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THEORY OF NEURAL-ANALOG REINFORCEMENT SYSTEMS AND ITS APPLICATION TO THE BRAIN-MODEL PROBLEM

By
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A D I S S E R T A T I O N

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in candidacy for the Degree

of Doctor of Philosophy

Recommended for Acceptance by the
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CHAPTER 1 INTRODUCTION

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Chapter 1

Introduction

The purpose of this paper is to develop a new approach to the "brain-model" problem. The following questions illustrate the most important problems in this field.

(1). What are the physiological processes through which animals perform such activities as "learning","memorization","recognition","attention","reasoning",etc.?

(2). How can these activities of so-called "sentient" organisms be duplicated in systems which we can actually construct? Is it possible to describe a system capable of behavior of humanoid complexity,yet simple enough in its physical structure that it can be understood? Can we describe such a system which is in addition,sufficiently resilient that its functioning can be maintained in the face of extensive injury,as in the case of the animal nervous system?

We begin by considering the properties of single neurons,and of simple sets of interconnected neurons ("nets"),with the objective of investigating phenomena which may be of importance in much larger nets and in the brain itself. The results of this analysis are then applied to the study of very large,"random",nets,and finally, to certain assemblies of very large nets. It is these assemblies which are our "brain models".

Each "brain model" is formed of a small number of very large "random" neural nets with a small number of channels connecting these nets;each connection is a large set of fibres which run between a pair of specified nets. In addition to these connections there must be a set of "output" or "motor" channels and a set of "input" or "sensory"

channels which run between the brain model and its environment.

Now the"quality" of a brain can hardly be evaluated except in terms of the relation between it and its environment. Our initially disorganised "random" model must be able to raise itself from its initial chaotic state to a higher degree of internal organization,and this organization must be measured in terms of the extent to which the brain can learn to deal with its environment. This latter capacity must itself be evaluated by some measure;for animals it seems natural to use as measure the ability to maintain the internal (physiological) state within some "normal" range,i.e., the ability to survive.

It will be shown that when our models are placed in an environment,and assigned (in a way described later) a range of "normal" internal states,that they will acquire a level of organization that can be compared only to that of the highest animals. Furthermore,this capacity for self-organization will in general,not be lost after injury,unless the injury is such as to change the gross topology of the system,e.g., if one of the basic nets is removed entirely,or if one of the gross connections is entirely destroyed. Thus these methods provide an approach to the problems of (2) above.

In addition,these models also provide an approach to the problems of (1) above. For the models are so constructed that they resemble the brain-net only in its higher organization,but also on the level of physical structure. Most of the properties of the "cells" of the theory are based on properties established for neurons in the experimental literature. A few of the properties may only be described as plausible. This situation is inevitable with the present state of information about the nervous system;while some new properties have to be assumedhere,they have been made as simple,plausible,and few as seems possible. In every such case the assumptions are based on a plausible analogy with other biological situations. The geometry of the nets is also based on real

biological data,in this case on the evidence of neuroanatomy. The viewpoint that the basic ingredients of the system can be taken as large unorganized "random" nets can be justified in several ways. The nets of the brain,for the most part,appear quite disorderly at the level of interconnections between cells.(There are exceptions to this,but they are usually confined to regions associated with certain special activities,and need not be considered in a theory of this generality.) There is no evidence of anything like the critically orderly connections of a modern computer. As the power of the microscope is reduced,order is perceived,and for the gross brain a pattern of a small number of discernable "regions" and distinguishable bundles of connections can be seen. The evidence provided by surgery and neuropathology support this picture. Also,it can be argued that the organization of biological structures in general cannot be too complex,without some process of self-organization;recent estimates (Quastler 1953, 265 ff) of the information carried by the genetic determiners may mean that tissues can be organized only along general plans;there is not enough information to determine many individual connections (unless it were done in regular patterns,which it is not.). Finally,as many of the results of this paper are to a high degree independent of the exact connection structure (on the"local" or "microscopic" level within a single random net),it is not necessary for us to specify this structure to any large extent.

In order to analyse the "behavior" of the brain models,itiis necessary to introduce a number of "learning-theoretic" notions. In this paper,the most prominent of these ideas are those of "reinforcement operator" and "reinforcement process". Because these ideas are basic to the present analysis,and are,in addition,very closely related

to a well-developed body of contemporary psychological theories, a separate chapter is devoted to this study. In a reinforcement process, the reactions of a system to external stimuli are originally a matter of chance. But the result, or immediate consequence, of each reaction is given a valuation, and this valuation determines the form of an operator which is applied to the system. If the valuation is "high", the effect is to raise the dependability of the associated reaction. Thus we have a sort of "trial and error" process.

It turns out that certain assemblies of random nets are capable of realizing this kind of process, if the valuation and reinforcement operator are controlled by an external "trainer". Then an important step is taken in showing that in assemblies of a very few nets, an "internal" or "secondary" reinforcement system can be made to evolve entirely within the net system; starting with a very simple primitive valuation system (such as is the basis of simple "reward-punishment" schemes of animal training) these assemblies reinforce themselves whenever any of a small distinguished set of stimuli occur. They learn to apply reinforcement also to behavior patterns which lead to the occurrence of these stimuli, as well, and can organize themselves to do this on higher and higher levels. It can then be seen that such systems, which initially have very little organization, evolve complex behavioral patterns which exploit the structure of their environment (or any environment which contains an appropriate degree of regularity) so as to force the occurrence of environmental events which have a high valuation in the reinforcement structure that has evolved within the system. Thus the system displays behavior which has, undisputably, the characteristics of both "goal-oriented" and "need-oriented" motivations. By relating the initial, primitive,

valuation to the "internal physiological state" of the system,in such a way that reactions which bring this "state" toward its "normal" value,the overall evolution of the system will be made to tend toward the establishment of behavioral patterns which are effective in satisfying the "physiological needs" of the system.

In additionato the highly developed reinforcement systems acquired by these assemblies, another process occurs which exhibits the features of what might be called "simple associative learning". (The system thus provides a model for theories of the "contiguity" group of contemporary theories of learning,and perhaps indicates how the controversy between the "reinforcement" and "contiguity" schools may have to be resolved.) Assemblies with no more than three or four nets are shown to have the capacity for organization into much more advanced activity;they are capable of "considering" alternative actions,making an estimate of the consequences of eahh alternative (using previously acquired information about the regularities of the environment) and preforming or rejecting actions on the basis of such an estimate. There is no evident limit to the degree of complexity of behavior that may be acquired by such a system. Their development will depend to a great extent on the environment (including here the physical body) in which it is embedded,the sensory and motor channels with which it is provided,and on the early experiences to which it is subjected.

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The paper is organized as follows:

The present chapter is a general introduction.

Chapter 2 is devoted to the study of the logical structure of neural nets. The central question is that of what kinds of behavior can be obtained from nets which contain only cells which satisfy certain postulates. While some of the results obtained here are more general than is actually required for the sequel, they throw some light on the question of neural inhibition. The biological data is so sparse in this area that it seemed appropriate to exploit the mathematical aspects of the problem, so as to best utilize the available information.

Chapter 3 discusses the neurophysiological basis for the systems of this theory. While there is very little information available about the properties of the cells of the central nervous system, what information exists strongly indicated that the nerve impulse mechanism is the same as for peripheral nerve. However, for reasons discussed in chapter 3, it is likely that there is a large "noise" component for activity at the interneural junctions. Accordingly, it is assumed that the properties of the junctions of our nets are like the excitability properties for peripheral nerve (which are well-established) except that a probabilistic uncertainty is attached to the classical notion of excitability "threshold". Thus the artificial axioms of chapter 2 are replaced by a set of biologically very plausible postulates.

Chapter 4 develops some of the learning-theoretic notions that will be required. "Reinforcement process" and "reinforcement operator" are defined, and a set of abstract "behavioral models" are examined. Each model has an abstract "environment" and we determine the extent

to which each model can exploit the structure of its environment. The notion of a simple "global" reinforcement process (which resembles the systems of contemporary "reinforcement theories of learning") and a related notion of "local" reinforcement are contrasted, and it is seen that the "global" notion does not provide a natural description for complex mechanisms. Therefore the "local" concept is used henceforth. A machine, the SNARC, has been constructed which realizes a local reinforcement operator, and its structure and behavior is described.

Chapter 5 is an analysis of the activity of closed re-entrant "cycles" of neurons. It is shown that under the neural postulates of chapter 3, certain special forms of neural activity, called F-active patterns, will be distinguished, in the behavior or a random net, by their peculiar persistence properties. In the absence of "noise" (defined here as pulses arising from outside the F-active pattern) the lifetime of such a pattern is extremely long, but in the presence of "noise" they are peculiarly fragile, and are destroyed, created, and mutated rapidly. It is seen that this fragility in the presence of noise is not dependent on the structure of the underlying net. The linking and mutual interference of these patterns are discussed, and their growth and destruction examined. It is shown that if it is not assumed that all the cells of the net have the same properties (and they certainly do not in the brain) then the net may be regarded, from the viewpoint of the theory of F-activity, as composed of more or less distinct "interval spectrum domains" and that an F-active pattern must remain in one such domain. Two F-active patterns in different domains cannot both survive if they happen to intersect in the net.

The chapter contributes to the general development of the theory of neural nets in that the "time quantization axiom", traditional in much of the work in this field is not used, and it is shown that

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It goes without saying that none of the above can be held in any way responsible for the innumerable weaknesses of this work, in particular,I must take full responsibility for the biological statements and conjectures.

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CHAPTER 2 FINITE AUTOMATIC NETWORKS

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FINITE AUTOMATIC NETWORKS

2/1 Terms like "neural network" or "nerve net" are used at present to denote the subjects of a number of theories, each of which represents an abstraction of some of the knowledge derived from contemporary neurophysiological theory. In this chapter we define a few such objects and establish some theorems on the equivalence of certain sets of axioms for such theories.

2/1.1 S. C. Kleene has defined a FINITE AUTOMATON as a generalization of some present theories. Because we have a different emphasis his system is presented in a slightly different form:

A FINITE AUTOMATON is a collection of elements called "cells" whose operation is determined by the following axioms:

F-1: TIME is "quantized" as a sequence of discrete moments (indexed by the integers).

F-2: CELLS: There are a finite number of cells, each of which admits of one of a finite, ≥ 2, number of states at any moment.

F-3a: Two kinds of cells are distinguished; INPUT CELLS and INNER CELLS.

F-3b: The state of an INNER CELL at a time t depends on the states of all cells at time t - 1.

F-3c: The state of an INPUT CELL at a time t is said to "depend on the environment".

F-3c means that the states of the "input" cells may be any function of time (of the integers), or be arbitrarily assigned by an "operator" of the automaton.[1]

2/1.2 The dependency relation of F-3 is entirely unspecified. There is one feature of the dependency relation that is common to all so-called "neural-network" theories. It is expressed by adding the following axiom to Kleene's system:

F-3n: There are a set of "CONNECTIONS" $\{C_{ij}\}$. A connection C_{ij} is said to "originate on cell C_i", and "terminate on cell C_j". There is a connection C_{ij} only for

---

[1] NOTE: Kleene restricts the input cells to take, at each moment, one of two states called 0 ("quiet") and 1 ("firing"). As he points out, one can always construct a logically equivalent finite automaton in which each cell has just two states, at the price of a uniform expansion of the time scale. However, the present theory is definitely not oriented in a logical-algebraic direction, and, replacing the axioms by a simpler equivalent system in which each cell has just two states would be an unnatural imposition.

certain pairs of cells (C_i, C_j). No connection terminates on any input cell.[1] The state of a cell C_j at a time t depends only on the states, at time t - 1, of those cells C_i for which there exists a connection C_{ij}.

Def. A system which satisfies F-1, F-2, F-3, F-3n will be called a "FINITE AUTOMATIC NETWORK".

2/1.3 A notable example of a finite automatic network is provided by the system of McCulloch and Pitts (1943). Axioms for this system are present in a form consistent with those in 1.1 and 1.2.

MP-1: MP-1 is F-1, the time quantization axiom.

---

[1] NOTE: F-3n states that no connection terminates on any input cell (which follows also from F-3c, if one regards a connection with no effect as vacuous). However, in the present theory, the "environment" of a given net will often be another net, and connections from the environment net will terminate on the input cells of the given net. The distinction between "input" and "inner" cells is to be regarded as a classification of a cell's position in a subnet, in relation to an observer's specification of which cells of a larger underlying net belong to the given subnet, and is not to be taken as a distinction between inherently different kinds of cells. At a later point, certain input cells will be designated as "receptor cells" (e.g., thermal receptors), and this designation will represent an inherent difference, or "specialization" of cells.

MP-2: MP-2 is F-2 with each cell restricted to two states, 0 "quiet" and 1 "firing".

MP-3: MP-3 is F-3n with the dependency law completely specified: The law can be given as follows (in a form arranged to match the F axioms):

Each connection C_{ij} has a numerical value which is either a positive integer or minus infinity. The value can be denoted by $\not\beta C_{ij}$. Let $\not\beta C_{ij} = 0$ in the case that there is no connection to C_{ij}.

Let $C_j(t)$ represent the function which has value 1 if C_j fires at time t and has value 0 if this is not the case. Let $C_j(t)$ also represent the proposition "C_j fires at time t".[1]

Finally, each cell C_j has a numerical "Threshold" $\not\#C_j$, which is a positive integer. The dependency law can then be stated as

$$C_j(t) \equiv \left[\sum_i C_i(t-1) \cdot \not\beta C_{ij} \geqslant \not\#C_j\right].$$

Note: In the MP theory, the cells are called "neurons".

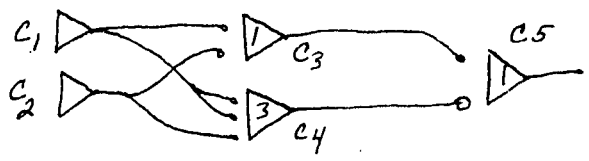
---

[1] This convention will be used throughout this paper.

