular transport structure between subsystems is preserved and linear coupling occurs. The analogy between volumetrical and chaotic couplings, and between superpositions and linear couplings is obvious. Noncommutativity in the ordering of averaging the local functions of the objects and of the inter-object transport mechanism determine all differences in coupling. (12) Although our capacity to represent a total nonlinear system symbolically in all details is incomplete, field-antifield duality provides a criterion for completing any system up to a given level of dynamical graining, while local deviations from field-antifield duality induce new dynamical hierarchies in the evolutionary

process.

From the most sequestered associational fields to the most peripheral neural stations, the embedding laws and principles are universal. Beyond the neural fields which these laws govern lie the physical fields which are their ultimate completion and the source of all of our information about the world. It is impossible that the principles governing these physical fields are extraordinarily different from the principles governing neural fields, for otherwise neural interactions would either be very unstable or would change their form as the primary receptive fields are approached--neither of which seem to be the case. Thus, we must be able to extend our embedding spaces still further until they include the physical fields which they can distinguish in a harmonious way. The ultimate completion of the field extension process, begun with p_W, is such an inclusion of physical fields into a total field frame, in which the physical fields are viewed as intrinsically described input sources. The quantitative fulfillment of this extension procedure will require a great deal of effort in the study of particular systems, but its philosophical necessity is now clearly before us, and the analogies of embedding results with known physical field results are quite encouraging, especially since the embedding results provide unifying dynamical stability conditions for field theoretical results which, today, stand on independent postulates. As this extension process proceeds, the classical view of a scientist as a dispassionate observer of physical laws evolving unperturbed by human intervention, extended by the quantum theory to a discussion of the limitations of meaningful questions by the initial conditions of particular experiments, and by the relativity theory to a discussion of the local observational biases of several observers in a given space-time manifold, will be extended still further to an intrinsic analysis of the interaction of the perceiving organism himself with the physical fields that he can discriminate through the various measuring devices--all ultimately coupled to his physiological devices--which he can conceive.

177. Conclusion

This paper contributes to a new program whereby quantitative methods can be used to integrate our knowledge of psychological, neurophysiological, and neuroanatomical facts into a coherent intellectual system. The development of such a system is a natural reflection of the needs of the times. For the unification of materialistic exchange and control processes that has so rapidly emerged in the past century must be balanced by a rational understanding of the human needs whose fulfillment is the proper aim of this unification. Such a

balance is, indeed, a prerequisite to the establishment of a truly harmonious social order.

Yet from the very outset of such a theoretical program, it is apparent that its results can immediately be applied to either enrich or to degrade humanity. One can no longer delude oneself, even for a moment, with the illusion that there exist scientific studies of humanity which are pure and therefore free from a moral concern with the direct application of the fruits of these studies. The study of mankind is not comparable to a study of initially unfamiliar natural occurrances whose understanding can be twisted, through a series of unforseeable and indirect steps, guided by bursts of social lunacy, into a tool for perpetrating human misery. When men study men, the reasons are familiar and the applications are direct.

The practitioners of this new science are therefore faced, from the beginning, with a grave personal responsibility in wisely applying their new knowledge. It is only through the good will of many well-informed individuals that good works can be brought to many peoples and social malpractices prevented. If individual scientists, small groups of scientists, and well-informed men of good will everywhere rebuke this responsibility, it will become the sole domain of rigid institutional organs. Such organs, by virtue of their massive bureaucratic structures, can only dimly distinguish the proper outlets for knowledge of such a subtle nature. The number of stable ways available to such institutional organs for the application of this knowledge on a global scale is therefore markedly reduced. One stable solution always exists: universal totalitarianism. One unstable, but ultimate, solution always exists: universal chaos. Insofar as individuals and small groups of individuals lose interest in the responsible application of knowledge about mankind, one of these solutions to the problem of distributing knowledge becomes inevitable.

These observations will eventually have a rigorous proof. It would perhaps be unwise to state them in such a speculative form at present were the opportunities of the present not so promising. For now we are embarking on a new enterprise, and we can work, in good faith, to newly establish an intellectual tradition worthy of this enterprise. If we ignore this opportunity, and if such a tradition is not established, all of our efforts shall be in vain.