If $\not\!\!C_{ij}$ is positive, we say that C_i has $\not\!\!C_{ij}$ "endbulbs" on C_j. If $\not\!\!C_{ij} = -\infty$, we say that C_i has an "inhibitory endbulb" on C_j. An example is provided to demonstrate the use of the network notation, and its description using the propositional calculus.

CELLS:	CONNECTIONS:
$\not\!\!C_3 = 1$	$\not\!\!C_{13} = 1$
$\not\!\!C_4 = 3$	$\not\!\!C_{14} = 2$
$\not\!\!C_5 = 1$	$\not\!\!C_{24} = 1$
	$\not\!\!C_{23} = 1$
	$\not\!\!C_{35} = 1$
	$\not\!\!C_{45} = -\infty$

DIAGRAM:



DEPENDENCY LAWS:

$$C_3(t) \equiv C_1(t-1) \vee C_2(t-1)$$

$$C_4(t) \equiv C_1(t-1) \cdot C_2(t-1)$$

$$C_5(t) \equiv C_3(t-1) \cdot \sim C_4(t-1)$$

THEOREM:

$$\therefore C_5(t+2) \equiv [C_1(t) \vee C_2(t)] \cdot \sim [C_1(t) \cdot C_2(t)]$$

None of the axioms above represent much of our real knowledge of neurophysiology. Certainly F-1, the time quantization axiom, is false. (In Chapter 5, we will discuss some consequences of making this false assumption.) The existence of specific inhibitor connections has not been established within the brain, although there is some evidence for them in the spinal cord.

2/1.4 The Rashevsky system seems, on the surface, more biological, since it requires cells to fire whenever local conditions are appropriate. It provides an example intermediate between the above Finite Automatic Networks, and the generalizations below.

R-1: No time quantization. Cells can fire at any time subject to the excitation axiom.

R-2: Each cell can have two states, quiet and firing. (Note: In Rashevsky's system, each cell has a continuously variable internal state function, "e-j", whose value is a function of time and of the history of pulses that have reached the cell through its connections.)

R-3: There are two kinds of connections, E_{ij} and I_{ij}, called "excitatory" and "inhibitory". The dependency law is somewhat as follows:

A cell fires when "e-j" exceeds some "threshold". The function "e-j" depends on the past stimulation in such a way that "e" increases with excitatory stimulation, and "j" increases with inhibitory. Both fall off exponentially with time.[1]

2/1.5 Let us consider a more general system (which includes all the above):

A NEURAL-ANALOG MACHINE is a collection of cells with the following axioms:

A-1 No time quantization.

A-2 Input cells are fired by the environment. Each cell has two "external states", "quiet", and "firing". Each cell has an infinite number of internal states, which are to be identified with its "pulse history"; the pulse history at time t, of cell C_j is the set of times up to and including t at which C_j has fired. (Firing is instantaneous.)

A-3 The firing of an inner cell depends on the pulse history of all cells.

---

1 See 2/7.2.2 We will not discuss this system in detail because the hypotheses are much stronger than necessary here.

Again a specialization is appropriate:

A NEURAL-ANALOG NETWORK is a collection of cells for which

A-1,

A-2, and

A-3n; There are a set of connections C_{ij}. The The firing of an inner cell C_j depends on the pulse history of all cells U_i for which there is a connection C_{ij}.

NOTE: These systems are more general than might appear. The form of the dependency may vary from cell to cell, etc. The form of the axioms makes them appear to contain one important commitment about the dependency, namely an "all-or-none" law for pulses. For the dependency law involves only the time-of-arrival of pulses, and there is no explicit dependence on any other property of a pulse. However, by allowing each cell to take one of a large number of states, and using a sufficiently complicated dependency law, one can construct a system equivalent to one in which pulses have, e.g., several degrees of amplitude and duration, and in which the effect of a pulse depends on these "form" parameters.

2/1.6 None of the above systems contains what may be considered the distinctive aspect of the present theory; the probabilistic firing condition. The following system will provide the structure underlying the machines considered in this thesis.

A STOCHASTIC NEURAL-ANALOG NETWORK is a system which satisfies the axioms:

A-1 (continuous time)

A-2 (Pulse-history internal state, hence bias toward all-or-none theory)

SA-3n For any given inner cell C_j, at time t, the probability distribution for the time of the next firing of C_j depends on the pulse history of each cell C_i for which there is a connection C_{ij}. NOTE: This distribution is a conditional probability on the hypothesis that the pulse histories of the C_i's do not change. Each time one of the C_i's does fire, a new distribution for the next firing time of C_j is applied.

2/1.7 DISCUSSION:

If, in application of SA-3n to the nervous system, we take the "cells" to be neurons, and the C_{ij} to be synapses, then a commitment is made which is involved with what is probably the most basic controversy in the field of physiological psychology.

For the subscript "n"[1] attached to an axiom is used to denote the assumption that the machine's components are related exclusively through discrete "connections", and that therefore the operation of the machine depends exclusively on this connection structure, and not on the spatial arrangements of the cells and synapses in their physical environment. (For any given machine, of course, the formalism could be adjusted by replacing any "geometric" influences by fictitious connections with appropriate dependency properties.)

It is by no means my intention to exclude the possibility that spatial relations are important in the operation of the nervous system. There are numerous theories that have been formulated by physiological psychologists in which so-called "field" effects are considered to be major functional entities. (e.g., Pavlov, Lashley, Gestalt theorists. Kohler's field theory for vision is the most developed such theory.) In my opinion, none of these theories has been advanced to the (primitive) level of describing how one such field affects another, or how they are associated with input and output of the machine. Nor

---

1 or "net"

are the physiological arguments for these field theories very impressive.

It is said that such theories are required to explain how the nervous system continues to function after physical injury, yet it appears that "fields" ought to be equally vulnerable to physical distortion; especially if they are to have the complexity presumably required to explain thinking.

If these "fields" are regarded as, say, containing their structure in topological form, rather than metrical, and as also having autonomous self-stabilizing properties, with the physical brain acting only as a sort of ether, then such a theory could indeed explain injury resistance. One cannot escape the feeling that Lashley (and Pavlov) has some such model in mind, but neither a physical basis is proposed, nor even a formal scheme as to how the field is related to behavior. All that is expressed is the hope that a "field theory" will be eventually constructed.

As for the "isomorphism" approach of Kohler, in which the fields associated with sensory events reflect in some almost geometric manner the sensory events themselves, it is hard to see how one can hope that even if, e.g., sensation in the visual areas is

in fact associated with "isomorphic" fields, that this will aid in the understanding of the rest of the nervous system. There certainly is a stage in brain function where the events bear little relation to the geometric structure of sensory events, e.g., in the assignment of spoken names of objects. At the "motor end" of the nervous system there is very little "isomorphism" left, but there has been a great deal of "computation". The physical similarities must be dropped somewhere in the process, and it has not been shown how their preservation can aid in the computation. In a paper on a possible mechanism for vision, McCulloch and Pitts (1947) suggest a process involving patterns geometrically related to the retinal pattern, and these neural patterns are used in making a computation about the geometric features of the retinal pattern. But if this mechanism were in fact the one operating in the human brain, it would not support the field-theorists' contention that the brain functions generally by the use of such fields. In fact the purpose of such a mechanism as that of McCulloch and Pitts is precisely to convert the input information into an abstract and profoundly non-geometric form, for the convenience of the rest

of the brain. We will return to this subject in a separate paper, where the argument can be more precise.

2/1.8 On the other hand it is quite possible that "fields", and in particular electrical, chemical, and mechanical gradients, do in fact play an essential role in brain function, although not necessarily as global patterns which represent in some way identifiable mental events. Two such possibilities are discussed in the present theory.

(1). "Microscopic" fields affecting the excitability of synapses, resulting from the metabolic and neural activity of cells in the neighborhood, and

(2). Larger fields affecting the activity within whole nets, resulting from electrotonic, chemical, or electric fields set up by the massive (or "synchronous") discharge of large groups of cells.

(1) is considered in the first part of Chapter 3. (2) is considered throughout Chapters 5 and 6. The larger fields play a rather important role in these chapters, hence in spite of the combinatorial form of the earlier chapters, the final theory is as much a "field theory" as it is a "network theory", to use terms often employed in the present psychological controversy.

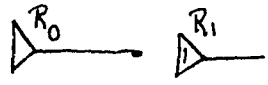
The present theory also avoids the difficulties

encountered by those network theories which use time quantization. This matter is discussed in Chapter 5.

2/2. Some neural nets and their function spaces.

Most of the nets in this section can be represented in the McCulloch-Pitts (1943) system, except where probabilities are used. While time quantization is not imposed, we assume a "unit synaptic delay" which serves much the same purpose.

2/2.1 UNARY JUNCTION, XP'a.



Two cells R_0 and R_1. One connection C_{01}. Dependency relation: $R_1(t) \equiv R_0(t-1)$.

This expresses the notion of "unit synaptic delay". It is used only for convenience, and dismissed in Chapter 5.

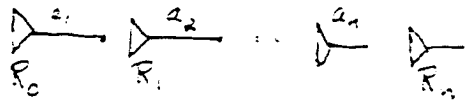
In the probabilistic case,

$\text{prob}[R_1(t)] = a \cdot R_0(t-1)$. Here "a" is the "transmission probability" or "XP" of the connection C_{01}. ("$R_0(t)$" is used here also as the truth value of the proposition $R_0(t)$.)

2/2.2 CHAIN OF UNARY JUNCTIONS.

$n + 1$ cells; $R_0, \ldots, R_n$. n connections $C_{i-1,i}$. If the XP of $C_{i-1,i}$ is a_i, then

$$\text{prob}[R_n(t)] = \prod_{i=1}^{n} a_i \cdot R_0(t-n).$$



This is called a "unary chain of length n" (n complete junctions).

2/2.3 Dis-junctions.

n + 1 cells; $R_0,\ldots,R_n$.

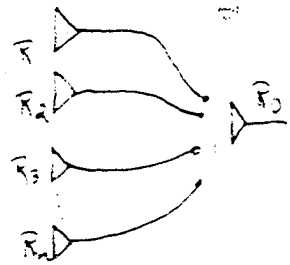
n connections; $c_{10}, c_{20},\ldots,c_{nc}$.

prob $R_0(t) = F[R_1(t),\ldots,R_n(t)]$.

We want to find a probabilistic analogy of the MP situation with threshold unity. A choice that suits the purposes of this chapter would be $F(R_1,\ldots,R_n) = a$ if not all variables are zero, $F = 0$ otherwise. Then MP = a for the junctions. See below; 2/2.5.

2/2.4 Con-junctions.

Same net as in 2/2.3



Let $F = 0$ if not all of $R_i(t-1)(i \neq 0)$

$= a$ if they all fire.

This is an analog of the MP junction with threshold n.

2/2.5 DISCUSSION

The elements of 2/2.4 and 2/2.3 are rather implausible physiologically. The argument in favor of a probabilistic transmission is to be found below in 3/1. In the light of the general arguments of that section, it would seem more natural to assume that the probability of firing a junction would, in general, increase with increasing stimulation. Hence, while the values of P (of $\mathfrak{P}$) are chosen in 2.3 and 2.4 to be very simple, in each case P should be thought of as a monotone increasing function of its (propositional) variables. The following arguments have this in view. Of course, if arbitrary monotone functions were permitted, then the functions of 2.3 and 2.4 would be the same. In order that they resemble the logical functions after which they are modeled, P must be chosen so that in case 2.3 the value of P is close to ~~unity~~ when any one input is fired, and in case 2.4, the value of P must be low unless all are fired. For the non-probabilistic cases, in 2.3, $\#'_0$ is 1 and in 2.4, $\#'_0$ is n.

2/2.6 Response functions

A net is described by cataloguing its input cells $\mathfrak{i}_i$, its inner cells $\mathfrak{n}_j$ and its connections $\mathfrak{c}_{mn}$ where m ranges over all the i's and j's,

while n ranges over the j's only.

STIMULUS is described by a set of pairs (i,s) where (k,t) ε (i,s) if and only if $S_k(t)$ is part of the stimulus. We [illegible]

When a stimulus is represented by a single letter S, the symbol will represent either the set of index pairs, or the proposition

$$\bigcup_{(k,t)\,\varepsilon\,S} S_k(t)$$

Stimuli will often be associated with a time index. If S(0) is a given stimulus, then

$$S(t_0) = \bigcup_{S_k(t)\,\varepsilon\,S} S_k(t + t_0).$$

Changing the time index translates (in time) the entire stimulus. We will require that

$$S_k(t)\ \varepsilon\ S(t_0) \Rightarrow t \geqslant t_0.$$ [1]

The behavior of a net N is described by its RESPONSE FUNCTION Res(S). The domain of Res(S) is the set of all stimuli, i.e., of all subsets of I x T.[2] Its range is the subsets of J x T and it

---

[1] No pulse of a stimulus can occur before the index time of the stimulus.

[2] I is the index set of the input cells, J that of the inner cells.

- is defined as follows: Assuming that the net is quiet when $t < 0$,

$$\mathrm{Res}(\mathfrak{Z}) = \lambda[(k,t) \;/\; \mathfrak{Z} \Rightarrow R_k(t)]\nu.$$

2/2.7 THEOREM: (2/2.7)

<u>If a net is composed entirely of dis-junctions, then for any stimulus</u> $\mathfrak{Z} = \mathfrak{Z}(0)_1$[1]

$$\mathrm{Res}(\mathfrak{Z}) = \bigcup_{(\gamma,t)\varepsilon\mathfrak{Z}} \mathrm{Res}(\mathfrak{Z}\gamma(t)).$$

Proof:

Consider any element $R_k(t)$ of $\mathrm{Res}(\mathfrak{Z})$. Since $R_k(t)$ is an inner cell, $t \geqslant 1$. Now $R_k(t)$ must be fired by either an inner cell or an input cell. Hence,

$$R_k(t) \equiv \lambda[(\exists k')(\not\exists C_{k'k} \neq 0 \cdot R_{k'}(t-1))]\nu\, \lambda[(\exists i)(\not\exists C_{ik} \neq 0 \cdot S_i(t-1))]\nu.$$

If the second proposition on the right holds, then $R_k(t) \varepsilon \mathrm{Res}(\mathfrak{Z}_i)$. If not, the second must hold, and $R_{k'}(t-1)$.

By iterating the same argument T times, where T is the largest integer in t, we have: If $R_k(t)$ is not in any $\mathrm{Res}(\mathfrak{Z}_\nu(t-j))$, $j = 1,2,\ldots,T$, then,

$$(\exists k^{(T)})\left(\not\exists C_{k^{(T)}k}(T-1) \neq 0 \cdot R_{k^{(T)}}(t-T)\right) \quad 7$$
$$\vee(\exists i)\left(\not\exists C_{ik}(T-1) \neq 0 \cdot S_i(t-T)\right).$$

---

1 No pulse of a stimulus can occur before the index time of the stimulus.

The first proposition is false because $\psi_k(x) \Rightarrow x \geqslant 1$. But if the second holds, then by the inductive argument, there exist connections $C_k l_k, C_k 2_k l, \ldots, C_{l_k}\lceil T-1 \rceil$. Also, $S_i(t-\lceil T$. But then $R_k(t) \varepsilon \mathrm{Res}(S_i(t-\lceil T'))$, contrary to the assumption. Since $R_k(t)$ is any element of Res(S), we have

$$\mathrm{Res}(S) \subset \bigcup_{(i,t)\varepsilon S} \mathrm{Res}(S_i(t)).$$

The reverse argument is a special case of theorem 2/2.8 below.

2/2.8 For the probabilistic case we define

$$\mathrm{Res}(S) = \lceil (k,t,a)\ S \supset \mathrm{prob} R_k(t) = a \rceil.$$

If R and R' are responses, we say that $R' \supset R$ if, and only if,

$$(k,t,a) \varepsilon R \Rightarrow (\exists b)_{b>q} (k,t,b) \varepsilon R').$$

Theorem: If a net is composed entirely of junctions with monotone[1] transmission functions (see 2.5) then

$$\mathrm{Res}(S) \supset \bigcup_{(i,t)\varepsilon S} \mathrm{Res}(S_i(t)).$$

In fact, more generally,

---

[1] By "monotone" we always mean "non-decreasing".

Theorem: (2/2.9)

$$J' \supset J \Rightarrow Res(J') \supset Res(J).$$

i.e., a net composed of monotone functions has a monotone response function.

PROOF: Choose a $(k,t,a) \in J$. Now

$a = prob(C_k(t)) = F_k(C_{k_1}(t-1),\ldots,C_{k_p}(t-1))$

where the C_{k_i} are the cells which connect to R_k, and F_k is the monotone function of p binary variables which describes the transmission properties of R_k. Replace F_k by the function $F'_k(C_1(t-1),C_2(t-1),\ldots,C_n(t-1))$ of all cells which has the same values as F_k. F'_k is also monotone.

Now if $prob C_1(t-1) = a_1$, then

$$a = prob(C_k(t)) =$$

$$\sum_{j_1,j_2,\ldots,j_n=0}^{1} \left[\prod_{i=1}^{n} a_i^{j_i}(1-a_i)^{(1-j_i)}\right] F'(j_1,j_2,\ldots,j_n).$$

$$= a_1 \sum_{j_2,\ldots,j_n=0}^{1} \prod_{i=2}^{n} a_i^{j_2}(1-a_1)^{(1-j_1)} F'(1,i_2,\ldots,i_n)$$

$$+(1-a_1)\sum \prod F'(0,i_2,\ldots,i_n)$$

$$= a_1\Sigma_1 + (1-a_1)\Sigma_2$$

$$= a_1(\Sigma_1 - \Sigma_2) + \Sigma_2$$

Now, because F' is monotone, each term of Σ_1 is $\geq$ the

corresponding term in Σ_2. Hence, $\text{prob}R_k(t) = a_1(\Sigma_1 - \Sigma_2) + \Sigma_2$ is monotone for a_1, and similarly for each of the a_i. This proves that for any i and j, increase of $\text{prob}R_i(t-1)$ cannot decrease $\text{prob}(R_j(t))$. By induction, increasing $\text{Prob}(R_i(t-1))$ cannot decrease $\text{prob}(R_j(t+d))$ for any $d \geq 0$. (It certainly cannot decrease $\text{prob}(R_j(t+d))$ for negative d; the causality is always forward in time.)

Now, since $S' \supset S$,

$$S' = S \cup Su_1(v_1) \cup Su_2(v_2) \cup \ldots \cup Su_i(v_i) \cup \ldots Su_z(v_z)$$

where the $Su_i(v_i)$ are the elements of S' not in S. Let $S^1 = S \cup Su_1(v_1)$, and inductively, $S^k = S^{k-1} \cup Su_k(v_k)$. Then $S^z = S'$, and $S = S^0$.

Now $\text{Res}(S^n) \supset \text{Res}(S^{n-1})$. For if $(k,t,a) \varepsilon \text{Res}(S^{n-1})$, the firing of $Su_n(v_n)$ cannot decrease $\text{prob}R_k(t)$. Hence for some $b \geq a$, $(k,t,b) \varepsilon \text{Res}(S^n)$.

Hence

$$\text{Res}(S) \subset \text{Res}(S^1) \subset \ldots \subset \text{Res}(S'). \quad \text{Q. E. D.}$$

Corr. If the monotone functions F_k are all of the form: F_k is unity if (and only if) any variable is not zero, and F_k is zero otherwise; then we have a net of (determinate) $\phi\varepsilon$-junctions, and the theorem in this case completes the proof of theorem 2/2.7.

NOTE: Theorems 2/2.7 and 2/2.8 are of particular interest because there is no restriction made on

the topology of the net, and cycles with infinite responses (see Chapter 5) are admitted. Theorem 2/2.7 can be read; nets with additive junctions have additive response functions. Theorem 2/2.8 states that nets with monotone junctions have monotone response functions.

2/3 REALIZABILITY OF OUTPUT FUNCTIONS.

In this section we examine the question of which functions can be realized by nets of given composition.

We distinguish three varieties of cells: input, I_i, inner, I_j, and output R_k. An <u>output function</u> is like a response function except that it describes only the activity of the output cells, and not the inner cells.

Let T_S be the time of the first pulse in stimulus S.
" T^S " " " " " last " " " ".
" T_P " " " " " first " " output P.
" T^P " " " " " last " " " ".

<u>Definition</u>: An output function P(S) has delay $\geq$ if for all S,

$$T_P(S) \geqslant T_S + d$$

<u>Definition</u>: An output function P(S) has ~~duration $\leq L$~~ length L if for all S,

$$T^P(S) \leqslant T^S + d$$

<u>Definition:</u> A simultaneous input volley at time t_0 (an $(S.I.V.)_{t_0}$) is a stimulus S for which $T_S = T^S$.

Note: Whenever it is to be shown that a Net N realizes a given output function $F(S)$, we will use the symbol $FN(S)$ to denote the output function of the net N, and show that $FN(S) = F(S)$.

2/3.1 Theorem (2/3.1)

Let $F(S)$ be a function of an S.I.V. which is additive, has finite duration, delay≥ 1 and for which $F(0) = 0$. Then $F(S)$ can be realized by a net composed entirely of dis-junctions:

Proof: Let $T_S = 0$ without loss of generality. Represent $S = \bigcup_{S_i(0) \in S} S_i(t)$.

Let S_i and R_k be input and output cells for N. For each $R_k(t)$ in $F(S_i)$ construct a chain of inner cells starting at S_i, with t-1 inner cells and terminating on R_k.

All junctions are dis-junctions (N-P junctions with threshold unity). Then for each $R_k(t)$ in $F(S_i)$ there is a path with delay t from S_i to R_k, hence

$$FN(S_i) \supset F(S_i)$$

and since these are the only paths from S_i to any output cells, $FN(S_i) = F(S_i)$. Also, by theorem 2/2.7, $FN(S)$ is additive. Since the values of $FN(S_i)$, and the fact that it is additive, determine a unique additive function $FN(S)$, it must be identical with $F(S)$ which satisfies the same conditions.

NOTE. Without the restriction that P(3) have finite duration, some S_i might need to have an infinite number of sedbulbs. The condition P(0) = 0 is a property of any realizable response function for nets not previously active.

2/3.2 THEOREM 2/3.2

Using only con- and dis-junctions, a net can be constructed to realize any monotone output function F of an S.I.V. for which F has finite length, delay ≥ 2 (and P(0) = 0).

Proof: Let $T_S = 0$. Construct the net N as follows: Let A be the index set for the S_i's. For each subset b of A supply an intermediate cell I_b, and a connection C_{ib} from S_i to C_b for each $i \in b$. Let I_b be a con-junction. (I_b is a MP neuron with threshold $\#I_b = \sum_{i \in b} 1$.) Then

$$(3 \Rightarrow I_b(1)) .\equiv. 3 \supset \bigcup_{i \in b} S_i(0).$$

Let $S_b(0) = \bigcup_{i \in b} S_i(0)$.

Now for each b and each (j,t) for which $R_j(t) \in P(S_b(0))$, construct a chain of cells of total number t, the first of which is I_b and the last of which is R_j. Let all junctions in such chains be ~~con~~-junctions; each R_j will be a multiple junction with threshold $\#R_j = 1$. Now the pathways

we have constructed insure that

$$PN(S_b) \supset P(S_b). \qquad (1)$$

On the other hand, suppose that $R_k(t) \varepsilon PN(S_b(0))$. Then, $R_k(t)$ was fired by a pulse arriving along a chain which originates on either I_b or on some I_c for which $c \in b$. For all chains originate on some I_x and only the above ones can fire in response to $S_b(0)$. Now since P is monotone, $R(S_b) \supset R(S_c)$, hence such a chain can exist if, and only if, $R_k(t) \varepsilon R(S_b)$. Thus

$$P(S_b) \supset PN(S_b). \qquad (2)$$

Hence the net N realizes the function P.

2/3.3 DISCUSSION: Serious difficulties appear when generalization of the above theorems (2/3.1 and 2/3.2) is attempted for more general inputs. This is due partly to the fact that, as the nets have no internal clock, their responses to time-translated inputs must have time-translated outputs. Thus one cannot construct arbitrary monotone functions over any class of inputs which contains any two patterns which are time translates. Theorem 2/3.4 below refers to a class of inputs which cannot have any such pairs.

The fact of translation in variance plus the superadditivity of nets of monotone junctions might be expressed by saying that a principle of

superposition operates in these nets.

Definition: An admissible class II of stimuli is one which is finite, and contains no pair of time-translated stimuli.

2/3.4 Consider the class II(a,b) of stimuli, defined by the relations $S \in II(a,b)$ if, and only if

1. $S_i(t) \in S \Rightarrow a \leq t \leq b$
2. $(\exists_i)\ S_i(a) \in S$
3. $(\exists_j)\ S_j(b) \in S$

It may be seen that II(a,b) cannot contain a pair of translated patterns, and is an admissible class.

Definition: An output function P of stimuli in II(a,b) is said to have delay d if

$$R_k(t) \in P(S) \Rightarrow t \geq a+d.$$

THEOREM 2/3.4 Using only con- and dis-junctions, a net N can be constructed to realize any monotone output function which is defined only on II(a,b), has finite length, and has delay $\geq 4+b-a$[1]

Proof: It is sufficient to prove the theorem for the cases II(0,b). Other cases are time-translates of these.

Construct a net with the following cells and connections:

---

[1] For $II_b(b)$ the delay can be $\geq 2+b$ by theorem 2/3.2

CELLS:

$S_1, \ldots, S_n$ are the given input cells.

Inner cells: I_{ij} and S_{ij}; $i = 1,2,\ldots,n$. $j = 0,1,\ldots,b$.

Two additional inner cells I_0 and I_b.

CONNECTIONS:

$S_i \longrightarrow I_{i1}$ all i.

$I_{i,j+1} \longrightarrow I_{ij}$ all i, all $0 \leq j < b$.

$I_{ij} \longrightarrow S_{ij}$ all i,j.

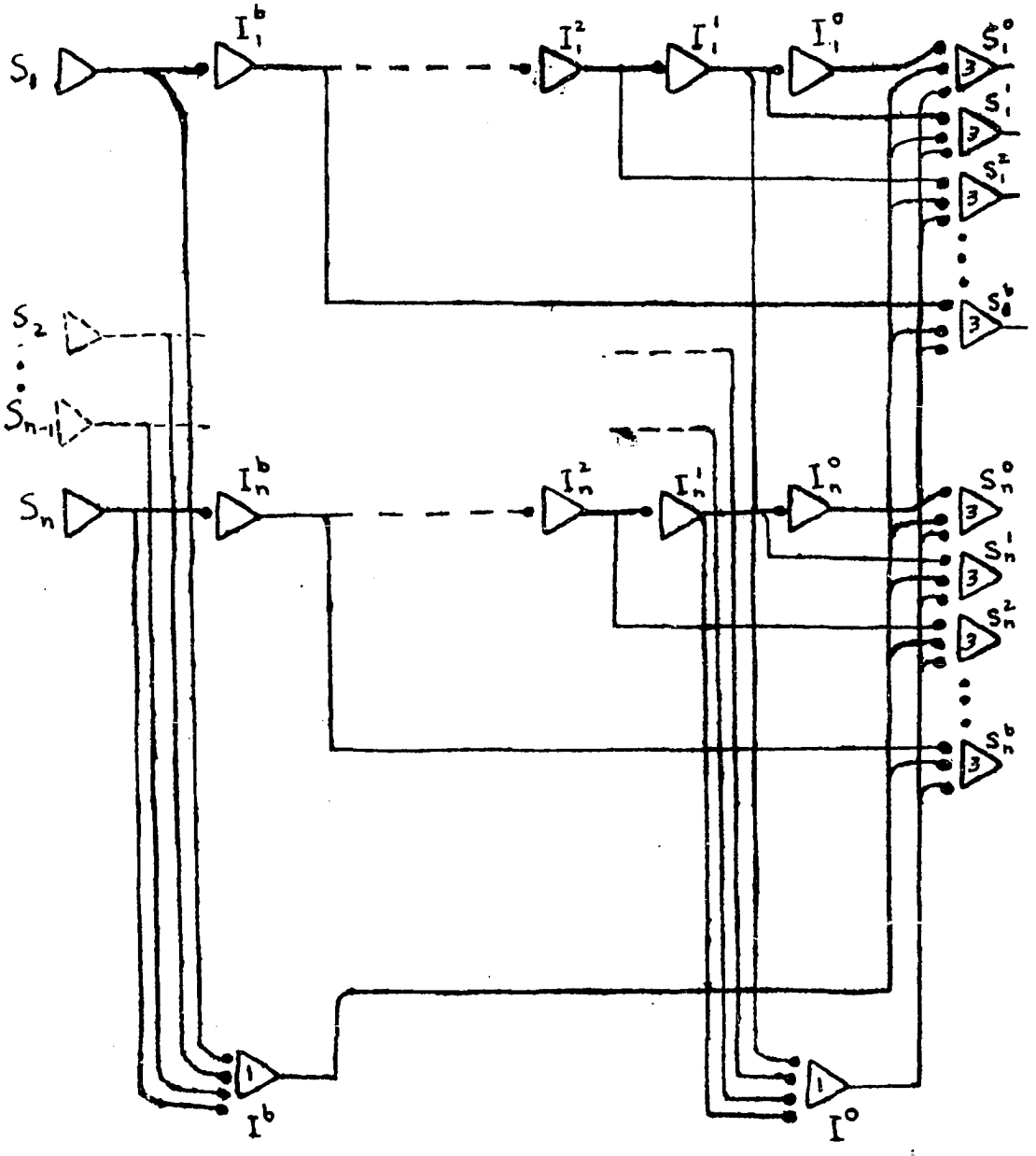
$I_b \longrightarrow S_{ij}$ all i,j.