DIAGRAMS

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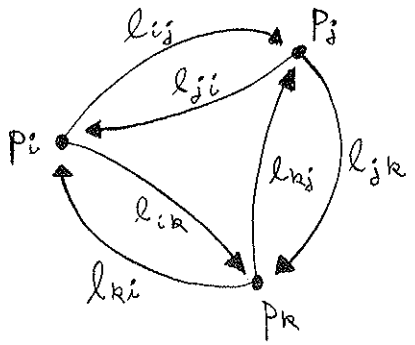

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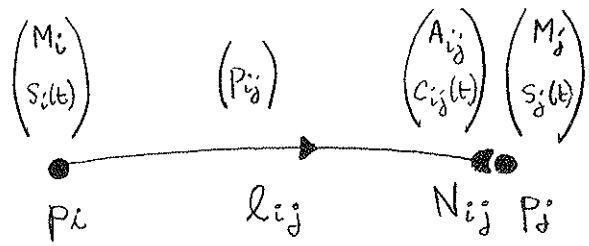

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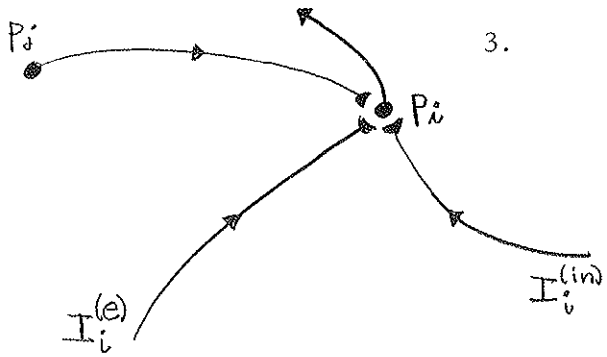

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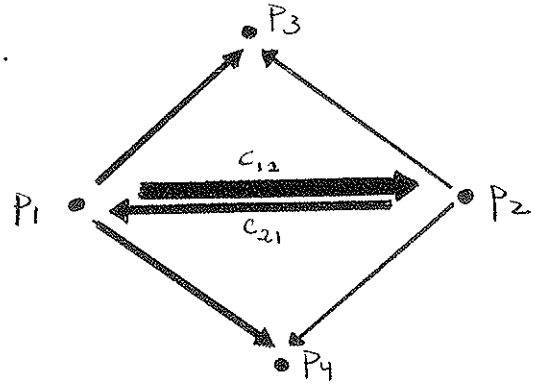

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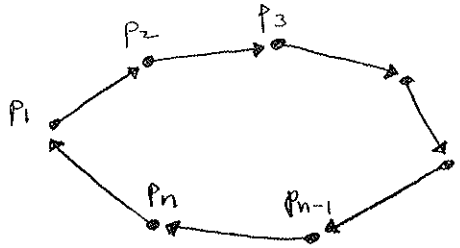

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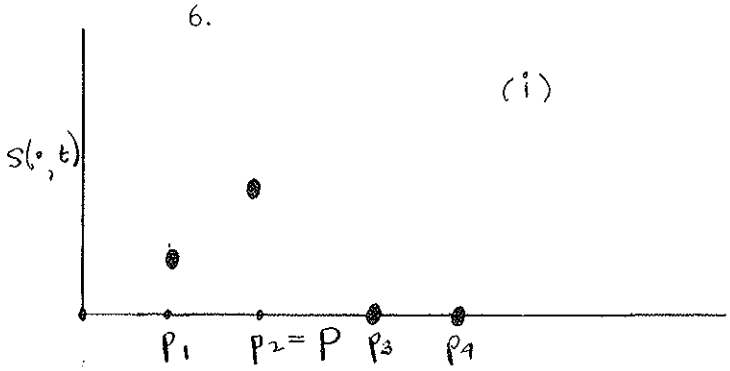

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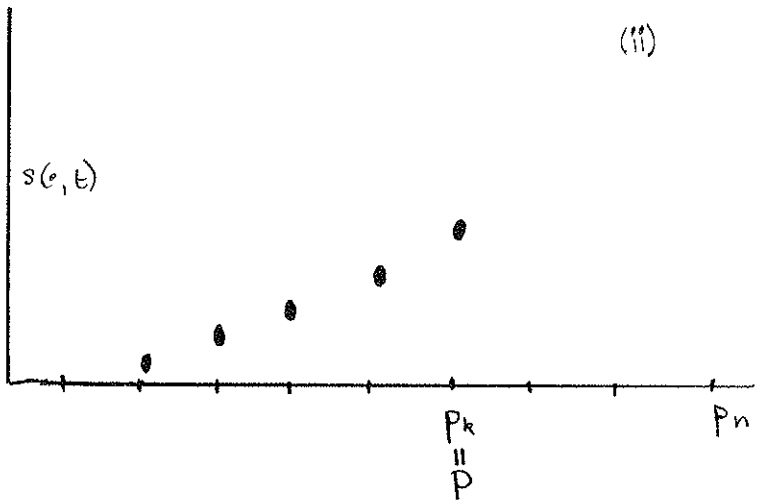