$I_0 \longrightarrow S_{ij}$ all i,j.

$S_i \longrightarrow I_b$ all i.

$I_{i1} \longrightarrow I_0$ all i.

This net is represented on the following page, using the McCulloch-Pitts (1943) symbolism.



THE NET OF THEOREM 2/3.4

Now $S_i^0(t) \equiv I_i^0(t-1) \circ I^0(t-1) \cdot I^b(t-1)$

and $S_i^x(t) \equiv I_i^x(t-x-1) \circ I^0(t-1) \circ I^b(t-1)$

Also, $I^b(t-1) \equiv (\exists i)(S_i(t-2))$ and

$I^0(t-1) \equiv (\exists j)(S_j(t-b-2))$. These two conditions can hold if and only if $t = b + 2$. Hence,

$$S_i^x(t) \equiv I_i^x(b+1) \quad S_i(x).$$

All cells are dis-junctions (threshold 1) except for the S_{ij} which are con-junctions with threshold $\#S_{ij} = 3$.

From the given net, we can see that

$$S_{ix}(t) \equiv I_{ix}(t-1) \cdot I_0(t-1) \cdot I_b(t-1), \qquad (1)$$

for S_{ix} has threshold 3, and I_{ix}, I_0, I_b are the only cells which connect with S_{ix}. Furthermore,

$$I_b(t-1) \equiv (\exists i) S_i(t-2) \qquad (2)$$

and

$$I_0(t-1) \equiv I(\exists i) I_{i1}(t-2). \qquad (3)$$

But

$$I_{i1}(t-2) \equiv I_{i2}(t-3) \equiv \ldots \equiv I_{ib}(t-b-1) \equiv S_i(t-b-2), \qquad (4)$$

and (3) and (4) imply

$$I_0(t-1) \equiv (\exists i) S_i(t-b-2). \qquad (5)$$

Conditions (2) and (5) can be simultaneously satisfied (as is required for (1) to hold) only when $t = b+2$. And the fact that $S \in \Pi(0,b)$ insures that this will be the case. Hence

$$S_{ix}(t) \equiv [t = b+2 \cdot I_{ix}(t-1)] \qquad (6)$$

Also,

$$I_{ix}(t-1) \Rightarrow I_{i\,\underline{x+1}}(t-2) \Rightarrow \ldots \Rightarrow I_{ib}(t-b+x-1) \Rightarrow S_i(t-b+x-2)$$

Hence

$$S_{ix}(t) = 0 \text{ if } t \neq b+2 \qquad (7)$$

and

$$S_{ix}(b+2) \equiv S_i(x). \qquad (7')$$

Equations (7) and (7') express the important feature of the net. At time t = b+2, there is displayed on the cells S_{ij} a simultaneous volley which contains all the information about the stimulus S. To complete the proof of the theorem, one needs only to adjoin a net of the type described in theorem 2/3.2 using the S_{ij} as the input cells of the new net.

A function of elements of II(0,b) is a function of sets of pairs $(i,j).\begin{pmatrix} i = 1,\ldots,n \\ j = 0,\ldots,b. \end{pmatrix}$ Replace the sets of pairs by sets of the corresponding events $S_{ij}(b+2)$. Any monotone function on II(0,b) with delay ≥ 4 can be realized by a monotone function on sets of the $S_{ij}(t+2)$ with delay ≥ 2. Since any monotone function of sets of the $S_{ij}(t+2)$ with delay 2 can, by 2/3.2 be realized by a net with the S_{ij} as input cells, the adjoining of such a net to the above will give a net which realizes the corresponding monotone function of II(0,b).

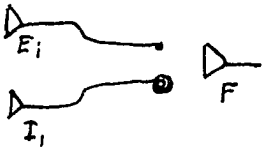
NOTE. The necessary condition P(0) = 0 must hold, but need not be stated, since neither II(a,b), nor any other admissible class, contains the 0 stimulus.

2/4 INHIBITORY CONNECTIONS

2/4.1 Inhibitory connections of the McCulloch-Pitts type were described in 2/1.3; an inhibitory connection I_{ij} has the property that if an inhibitory connection originates on a cell C_i and terminates on a cell C_j then $C_i(t) \Rightarrow \sim C_j(t+1)$ regardless of what other connections may terminate on C_j.

For reasons that will be clear in 2/5, we have a special interest in binary inhibitory junctions (b.i.j's):

DEFINITION: A <u>binary inhibitory junction</u> (b.i.j.) is a cell F on which terminates exactly one excitatory E_1 and one inhibitory connection I_1: $F(t) \equiv E_1(t-1) . \sim I_1(t-1)$.



2/4.2 Given nets with inhibitory connections, as well as with con- and dis-junctions, one can construct a larger class of response functions.

DEFINITION: Let $C_1, \ldots, C_n$ be the input cells of a net N. <u>The class $\Pi_a(b)$ of stimuli is</u>

$S \in \Pi_a(b) \equiv C_1(t) \in S \Rightarrow a \leqslant t \leqslant b \;.\; (\exists i)(C_i(a) \in S)$.

(This is the class of stimuli which begin with one or more pulses at $t = a$, and for which firing ceases after $t = b$. It is an admissible class (see 2/3.3).) Note difference between $\Pi_i(a)$ and $\Pi(a,b)$

$S \in \Pi(a,a) \Rightarrow S \in \Pi_a(b)$

THEOREM 2/4.2:

Using only con-junctions, dis-junctions, and binary inhibitory junctions, a net can be constructed to realize any output function F defined on $II_a(b)$ with delay $\geq b \cdot 4 - a$. If any number of inhibitory connections are permitted to terminate on a con-junction, the delay may be cut to $\geq b \cdot 2 - a$. For the class $II_a(n)$, the delay may be cut to 3 and 2 in the two cases.

PROOF: Because any such net must be time-translation-invariant, it is sufficient to prove the theorem for the classes $II_0(b)$. There are two cases; $b = 0, b \geq 1$.

2/4.2.1 Case 0: The procedure is like that in Theorem 2/3.2. Let $A = (i/i = 1,\ldots,n)$ = the set of indices of the S_i. For each subset $x \subset A$ supply three cells E_x, I_x, and F_x. E_x is a conjunction with threshold $\sum_{i \in x} 1$, and on E_x terminates a connection from each S_i for which $i \in x$. I_x is a dis-junction (threshold 1) on which terminates a connection from each S_i for which $i \notin x$. F_x is a b.i.j. on which terminates one excitatory connection from E_x and one inhibitory connection from I_x. Then, if

$$S_x = (S_i(0))(i \in x), \quad \text{then,}$$

$$F_x(2) \equiv \langle E_x(1) \cdot \sim I_x(1) \rangle \equiv \langle i \in x \equiv S_i(0) \rangle \equiv S_x.$$

or

$$F_x(2) \equiv S_x.$$

Now, for each $R_k(t) \in P(S_x)$ construct a chain of disjunctions beginning with F_x, ending with a connection on R_k, and containing $t-3$ intermediate cells $C_{x,n}(n = 1,..,t-3)$. This is possible if $t \geq 3$ and this will hold if $N(3)$ has delay ≥ 3.

Now,

$$[R_k(t) \in P(S_x)] \Rightarrow$$
$$F_x \Rightarrow F_x(2) \Rightarrow F_{x1}(3) \Rightarrow F_{x2}(4) \Rightarrow \ldots$$
$$\Rightarrow \ldots \Rightarrow F_{x\ \underline{t-3}}(t-1) \Rightarrow R_k(t).$$

Hence

$$P(S_x) \subset N(S_x). \qquad (1)$$

On the other hand, $R_k(t) \in N(S_x)$ means that $R_k(t)$ is fired by a pulse from $F_x(2)$, since pulses can get to the R_k's only through the intermediate chains which originate on the F_x's, and $F_x(2) \equiv S_x$. But then $R_k(t) \in P(S_x)$, by construction of the chains.

Hence,

$[R_k(t) \in N(S_x)] \Rightarrow [R_k(t) \in P(S_x)]$, or

$$N(S_x) \subset P(S_x). \qquad (2)$$

NOTE: If free use of inhibitory connections are permitted, we can replace E_x, I_x, and F_x by a con-junction G_x with threshold $\sum_{i \in x} 1$ on which terminates an excitatory connection from each S_i for which $i \in x$ and an inhibitory connection from each S_i for which $i \notin x$.

Then $T_x(1) \equiv S_x$. The remainder of the net is constructed as above, but due to the removal of one computation level, the functions may have any delay ≥ 2.

This concludes the proof for case 0.

2/4.2.2 $b \geq 1$.

For $b \geq 1$ we construct a net in two parts; the first part converts the sequential stimulus into an equivalent simultaneous volley, much as in the proof of theorem 3/2.4; the second part of the net is a copy of the net of case 0, which converts the simultaneous volley (at time b+1) into arbitrary outputs. The sequential net is constructed as follows:

OMEGA: $\Omega_i \quad i = 1,2,\ldots,n$

$S_{ij} \quad i = 1,2,\ldots,n \quad j = 0,1,\ldots,b$

$T_{ij} \quad i = 1,\ldots,n \quad j = 0,\ldots,b$

NOTE: $T_{i0} = S_i$.

$V_k \quad k = 0,\ldots,b-1.$

EXCITATORY CONNECTIONS:

$k \neq 0$

$S_i \longrightarrow V_{b-1}$ all i

$V_0 \longrightarrow S_{ij}$ all i,j.

$T_{ij} \longrightarrow S_{ij}$ all i,j.

$T_{ij} \longrightarrow T_{i,j-1}$ all i, all $j \geq 1$.

INITIAL CONNECTIONS

$V_0 \longrightarrow S_{1j}$ all 1, all $j < b$.

Note that there is just one multiplicity and one antidromic fibre on each of these S_{1j} $(j < b)$, hence they are all b.i.j's.

Thus:

$\#V_k = 1$

$\#S_{1j} = 1$

$\#T_{1j} = 2$ Thus the S_{1j}'s are conjunctions, the others are dis-junctions or b.i.j's.

Lemma: If $j \in II_0(b)$,

Then,

$S_1(j)(t) \equiv S_{1j}(b+1)$, and $T_{1j}(t) \equiv t \equiv b+1$.

Proof: The connection scheme reveals that

(1): $S_{1j}(t) \Rightarrow V_0(t-1) \Rightarrow \ldots \Rightarrow V_{b-1}(t-b)$

$\Rightarrow (\exists k)(S_k(t-b-1))$

$\Rightarrow 0 \leqslant t-b-1 \leqslant b$

$\Rightarrow t \leqslant b+1$

(2): $j \in II_0(b) \Rightarrow (\exists i)(S_1(0)) \Rightarrow V_{b-1}(1) \Rightarrow \ldots \Rightarrow V_0(b)$

$\Rightarrow \sim T_{1j}(b+1) \quad 0 \leqslant j < b$

(3a): $S_{1j}(t) \Rightarrow T_{1j}(t-1) \Rightarrow \ldots \Rightarrow T_{1\ j+s}(t-s-1) \quad (0 \leqslant s \leqslant b-j)$

(3b): $\Rightarrow T_{1b} \equiv S_1(j+t-1-b)$

(hence $0 \leqslant j+t-1-b \leqslant b$).

Now, let $x \equiv t-b-1$. Suppose that $x \geqslant 0$,

Then, $S_{ij}(t) \Rightarrow$ (by 3b)

$$j \leqslant j+x = (j+t-b-2) < b \quad \text{or} \quad 0 \leqslant x < b-j \qquad (4)$$

Hence by 3a,

$$\sim T_{ix}(t-t+b+2-1) = T_{ix}(b+1) \qquad (5)$$

However, by (2), and (4),

$$\sim T_{ix}(b+1), \qquad (6)$$

(5) and (6) are a contradiction, implying that it is not the case that $x \geqslant 0$. Hence $x < 0$, i.e.,

$$t-b-2 < 0.$$

But by (1), $t \geqslant b+1$.

Hence $t = b+1$

and by (3) $S_i(j)$.