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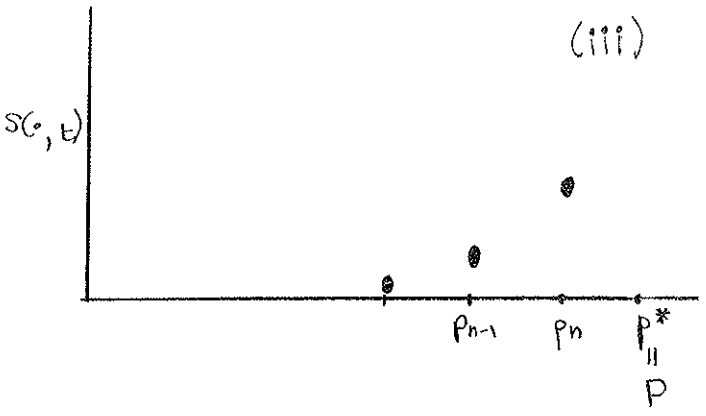

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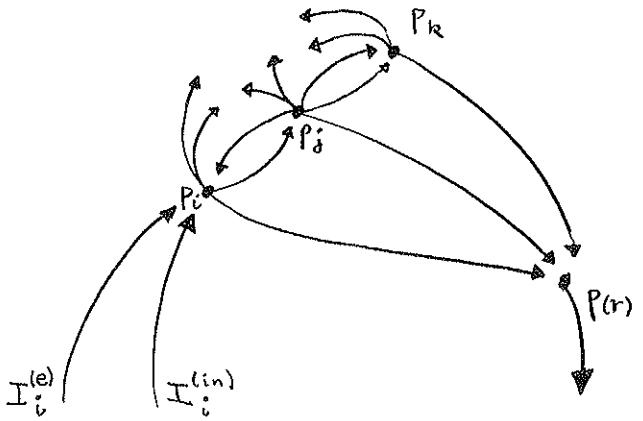

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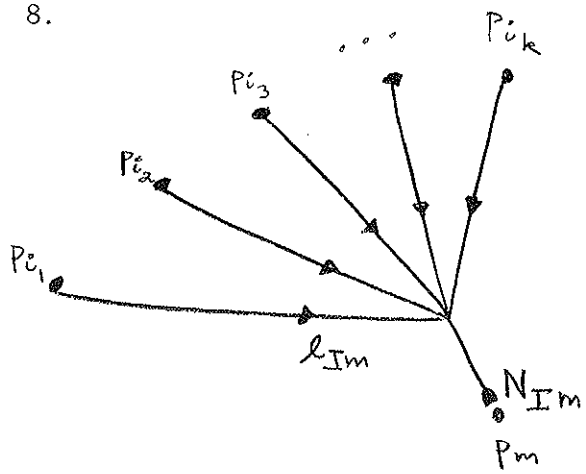

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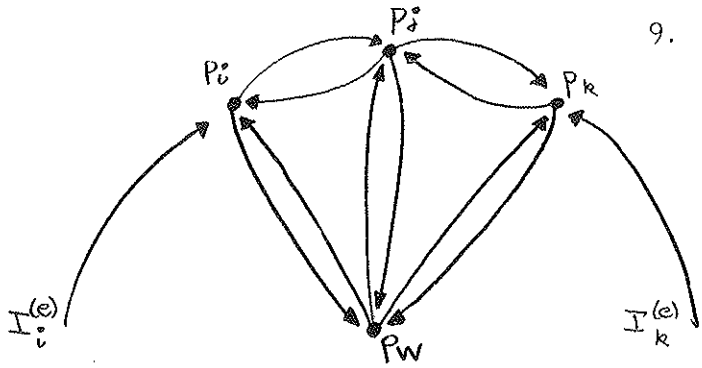

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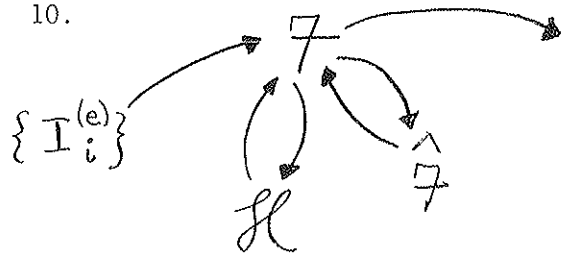