Hence, $\underline{S_{ij}(t) \Rightarrow t=b+1 \cdot S_i(j)}$. (7)

Also,

$$S_i(j) \Rightarrow T_{i\ \underline{b-1}}(j+i) \Rightarrow T_{i\ \underline{b-2}}(j+i+1) \Rightarrow \ldots \Rightarrow$$
$$\Rightarrow T_{ij}(b). \qquad (A)$$

and

$$S \varepsilon II_0(b) \Rightarrow (\exists i)(S_i(0)) \Rightarrow V_{b-1}(1) \Rightarrow V_{b-2}(2) \Rightarrow \ldots \Rightarrow V_0(b). \quad (B)$$

If $S_i(j) \varepsilon S \varepsilon II_0(b)$, then (A), (B), and the fact that $\#S_{ij} = 2$ imply that

$$S_i(j) \Rightarrow S_{ij}(b+1). \qquad (8)$$

Hence the elements $S_i(j)$ of a stimulus $\varepsilon II_0(b)$ are displayed, at time $t = b+1$, as pulses $S_{ij}(b+1)$. (By (7) and (8).

Now let $A = (i,j)$ $i = 1,\ldots,n$ $j = 0,\ldots,b$.

Then each subset x of A, for which there is an (i,0) in x, corresponds (naturally and uniquely) to an element A of $II_a(b)$. For each such x, supply three cells, E_x, I_x, and F_x with the following connections: For each (i,j) $\in$ x a connection from S_{ij} to E_x. For each (i,j) $\notin$ x a connection from S_{ij} to I_x. Also run an inhibitory connection from I_x to F_x and an excitatory connection from E_x to F_x. Let E_x be a disjunction and I_x a con-junction, and F_x be a b.i.j. By an argument parallel to that of case 0, $F_x(b+3) = A_x$. One may then construct chains of proper lengths from the F_x's to the output cells S_K, as in case 0; and as in case 0, show that the net realizes the given function $F(A)$. F must have delay $\geq b+4-\epsilon$ since no S_K can fire before this time. If free use of inhibitory connections is permitted (or if one allows an arbitrary number of inhibitor connections to terminate on what would otherwise be the con-junction F_x) then the delay can be cut to b + 3, as in case 0.

2/4.2.3 However, if we permit the use of inhibitor connections as above, the functions of delay $\geq b+2-\epsilon$ (on $II_a(b)$), can also be realized, by the

following net. In this construction one level is eliminated; that of the "display" cells S_{ij}. (Because this level is not present in the 0 case, there is no opportunity for a further cut in delay there.) Construct the net as follows:

CELLS: S_i: $i = 1,\ldots,n$

T_{ij}: $i = 1,\ldots,n \quad j = 0,\ldots,b$

NOTE: T_{ib} is S_i.

V_k: $k = b+1,\ldots,2b$

F_x: x ranges over all stimuli for which $S_x \subset \Pi_0(b)$.

CONNECTIONS:

(EXCIT.) $T_{ij} \rightarrow F_x$ for all i, j, x such that $(i,j) \in x$.

(INHIB.) $T_{ij} \multimap F_x$ " " " " " " " " " " $\notin$ "

(EXCIT.) $T_{ij} \rightarrow T_{i,j-1}$ $(j=1,\ldots,b)$

(EXCIT.) $V_k \rightarrow V_{k+1}$ $(k=b+1,\ldots,2b-1)$

$T_{i0} \rightarrow V_{b+1}$ (all i)

(INHIB.) $V_k \multimap F_x$ (all x, all $k=b+1,\ldots,2b-1$)

THRESHOLDS: all thresholds are 1 except for

$$\#F_x = \sum_{(i,j) \in x} 1.$$

Operation of the net is described by

$$T_{ij}(t) \equiv T_{ij+1}(t-1) \equiv \cdots \equiv T_{ib}(t+j-b) = \beta_i(t+j-b) \qquad (1)$$

$$\text{or } \beta_i(j) \equiv T_{ij}(b).$$

$$F_x(t) \equiv \quad (i,j) \in x \Rightarrow T_{ij}(t-1)$$

$$\cdot (i,j) \notin x \Rightarrow \sim T_{ij}(t-1)$$

$$\cdot \sim V_k(t-1) \qquad k = b+1, \ldots, 2b. \qquad (2)$$

(1) follows from the connection scheme.

(2) follows from the connection scheme and the definition of $\#F_x$.

Now $x \in \Pi(b)$ means that for some i, $(i,0)$ is in x. Hence by (2), $F_x(t)$ implies that for some i, $T_{i0}(t-1)$, which in turn, by (1), implies that $\beta_i(t-b-1)$. Hence $F_x(t) \Rightarrow b+1 \leqslant t \leqslant 2b+1$. (A)

Also, $[\overline{x} \in \Pi_0(b)] \Rightarrow (\exists i)(\beta_i(0))$. (Def. of $\Pi_0(b)$)

$$\Rightarrow T_{i0}(b) \qquad \text{(by (1))}$$

$$\Rightarrow V_{b+1}(b+1)$$

$$\Rightarrow V_{b+2}(b+2) \Rightarrow \cdots \Rightarrow V_{2b}(2b).$$

Hence,

$$b+1 < t \leqslant 2b+1 \Rightarrow \sim F_x(t), \qquad (B)$$

$$\underline{\text{if}}\ x \in \Pi_0(b).$$

(A) and (B) together show that if $x \in \Pi_0(b)$,

$$\underline{F_x(t) \Rightarrow t = b+1.} \qquad (3)$$

(1), (2), and (3) combine to form

$$F_x(t) \equiv (t = b+1) \cdot (\beta_x).$$

Using this fact, and constructing the chains from the F_x to the F_k, one can show, exactly as in the two previous nets, that the complete net realizes the given function P. And, while the F_K cells were at the $t = b+3$ and $t = b+2$ time levels in the previous nets, they are on the $b+1$ time level for the present net, hence we can realize any function defined on $\Pi_0(b)$ whose delay is $b+2$ or greater. This completes the proof of theorem 2/4.2.

Note that at time $t = 2b+1$, the interior of the net is clear, and its functional properties are restored.

2/5 Non-monotone elements.

Definition: A "finite network element" J is an object with a set e_i of input terminals, a set f_j of output terminals, and a response function J(X) with the properties:*

1. $J(\Gamma) = \Gamma$.
2. J(X) operates within the fixed synaptic delay scheme.
3. J has a "finite recovery time" L. This means that for any stimulus X, if the last firing of any e_i occurs at time t_X, then the activity of the f_j ceases before $t_X + L$, and J has its original response properties (translated) at times $t_X + L$ or later.

Definition: A network element is non-monotonic if for some pair X, X' of stimuli, $X \subset X'$ and $J(X) \not\subset J(X')$.

2/5.1 Lemma (2/5.1): Given a non-monotonic network element, a net $\mathcal{J}$ can be constructed, using only J, con-junctions, and dis-junctions, which has two input cells $\mathcal{I}_1$ and $\mathcal{I}_2$ and one output cell $\mathcal{R}_0$, and which (for some $d_0(J)$ and all t) has the property:

$$[\mathcal{I}_1(s) \vee \mathcal{I}_2(s) \Rightarrow (s \leqslant t-d_0) \vee (s = t) \vee (s \geqslant t+d_0)]$$
$$\Longrightarrow [\mathcal{R}_0(t+d_0) \equiv \mathcal{I}_1(t) \cdot \sim \mathcal{I}_2(t)].$$

PROOF: Let X and Y' be such that $X \subset Y'$ and $J(X) \not\subset J(Y')$.

* This definition is not intended to be final, but will suffice here.

Connect a cell S_1 to each input terminal e_1 of J. Let $\#S_i = 1$. For each $e_i(t) = X$ construct a chain from S_1 to S_i having t intermediate cells. Then

(1) $$s_1(t) = X(t-1). \qquad (1)$$

Do the same for Y. Then

(2) $$s_2(t) = Y'(t-1). \qquad (2)$$

Note also that $s_1(t) \cdot s_2(t) = Y'(t-1)$. (3)

Now since $J(X) \not\subset J(Y')$ there must be a (X,n) for which $s_1(n) \to f_k(n)$ and $s_2(n) \to \sim f_k(n)$. Let L be the recovery time of J. Let d be the length of the longest chain from an S_1 to an e_j. (If a chain has r intermediate cells, its length is r+1). Then if either $s_1(t)$ or $s_2(t)$, the net will be returned to its resting state before time t+d+L. Run a chain from f_k to S_j with $t+L-d-1$ intermediate cells, and also run a chain from S_1 to S_j with t+L-1 intermediate cells, and let $\#S_j = 2$. Then let $d_0 = 1$.

$$s_j(t) = f_k(t+n-1_j) \cdot s_1(t-d_j) \qquad (4)$$

The condition

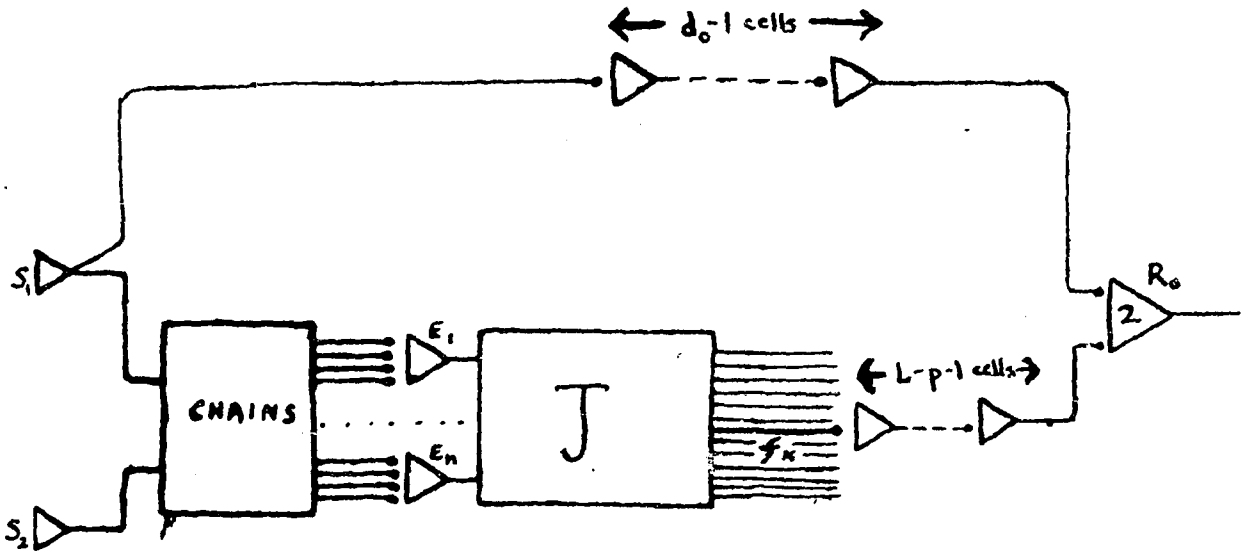
$$s_1(s) \to (s \leqslant t-1_j)_v(s = t)_v(s \geqslant t+d_j)$$

implies that

$$f_k(n+t) \equiv s_1(t) \cdot \sim s_2(t) \qquad (5)$$

The reverse implication follows from the construction, the forward direction follows from the condition

on t, since $S_1(t)$ and $S_2(t)$ and $S_1(t) \cdot S_2(t)$ are the only stimuli that could have caused f_k to fire. (Otherwise the net would be in the resting state of time t, by definition of d_0). (4) and (5) imply the conclusion of the lemma. Call the whole net Q^J.



2/5.2 DEFINITION: A neural net operates in the <u>expanded time scale</u> T_s if all input and output pulses occur at times st.

DEFINITION: A stimulus S is in $II_a^s(b)$ if, and only if

$$S_1(t) \in S \Rightarrow (\exists k)(t = ks) \cdot (a < k \leq b) \text{ and also}$$

$$(i)(S_1(t) \in S \cdot t = sa) \cdot (\exists i)(S_i(a+i) \in S)$$

This class is $II_a(b)$, "expanded".

<u>Theorem 2/5.2. Using only con-junctions, dis-junctions, and copies of any given non-monotone network element J, a net can be constructed to realize any output function P defined on $II_a^s(b)$ with</u>

(b-4)+3

delay (b+4)s (or (b+3)s in the case $U_{(a)}^{s}(a)$) and operating in the expanded time scale T_s, for any $s \geq d_{(J)}$.

PROOF: Replace each cell J_i in the proof of part 1 and part 2, theorem 2/4.2, by a chain of cells starting with J_i and having s-1 intermediate cells, except that each b.i.j. should be replaced by a copy of the net c^J (of lemma 2/5.1) followed by a chain of $s-d_{(J)}-1$ intermediate-cells. This process converts these nets for operation in the time scale T_s. Lemma 2/5.1 shows that c^J acts like a b.i.j. in $T_{(J)}$, and with the extra chain, like a b.i.i. in T_s. Part 2/4.2.3 of theorem 2/4.2 does not apply since it uses other network elements.

2/5.3 Definition: An element J is "II-potent" (for an admissible Class II of stimuli) if copies of J, con-junctions, and dis-junctions are sufficient to construct a net to realize any given function on II which has delay equal to or greater than some time T(II,J).

Note: In the following sections, no effort will be made to determine the minimum delay times realizable for special nets. For any particular J this might be worth while.

Definition: A II-clock is a net C(II) with the following property: C(II) has input cells S_i and an output cell R^C. For any stimulus S in II, R^C fires exactly once, and at a time T^C which is independent of the stimulus S, provided that $S \in II$.

2/5.4 Theorem: Any non-monotone element J is II-potent if and only if a II-clock can be constructed using only con-junctions, dis-junctions, and copies of J.

1. The forward direction of the theorem is trivial: If J is II-potent then a net can be con-constructed using J, con, and dis-junctions to realize the function $P(S) = R^C(T(II,J))$. This net is a II-clock with $T^C = T(II,J)$.

2. The reverse direction requires a construction which is a generalization of those in Theorems 2/3.4 and 2/4.2.

The first step in the construction is to describe a "display" net, D, with which the stimuli may be converted into simultaneous volleys. (The net of 2/4.2.2 is a special case of this net.)

Let T^{II} be the time of the latest pulse in any S of II. Let $z = \max (T^C, T^{II})$. Construct the net D as follows:

CELLS: S_i $(i = 1, \ldots, n)$ (input cells).

I_{ij} $(i = 1, \ldots, n)$

$(j = 0, \ldots, z)$

Note: Cells S_i and cells I_{iz} are identical.