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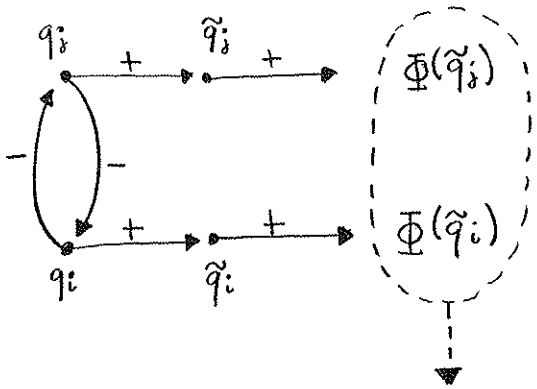
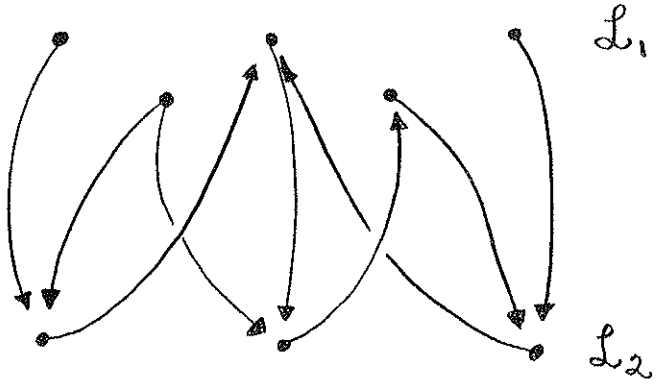


12.



ERRATUM: On page 12, Section 4, the definition for c_{ij}^* should read $c_{ij}^* = c_{ij} / \Sigma_k p_{ik} c_{ik}$. Thus, the transmission from p_i to p_j received at p_j at time t is proportional to $s_i(t-t_{ij}) p_{ij} c_{ij} / \Sigma_k p_{ik} c_{ik}$. In particular, when $p_{ij} = \lambda(1-\delta_{ij})$, $c_{ij}^* = c_{ij} / \Sigma_{k \neq i} \lambda c_{ik}$, whence the transmission from p_i to p_j is proportional to $s_i(t-t_{ij}) c_{ij} / \Sigma_{k \neq i} c_{ik}$, $j \neq i$.

ERRATUM: The term "coenzyme" (e.g., in Sections 125-6) should be thought of

as the general term "cofactor". In particular, the coupling of metal ions to membrane polarity is compatible with the operation of these ions as metal activators and/or inhibitors of the $c_{ij}^{\pm}$ processes. The equations are compatible with the coupling of many cooperative cofactors in parallel to the membrane polarity, if they are linearly coupled to the polarity with respect to their interactions with the ensembles $c_{ij}^{\pm}$. Linearly coupled chains of reactions can therefore be envisaged, as well as sets of subchains mutually interacting in parallel or merging to form new subchains. Coenzymatic reactions, in the narrow sense, can occur in these chains, in principle. In this paper, we restrict ourselves to first-order, rate-limiting factors by collapsing an entire set of subchains, linearly coupled to membrane polarity, modulo $(c^{\pm})$, into a single (s, c) coupling pattern.

The coupling of antagonistic ions to the membrane polarity is compatible with the existence of known ion antagonisms, say between K^+ and Na^+, and between Ca^{++} and Mg^{++}, and with the existence of the significant metalloenzymatic effects, both facilitative and inhibitory, which these ions have on metabolic control. The synergistic effects of Mg^{++} and K^+, and of Ca^{++} and Na^+ are of particular interest, and will be discussed as extensions of the processes herein presented, in the light of known ATP cycles, in another place.

It should not be overlooked that, in the first-order theory, wherever membrane polarity is coupled to a nondegenerate pair of $c^{\pm}$ functions, these functions generate nontrivial $C^{\pm}$ values of " transmitter. " In particular, if a $c^{\pm}$ structural carrier exists in an axon, brief $C^{\pm}$ transmitter release is to be expected when a suitably polarized pulse propagates over its structural carrier. Similarly, a dual C^{+} transmitter release will occur in the dual sheath, and a $C^{\pm} - C^{+}$ annihilation will occur in the space adjoining the two membranes, hereby restoring equilibrium. Neither the transmitter release nor the annihilation will necessarily be measurable from outside the dual sheath, which acts as a functional boundary for the total system. If, on the other hand, no nontrivial $c^{\pm}$ function exists, then the system must be extended to include repressor functions, themselves at best weakly coupled to membrane polarity.

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