Cells S_i are also the input cells of C(II).

S_{ij} $(i = 1, \ldots, n)$

$(j = 0, \ldots, z)$

B_k $(k = 0, \ldots, z - T^C)$

Note: Cell B_0 is identical with R^C. There may be no other B_k's.

CONNECTIONS:

$I_{ij} \longrightarrow I_{ij-1}$ (all i; $j = 1, \ldots, n$)

$I_{ij} \longrightarrow S_{ij}$ (all i, all j.)

$B_k \longrightarrow B_{k+1}$ $(k = 0, \ldots, z-T^C-1)$.

$B_{z-T^C} \longrightarrow S_{ij}$ (all i, all j.)

Thresholds: All cells have threshold unity except the S_{ij} which all have threshold 2.

It can be seen, by an argument similar to that of 2/3.4, that for this net, if S ϵ II, then,

$$S_{ij}(t) \equiv .[t = z+1] \cdot [S_i(j)]$$

To complete the proof, a net is adjoined of the type constructed in 2/4.2.1 and 2/4.2.2. In 2/4.2.2, for each subset x of the S_{ij}'s, there was adjoined a subnet of three cells E_x, I_x, and F_x, (where F_x was a b.i.j.) in such a way that there was a connection from S_{ij} to E_x if and only if S_{ij} e x, and one from S_{ij} to I_j if and only if S_{ij} x, (with E_x a con-junction, and I_x a dis-junction). For the present proof, instead of using a b.i.j.,F_x, supply, for each x, a copy of the net Q^J as constructed in 2/5.1. Let R_x be the output cell of the Q^J associated with x. Let d(J) be as defined in 2/5.1. Then it is easy to show that if $x(S) = \{S_{ij}/S_i(j) \text{ e } S\}$, then for any S e II,

$$R_x(t) \equiv [S].[t = z + 2 + d(J)]$$

Thus for any S in II, exactly one R_x will fire, and at time z + d + 2. This R_x will be the cell $R_{x(S)}$ as defined above. The last step is to construct chains of the proper lengths from the Rx's to the output cells, as was done in 2/4.2.2.

2/5.5 Theorem 2/5.5

Any non-monotone element J is II(a,b)-potent for all II(a,b).

Proof: A II(a,b)-clock is constructed as follows:

Cells: S_i, $i = 1, \ldots, n$.

C_j $j = a, a+1, a+2, \ldots, b$.

R^C. (an output cell)

$\#C_j = 1$ $\#R^C = 2$.

Connections: $S_i \longrightarrow C_a$ all i.

$C_j \longrightarrow C_{j+1}$ all $a \leq j < b$

$C_a \longrightarrow R^C$

$C_b \longrightarrow R^C$

Then $R^C(t) \equiv C_a(t-1).C_b(t-1)$. (1)

$C_b(t-1) \equiv C_a(t-1-b)$. (2)

(1) and (2) imply (3)

$R^C(t) \equiv C_a(t-1-b).\ C_a(t-1)$

Also $C_a(t) \equiv (\exists i)(S_i[t-1])$. (4)

(3) and (4) imply that

$R^C(t) \equiv (\exists i)(\exists j)(S_i\ t-2-b).S_j(t-2)$ (5)

Because the stimulus is in II(a,b), the event on the right side of (5) occurs if and only if $t-2-b = a$ and $t-2 = b$.

Hence the cell R^C fires exactly once, and at the time $t = b+2$, and the net is a II(a,b)-clock with $T^C = b+2$.

Note: The above net was constructed without using any non-monotone elements. A similar construction was employed in the proof of theorem 2/3.4, but in that construction, there was no isolated "gating" cell R^C, and the S_{ij}'s with threshold #3 absorbed this function. This technique could also be used in the proof of theorem 2/5.4, with the saving of one unit of delay time.

2/5.6 The definition of II-clock can be weakened somewhat and still be adequate for the truth of theorem 2/5.4.

Definition: a weak II-clock W(II) is a net which has the following properties:

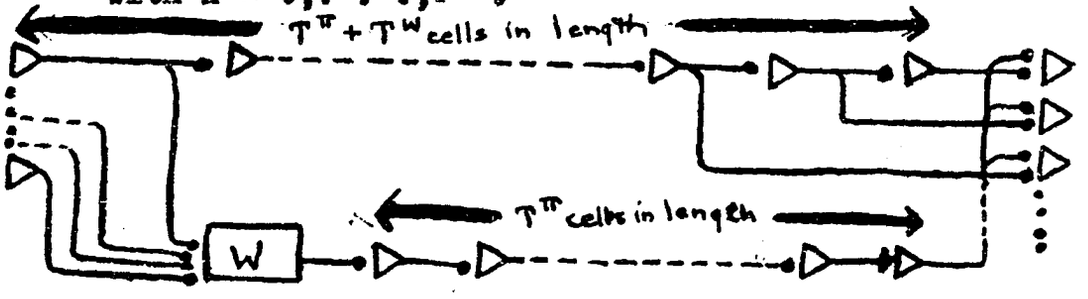
W(II) has input cells S_i and an output cell R^W. For any input S in II, R^W fires exactly once, but the time of this pulse $R^W[(S)]$ is not necessarily independent of S.

Theorem: 2/5.6 If with a non-monotone element J, and also con- and dis-junctions, a weak II-clock W(II) can be constructed, then so can a strong II-clock, and J is II-potent.

Proof: One can either show that J is II-potent directly, or that a strong II-clock, can be constructed. The latter is done below:

Let T^W be the time of the latest response of

(II) to any stimulus S of II. Let T^{II} be the time of the latest pulse of any stimulus of II. Construct a net exactly as in the first part of the proof of theorem 2/5.4, except with $j = 0,\ldots,T^W + T^{II}$, and with $k = 0,\ldots,T^{II}$.



Then it is easy to see that for each stimulus S of II, the S_{ij}'s can fire only at some time T(S) for which

$$T^{II} < T(S) \leq T^{II} + T^W + 1.$$

Let the pattern which appears (as a simultaneous volley) at the S_{ij} at time T(S) be called S*. Then,

$$S,S' \in II \Rightarrow (S' \neq S \equiv S^* \neq S'^*). \qquad (1).$$

The reverse direction of (1) is trivial. To prove the forward implication, note that for any S in II, and at any time t for which

$$T^{II} \leq t \leq T^{II} + T^W, \qquad (2)$$

the display net I_{ij} contains a pattern which is a complete spatial display of the stimulus S. Furthermore, any such pattern determines the stimulus uniquely up to time translation, for stimuli in II. Furthermore, for stimuli in II, the patterns S^* on

the S_{ij} at time T(S) will be in one-one correspondence with the patterns on the I_{ij} at time T(S)-1 (which is in the interval defined in [2]). But since S and S' are both in II, if they are distinct they cannot be translates, hence $S^* \neq S'^*$.

To complete the proof, the net is completed in a manner similar to that of the proof of theorem 2/5.4. For each S ε II, adjoin cells E_S, I_S and nets $Q^J{}_S$ as in the proof of theorem 2/5.4. Then,

$$Q^J_S(t) \equiv S(0).(t = T(S) + 1 + d(J))$$

(where d(J) is the delay of the net Q^J). Then if

$T = [T^{II} + T^W + 1 + 1 + d(J) + 1]$, it follows that for a given stimulus S, the only Q^J that can fire is $Q^J{}_S$, and that $Q^J{}_S$ will fire at exactly one time t(S) and that for all S,

$$T(S) + 1 + d(J) = t(S) < T.$$

Let R be an additional cell (with threshold 1). For each S, run a chain of length T - t(S) from the output cell of $Q^J{}_S$ to R. Then for all S ε II, R will fire at time T and only then. Thus the entire net is a strong II-clock with input cells S_i and output cell R.

Hence II-potency is equivalent to the constructability of a weak II-clock.

2/5.7 The DURATION of a stimulus is 1 plus the difference between the times of the last and first pulses.

LEMMA 2/5.7. Let L be the duration of the longest element of II . Then if an element J is $II_0(L)$-potent, it is II-potent.

Proof: Each element of II is a translate of an element of $II_0(L)$. Hence any net which is a strong $II_0(L)$-clock will be a weak II-clock. Then by theorem 2/5.6 a strong II-clock can be constructed, and by 2/5.4 J is II-potent.

2/5.7.1 It follows from 2/5.7 that if an element J is $II_0(b)$-potent for each b, it is II-potent for every admissible class II. (Note: It is obvious that if an element is $II_0(b)$-potent, it is $II_0(c)$-potent for all $c \leq b$. For then $II_0(c)$ is a subset of $II_0(b)$.)

2/6 PARTICULAR NON-MONOTONE ELEMENTS.

2/6.1 Refractory periods.

Let us consider an element K which has the following properties:

1. $K(t) \Rightarrow \sim K(t+1)$.

2. Except for (i), K has the properties of an ordinary cell. (In the theorem below, K will be used only as a junction with one endbulb and threshold $\#K = 1$.)

Property (1) above is read: "K has a refractory period of unit duration". In the general case, K has a "refractory period of duration d", and this is expressed formally by

$$K(t) \Rightarrow \sim K(t+1) . \sim K(t+2) . \ldots . \sim K(t+d) .$$

Theorem 2/6.1.1

The element K is II-potent for all admissible classes II.

Proof: Consider the class $II_0(b)$. Construct the following net.

Cells: $S_i \ (i = 1, 2, \ldots N)$

$N_j \ (j = 0, \ldots, b)$

$K_1, M_2, M_3, M_4, M_5, M_6, M_7, M_8, K_2.$

K_1 and K_2 are K-elements. M_7 has threshold 2. All others are dis-junctions with threshold 1.

Connections: $N_o \longrightarrow N_j \quad (j > 0)$

$N_1 \longrightarrow N_{j+1} \quad (j < b)$

$N_b \longrightarrow K_1$

$K_1 \longrightarrow M_2 \longrightarrow M_3 \longrightarrow M_4 \longrightarrow$

$M_5 \longrightarrow M_6 \longrightarrow M_7$

$K_1 \longrightarrow M_8 \longrightarrow K_2 \longrightarrow M_7$

$M_4 \longrightarrow M_8$

$S_j \longrightarrow N_0 \quad$ all j

Now consider any S in $\Pi_0(b)$. We know that for some i, $S_i(0)$. Let t_0 be the time of the last pulse (or pulses) of S. (Then $t_0 \leq b$).

Now $S_i(t) \Rightarrow N_0(t+1)$ (1)

And, $N_0(t+1) \Rightarrow N_j(t+2)$ $(j > 0)$ (2)

Also, $N_j(t+2) \Rightarrow N_b(t+2+b-j)$ (3)

(1), (2), and (3) imply that

$$S_i(t) \Rightarrow N_b(t+2).N_b(t+3). \ldots .N_b(t+b+1). \quad (4)$$

Then

$$S_i(0).S_j(t_0) \Rightarrow [N_b(2). \ldots .N_b(b+1)].[N_b(t_0+2). \ldots .N_b(t_0+b+1)].$$

These two sequences must cover the interval

$$N_b(2). \ldots .N_b(t_0-b-1), \text{ since } t_0 \leq b. \quad (5)$$

Furthermore, the pulses at N_b due to any other $S_k(t)$ must lie in this time interval, since $S_i(0)$ is the first pulse and $S_j(t_0)$ is the last. (By theorem 2/2.7, (5) gives the complete response at N_b). Thus, $S(0) \Rightarrow N_b(2).N_b(3). \ldots .N_b(t_0+b+1)$ and only these.

Now K_1 will fire first at time $t = 3$, and at odd times thereafter. Let T be the last time that K_1 fires. ($T = t_0+b+2$ if this is odd, t_0+b+1 if not.) M_4 will fire three units after K_1 fires, so M_4 fires first at $t = 6$, and at <u>even</u> times thereafter up to and not after T+3. (6)

M_6 fires two units later than M_4, so M_6 fires first at t = 8, and at all even times up to but not after T+5. (7)

Now, $M_8(t) \equiv M_4(t-1) \vee (k_1(t-1)$. From (6) it is seen that M_8 fires at

a.) all even times between 4 and T+1. (8a)

b.) all odd times between 7 and T+4. (8b)

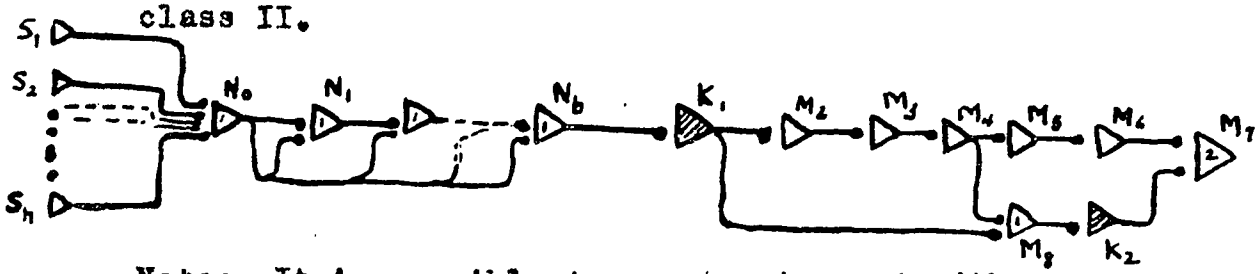
Because of (8a), K_2 fires at all odd times between 5 and T+2. (and each firing at an odd time in this interval prevents a pulse at the succeeding even time). But then, because of (8b), <u>K_2 fires also at time T+5. T+5 is an even time, and it is the only even time at which K_2 fires.</u> (9)

Finally, because $\#M_7 = 2$,

$M_7(t) \equiv M_6(t-1) \cdot K_2(t-1)$. (10)

But M_6 fires only at even times (including T+5), by (7). And K_2 fires only at odd times, except for the even time T+5, by (9). Hence condition (10) is fulfilled when and only when t-1 = T+5, or t = T+6.

It follows that the net constructed above is a weak $II_0(b)$ clock, for when presented with any element S of $II_0(b)$, it responds with a single output pulse. Hence the element K is $II_0(b)$ potent for each b. By 2/5.7.1 K is II-potent for every admissible



Note: It is possible to construct a net with similar properties in which all cells have a refractory period of unit duration.

Theorem 2/6.1.d:

Theorem 2/6.1.1 holds for elements K with arbitrary refractory periods d.

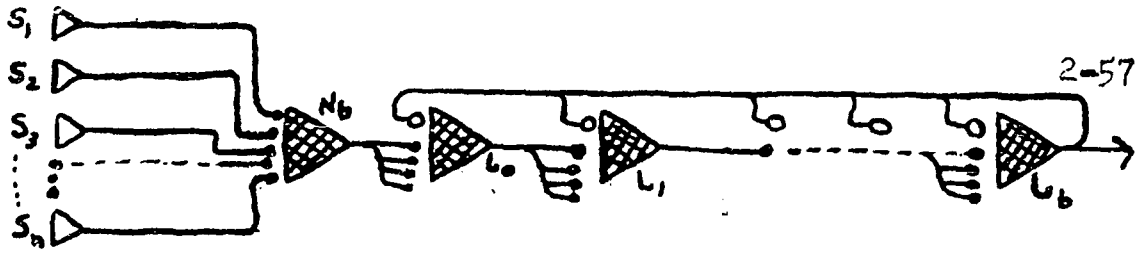
Proof: The proof is like that of Theorem 2/6.1.1, except that between cells K_1 and M_2 one must insert a chain of d-1 intermediate cells. The remainder of the proof is essentially the same, if for "odd times" we use "times $\equiv$ 1 mod(d)" and for "even times" use "times $\equiv$ 2 mod(d)".

Thus all elements with refractory periods are II-potent over all admissible classes of stimuli II.

2/6.2 Inhibitors.

Theorem 2/6.2. Any element with one or more inhibitory connections is II-potent over any admissible class II.

Proof: Let L be the element. Consider the class $II_0(b)$. Construct the net:



Note: If L has threshold $\neq L$ greater than one, run $\neq L$ fibres from each L_i to L_{i+1} $(i < b)$. It is easy to see that L_b first fires at time $t = b+2$ (since some S_i must fire at time 0). However, when L fires at time b+2, it prevents any L_i from firing at $t = b+3$. Thus at time b+3 there are no pulses in the net, since none can enter from the S_i after $t = b$. Since there is now no source of pulses, the net will remain quiet. Hence the net is a (strong) $\Pi_0(b)$ clock. The theorem follows now from 2/5.7.1.

NOTE: Theorem 2/6.2 is related to 2/4.2; it asserts that with inhibitors one can construct any function on any admissible class which has sufficient delay. Theorem 2/4.2 asserts the constructability of arbitrary functions on a smaller set of stimulus classes, but it specifies a minimum delay.

2/6.3 The elements K and L of 2/6.1 and 2/6.2 are the only non-monotone elements of immediate interest. It was shown earlier that <u>any</u> non-monotone element J is II-potent for all II in an appropriately expanded time scale, but I have been unable to establish the corresponding result in the unit time scale.

Neither have I found a counterexample, but I incline toward the belief that there is one.

2/7 Physiological note.

We cannot remain restricted to monotone nets and hope to obtain machines of much interest. Theorem 2/2.8 shows that in both probabilistic and non-probabilistic cases, a net which has a non-monotone output function must contain one or more non-monotone elements, in addition to the (given) elements of the threshold type. In sections 2/6.1 and 2/6.2 it was shown that either "inhibitory" or "refractory" elements are adequate for the construction of functions which are arbitrary, except for the restrictions to sufficient delay and to domains of "admissible classes of stimuli. (It should be noted that the restriction to "admissible classes" i.e., those classes of stimuli which are finite and contain no pair of time-translates, reflect the finiteness of the nets, and the manner in which they are used, rather than any coarse limitation of their functional capabilities. If part of the net were to be set into operation at the origin t = 0 of time, before the presentation of external stimuli, thus setting up a sort of absolute clock with reference to a definite point in time, then some translated stimuli could be

distinguished. Even so, there would still have to be some restriction on translates, since the clock, being finite, would ultimately become periodic, and stimuli translated by this period would be functionally indistinguishable. The same argument would hold for the brain, in so far as it were regarded as a finite network; however, the period of that clock need not be shorter than a lifetime, and what might be called the problem of "dating" events need not arise.)

The origin of the evident non-monotonicity of the central nervous system is far from clear at present, but it is appropriate, at this point, to examine a few possibilities, and incidentally to discuss the evidence for the existence of the monotonic elements that have played a basic role in sections 2/2 to 2/5.

2/7.1 Con-junctions and dis-junctions.

The existence of these basic monotone elements as relatively discrete entities within the brain seems to be generally accepted by physiologists. The evidence seems to be of two kinds. In experimental studies on spinal reflexes the evidence for "spatial summation" is generally considered to reflect the presence of junctions which require a minimum of more

then one impulse for transmission. Secondly, each of the major theories of interneural transmission predict such effects, and the anatomy of junctions appears to be such as to make it plausible that such effects can take place. The electrical and chemical transmission theories each require a minimum density of stimulation at the surface of the efferent cell, in the one case a minimum current density, in the other a minimum concentration (or quantity, in Rashevsky's theory) of the excitatory chemical. If a single endbulb can supply the minimum, we would have a dis-junction; if the firing of several endbulbs is required, a junction with threshold higher than unity results. (The possibility of variation of threshold is discussed later.) Anatomical photographs support this principle, in that they reveal terminations from several cells in any small surface region of a given efferent cell. (Generally speaking, most of this type of evidence is derived from studies on large motor cells, where a good deal of structure may be discerned.)

2/7.2 The nature of non-monotonic junctions is not nearly so clear. We shall discuss a few possibilities.

2/7.2.1. Inhibitory connections.

The axioms of 2/1.3 admit the possibility of a connection C_{ij} for which $\not\!\!C_{ij} = \infty$. Thus $C_i(t-1) \Rightarrow \sim C_j(t)$. This may be called <u>absolute inhibition</u> of the cell C_j.

If this ideal connection were a realistic model of a biological entity or process, it would mean that the firing of cell C_i had an effect on cell C_j which would prevent it from firing in a (definite) later interval, no matter what the stimulus to cell C_j. However, since in any fixed situation, there will be only a finite number of terminations on any cell, and the activity of each termination will also be bounded, the stimulus to cell C_j will always be bounded, in some biologically appropriate sense. The notion of absolute inhibition, is then not essential for any biological model of inhibition, for there is no need for a process wherein the cell C_j is rendered inherently unable to fire at time t; it is sufficient that the effect of the firing of C_i render C_j unresponsive to any stimulus that it can possibly encounter. For practical purposes, it would be sufficient to allow $\not\!\!C_{ij}$ to take on only finite negative values. This is essentially the approach in the Rashevsky

theory, where inhibition is "relative". It is nevertheless perfectly possible that a real absolute inhibition occurs in the central nervous system.

2/7.2.2 Chemical theories of inhibition.

If it is to be postulated that certain connections under proper conditions release an "inhibitory substance" J, then J must act in one of two ways:

A. J in some way directly neutralizes the excitatory stimuli. E.g., if excitation were mediated by an excitatory substance E, J could form an inert chemical combination with E, or otherwise inactivate it.

B. J in some way protects the cell surface from the effects of E. This effect could be absolute or relative.

Rashevsky does not explicitly choose any one mechanism, but he does postulate that E and J behave in such a way as to resemble a quantitative neutralization.

2/7.2.3 Electrical theories.

If it is postulated that excitation takes place due to the passage of a sufficient current density in the proper direction through (a sufficiently large area of) the efferent cell membrane,

then there are a number of possible inhibitory mechanisms.

A. Inhibition could be produced by an opposing electric field; such a field could occur by the firing of fibres so placed (geometrically) that their firing potentials oppose the excitation potentials. This effect could be local, confined to the neighborhood of the junction, or could be due to larger electrical fields covering relatively large neural subnets.

B. An electrical transmission theory is not incompatible with a chemical inhibition theory. The inhibitory substance could act to render the membrane unable to fire. (It is conceivable that the inhibitory substance could affect the dielectric or resistivity properties of the junction to an extent sufficient to prevent threshold stimulation of the efferent cell.)

2/7.2.4 Anatomical theories:

It is conceivable that the axon hillock could be so affected by nearly excitation as to prevent later excitation from reaching the axon. (There is no evidence for this.) In fact, any process by which retrograde conduction is established from the axon, or axon hillock, without simultaneous

initiation of forward conduction, would put the cell into a refractory phse, and thus act as an inhibitor.

2/7.3 Location of inhibition.

The location of the non-monotone processes of the nervous system is also a matter of speculation. All neural elements have a refractory period, hence every net is potentially capable of non-monotone activity, including behavior which resembles inhibitory. Nevertheless, there may be more specialized forms of inhibition than this distributed local type. We shall later consider more global forms; suppression of activity in the "final common path" or motor tracts, and processes which extinguish, or greatly modity the activity of entire nets. Some of the machines to be described will contain subnets which exercise a comparatively large control over inhibitory processes in other sublets.

The basic physiological fact that all neural elements have appreciable refractory periods, coupled with theorem 2/6.1.d, will justify our subsequent lack of concern with the problem of non-monotonicity. There is far too little real physiological information available to justify the assumption that any other <u>particular</u> non-monotonic element is present

in the nervous system.

2/8.0 Anatomical structure of junctions.

2/8.1 Little is known about the anatomy of interneural junctions within the central nervous system. The best information is to be found in microphotographs of the surface of the giant motor cells of the ventral columns of the spinal chord. Fibres terminate, on or near the dendritic or perikaryon surface with distinguishable "endbulbs", and it is believed that these represent the anatomical site of the interneural junctions.

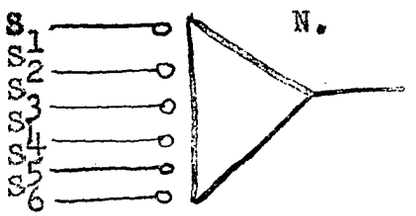
Whether the same structures exist throughout the CNS is not clear, and there is no reason to believe that the endbulbs are the only possible interneural junction.

2/8.2 There is another possible site for transmission of pulses, and in order to describe it, it is necessary to suggest a modification of the usual view of the role of neurons in the functioning of the nervous system. It is unnatural to regard the neuron as the basic unit of activity, inasmuch as a single neuron usually has more than one junction associated with its dendritic and perikaryon surface. If the nervous system is to be described

in terms proper to a neural-analogue net, and the neurons are taken to be the "cells" of the axiomatic theory, then the condition for firing of a neuron will be a disjunctive expression where each term is the firing condition for one of the junctions on the neuron. It is more natural to regard each junction as a basic unit of activity, and to regard the neuron as an "identification" of the output fibres of each of the junctions on the neuron. This identification can be taken to be a multiple junction with threshold one (with probability one) and having little or no synaptic delay. Let the symbol  represent such an element.

Thus, suppose a neuron N has three junctions on its surface so that its firing condition is

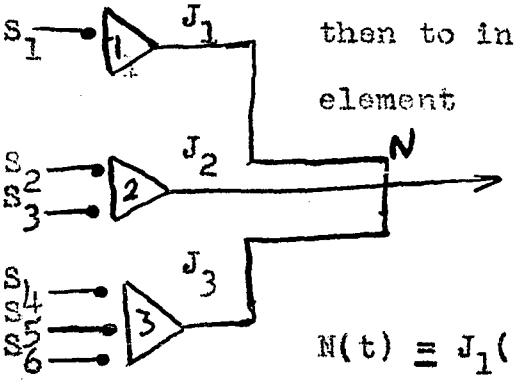
$$N(t) \equiv [S_1(t-1)] \vee [S_2(t-1).S_3(t-1)] \vee [S_4(t-1).S_5(t-1).S_6(t-1)]$$



(following McCulloch-Pitts.)

It is more natural to regard the junctions as functional units:

$$J_1(t) \equiv S_1(t-1)$$
$$J_2(t) \equiv S_2(t-1).S_3(t-1)$$
$$J_3(t) \equiv S_4(t-1).S_5(t-1).S_6(t-1), \text{ and}$$



then to introduce the neuron as an element

$$N(t) \equiv J_1(t) \vee J_2(t) \vee J_3(t).$$

Note that the element N in the above expression has no synaptic delay. Such an element cannot be represented in the Pitts-McCulloch axiom system. The reason for this is that the firing condition for N(t) has a computation time of just 1 time unit. A Pitts-McCulloch net having one output fibre and time depth 1 can only be a single cell with a definite Threshold, T. Each of $S_1, \ldots S_6$ can have any number of endbulbs on N, say S_1 has b_2, etc. Then

$$[b_1 \geq T].[B_2 + b_3 \geq T].[b_4 + b_5 + b_6 \geq T.].$$

In this case, one of b_2 or b_3 must be $\geq T/2$, and one of $(b_4 + b_5), (b_5 + b_6), (b_4 + b_6)$ must be $\geq 2T/3$. Let, e.g., $b_2 \geq T/2$, and $b_4 + b_5 \geq \frac{2}{3}T$ Then

$$[b_2 + b_4 + b_5] \geq [T/2 + 2T/3] > T$$

hence the combination $(b_2 + b_4 + b_5)$ exceeds the threshold, and

$S_2(t).S_4(t).S_5(t) \Rightarrow N(t+1)$. This contradicts the original firing condition.

Note: If elements of the form d N are permitted, then minimum delay may be cut by 1 unit in several theorems d 2/3 and 2/4.

2/8.3 Now if the biological neuron is regarded only as an association of junctions, with an element N, nets can be constructed without any biological neurons at all, at least in the axiomatic framework, simply by not using any elements of the N-type. In fact all previously described nets may be regarded in this way. This could conceivably have a direct physiological parallel, as follows.

2/8.3.1 Consider a branch point of an (actual) nerve fibre. The branch can be regarded as an entity from which originate three nerve fibres. The firing of any of the fibres is some function of earlier firing of the others. In the usual case (anatomically), one of the fibres will be larger than the others. For each fibre is part of a tree terminating on some neuron, i.e., of the axonal or dendritic tree, and that one of the three fibres which is nearest (topologically) to the neuron will usually be larger. Now it seems plausible that when an impulse arrives at

the branch point from the larger fibre then both of the smaller fibres will be fired, with high probability. For the amount of electrochemical activity at the junction is then relatively large. On the other hand, when the input pulse comes along one of the smaller fibres, or even both, the amount of activity may not be enough to fire the larger. Certainly the probability of firing the larger fibre is greater when both small fibres fire, than when just one fires. Hence the branch point has to some extent the properties of a binary junction.

2/8.3.2 In the absence of a well-confirmed quantitative theory of nerve fibre conduction, there is no way to estimate the plausibility of 2/8.3.1. It is possible that all of these probabilities are so close to 1 that whole synaptic and dendritic trees fire as one, but to assume this to be the case is not less reckless than to assume that the probabilities are such that summations conditions are necessary for firing a branch, at least from the smaller fibres to the large ones.

There is one likely source of asymmetry between the small and large fibres; for all peripheral fibres, the refractory period is longer for smaller fibres, and the relation is quite

marked. Thus, if we have a small branch and two large ones, as in the figure, and F_1 is fired twice, with an interval between which is less than the refractory period of F_3, then F_2 will fire twice, but F_3 only once. This would seem very likely, physiologically. I point it out, not to use it in any essential way in the theory, but to indicate that the branches may ultimately have to be regarded as functional elements.

Another fact bearing on this branch theory is that all known cases fibres conduct equally well in either direction. It is generally believed that anatomical synapses (with endbulbs) can only conduct in one direction; from axonal tree to dendritic tree. If this is true, and if there are the only interneural pathways and if the neuron must fire to initiate axonal activity, then the neurons play an essential role in the net; for then a neuron must fire whenever activity spreads to involve the fibres of another neuron.

2/8.3.3 If, however, there is any way in which activity may be conducted from one tree to another, without firing of a neuron, then, at least in the

axiom system, the neuron loses its special importance and becomes merely a convenient logical disjunctive connective. Such conduction might exist in the nervous system simply as a property that if two fibres come sufficiently close, cross transmission is likely. Thus the close proximity of two or more fibres might have firing conditions entitling it to the classification of junction. If there are sufficiently many of such pathways, then the neural net can be regarded as a huge network of fibres in three dimensions, in which junctions occur in the form of branches, contiguities, and classical synapses, and which the neurons have a role of coupling the firing conditions of large numbers of fibres. The neurons probably also have a role in that they set up relatively large fields when they fire, affecting the transmission probabilities of all nearby junctions. It is conceivable that the neurons will not play a fundamental role in a neurological theory (at least in the cortex) and that their presence is largely a matter of biological necessity, since the neurons are the metabolic center for the fibres. One conclusion would be that, if the neurons are regarded as the primitive element, there are only about 10^{10} of

them, while if the branches and other junctions are regarded as also primitive, then there are at least 10^{12}, and perhaps 10^{13}, elements in the human brain.

CHAPTER 3 PROBABILISTIC ASPECTS OF NEUROLOGY

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3/0 In this chapter we attempt descriptions of two aspects of the central nervous system: The behavior of central interneural junctions, and the structure of the networks of the central nervous system. In each case the amount of real experimental and theoretical information is so small that any such discussion must be regarded as highly speculative.

One thing appears fairly certain, however. In each case the system is almost certainly so complicated that an exact description is not feasible. Interneural transmission probably depends both on highly fortuitous geometric contingencies, and on highly complex neural events occurring in the neighborhood of any given junction. And the structure of the nets, i.e., locations of cells and connections, probably depends on a tremendous number of biological variables.

In regard to the possibility of determining experimentally the quantitative laws involved here, there are serious difficulties both in practice and in principle. The practical difficulties are due, in the case of the behavior of junctions, to the fragility and minuteness of the junctional structures, and the consequent obstacles to isolation and extended study of single or small numbers of junctions. (Note: This does not apply to peripheral fibres and junctions; for this reason most information is derived from study of peripheral (and invertebrate) nerve.)

In the case of the structure of the neural nets, techniques for visualizing neural interconnections are as yet incomplete and undependable. There seems to be no technique at present which can provide reasonable complete information about all the connections in a volume of nervous tissue.

In the case of the interneural junction, it seems that even if the transmission laws were completely known, they may be so complex that a precise computation of neural activity in a net would still be impractical, e.g., if many of the variables indicated below play an important role.

For these reasons it would seem appropriate to attempt to replace those laws whose form is unknown but which are suspected to be dependent on many fortuitous variables, by simple statistical hypotheses and parameters, and attempt to find theorems which have a minimum dependence on the exact form of these laws.

This approach seems at least as sound as that of those models in which complex and unknown dependencies are replaced by simple deterministic functions, and the conclusions are expected to have some validity by "analogy". It may seem proper, e.g., to assume that an unknown monotone decreasing function asymptotic to zero is approximated by, say, $k_1 e^{-k_2 t}$, but it is <u>not</u> proper to assume that the derivatives of the unknown and the hypothetical functions are then also approximations.

/Rashevsky's extensive use of differential and integral equations seems to have little justification in this regard./

The statistical approach also avoids certain difficulties attendant on the use of the discrete models of the form of 2/1.1 - 2/1.4. In nets composed of these discrete elements, the behavior seems often to be dominated by special combinatorial artifacts, e.g., periodicities, which cannot be expected uncritically, in the biological systems.

3/1 The concept of excitatory threshold.

3/1.1 If a nerve fibre is isolated and maintained under constant laboratory conditions it will exhibit certain uniformities of behavior over relatively long intervals. A notable such uniformity is that of "threshold" to electrical excitability. Let a fibre be stimulated by passage of a measured electrical current for a constant time. It is observed that, if the excitations are spaced apart by a sufficient interval R then the fibre will fire, (a propagated impulse will arise) if, and only if, the stimulating current exceeds a certain value T (which depends only on the length of the applied stimulus, for a given fibre and experimental set-up).

R may be called the <u>full recovery time</u> of the fibre. If the fibre is stimulated at a time closer than R to the preceding excitation the minimum current required

for firing may be greater or less than T. We will discuss these fluctuations in 3.2ff.

The value T so obtained is called the (excitatory) "threshold" for the fibre (and for the experiment). Presumably the existence of such a threshold reflects some property of the excitable tissue which is common to all nervous tissue. (Muscle fibre exhibits the same property as well.)

However, even if we were to accept the proposition that essentially the same processes occur at the inter-neural junction, and that firing there too depends on the stimulation exceeding a definite threshold, there are some considerations which make it appear that the situation may be more complicated. Both the notions of "interneural junction" and of "stimulation" require clarification.

3/1.2 The question may arise as to whether there is justification for the belief that there exist distinguishable junctions; i.e., that the nervous system has an acceptable model in the axiom systems of the networks 2/1.5 or 2/1.6. If the connections c_{ij} are identified with anatomically discernable fibres originating on neuron c_i and terminating near or on neuron c_j, then the axiomatic model is realistic if, and only if, the firing (or for 2/1.6, the probability of firing) of c_j depends only on the activity of those c_i for which there are fibres c_{ij}.

But this is certainly not true in the brain. The firing of a given cell will depend on a number of factors

which are not so directly modeled in the abstract network. With any theory of interneural transmission involving electric intensities, chemical concentrations, and local membrane thresholds, the firing conditions at the junction will, almost certainly, be a complicated function involving the space-time distribution of activity over the surface of the cell. There is no evidence that any of the following features can be completely ignored:

a). Metabolic products of nearby cells. The concentrations and gradients of such substances will depend on the activity of all nearby cells, and may be expected to have an effect on the excitability of any given cell. Substances directly involved in nerve transmission may be expected to have important effects. The distribution of electrolytes will affect the geometry of electric fields.

b). The electric fields incidental to nearby activity will influence the activity at a given junction.

c). Activity of nearby cells will be reflected in thermal changes, which may have an appreciable effect. Thermal changes in the general circulation cannot be neglected.

d). Even local mechanical effects may be considerable. There are distortions due to circulatory pulsations, variations in mechanical strength of structures with any metabolic change, osmotic forces changing with redistribution of metabolites, and variations of the local

geometry due to amoeboid activity of supporting tissue cells. Finally, there is reason to believe that the endbulbs themselves may undergo changes consequent to recent activity.

e). Each cell and fibre involved will itself have a fluctuating internal state, causing variations in the magnitude of excitatory pulses.

f). Humoral substances in the local and general circulation will affect firing conditions locally and generally, (and not necessarily uniformly, since there is considerable differentiation of nerve cells). It is known that certain nervous centers are especially sensitive to the concentration of special substances.

g). Finally, the structures that are observed microscopically and believed to be "interneural junctions" are small, near the limits of optical resolution, (and these may not be the smallest). Little is known about their structure, and there is no reason to believe that they are the smallest entities involved in interneural transmission. For structures of this size operating at biological temperatures, thermal agitation effects may be appreciable. (Not knowing the magnitude of the processes essential for transmission, I cannot compute the importance of thermal effects.)

Note: In recent years it has been discovered that the central nervous system is provided with an

effective shield from the large concentration fluctuations of most substances in the general circulation. If a teleological argument is permitted, this may be taken to indicate that the nervous system is especially sensitive to chemical changes, hence such protection is necessary. But there is no evidence that the same protection is available for local fluctuations within the nervous tissue.

3/1.3 We have no way of evaluating the importance of these effects. Hence it would seem that if any theory is to be constructed, the firing conditions at a junction must be expressed in probabilistic terms. We will proceed on the assumption that the firing condition at a junction j_j is a probabilistic distribution which is a function of the activity of those cells for which there are fibres c_{ij}, as in axiom 3A-3n (2/1.6). This means that in spite of the possible non-net influences of 3/1.2, we commit ourselves to the belief that the visible network structure of nervous tissue is of essential importance, and that the other aspects of neural activity may safely be considered as contributing indeterminacy to the network structure. In the final chapters we shall return to this discussion.

3/2 Electrical excitability in nerve fibres.

3/2.1 contains a general description of certain fluctuations in the electrical threshold of some nerve fibres. 3/2.2 discusses the question of which fibres behave in this manner, and 3/2.3, the extent to which interneural junctions may be expected to behave in an analogous manner.

3/2.1 We consider the behavior of a nerve fibre under the same conditions as in 3/1.1. Assume that at $t = 0$ the fibre is in its "resting state", i.e., it has not been fired or stimulated for a sufficiently long time (e.g., "T" of 3/1.1) that its properties are constant, or nearly so. Let the fibre be fired at $t = 0$ by a sufficient stimulus, i.e., an electric current of amplitude exceeding I (3/1.1) of standard duration. It is an experimental fact that the excitability of the fibre at a later time d is substantially independent of the size of the stimulus at $t = 0$, provided that pulse is greater than I and not so large as to permanently injure the tissue. But the excitability does depend on the time d. In fact, suppose that the fibre is fired first at $t = 0$ by an adequate stimulus, and next excited at time d. Then the threshold $\mathrm{I}(d)$ at time d depends on d in the following manner: $\mathrm{I}(d)$ varies continuously and may be said to pass successively (in time) through five "phases".

/0/. $(0 < d < r)$ An "absolute refractory period" during which, i.e., no stimulus can initiate a pulse.

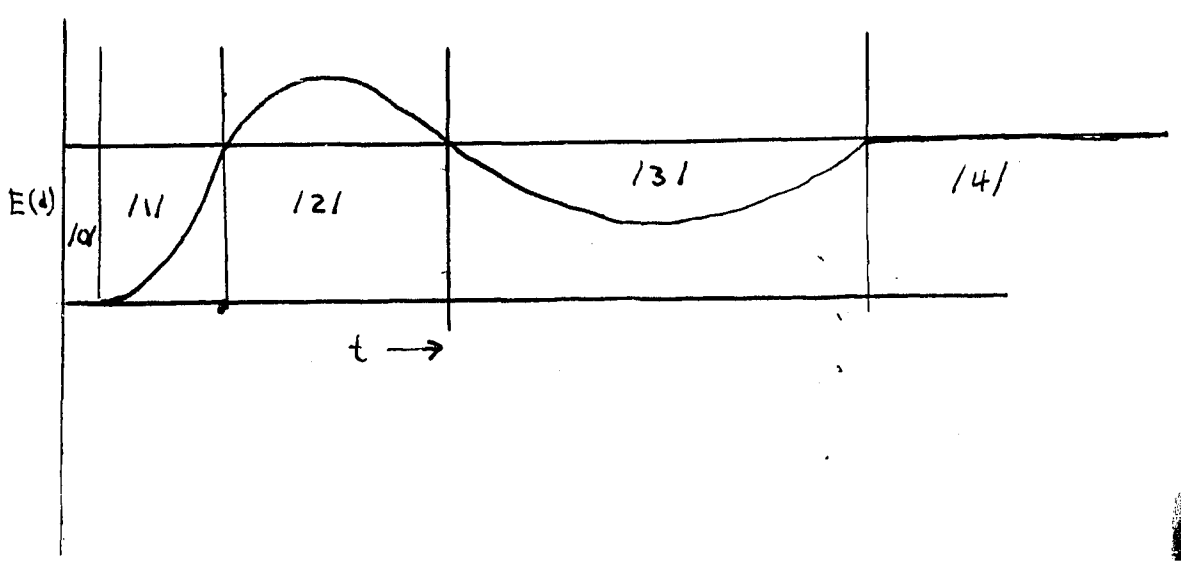
/1/. $(r < d < s)$ A "relatively refractory period", or, to use another equivalent expression, a period of "subnormal excitability", during which a stimulus greater than T is required to initiate a pulse.

/2/. $(s < d < s')$ A period of "supernormal excitability" during which the fibre can be excited by stimuli smaller than T.

/3/. $(s' < d < s'')$ A period of "subnormal excitability" again.

/4/. $(s'' < d)$ An indefinite period of resting or normal threshold T.

Figure 3/2.1 illustrates a typical curve of "excitability" as a function of d. ("Excitability" is the inverse of "threshold"; $E(t) = 1/T(t)$.)



The curve does not accurately represent the proportional duration of the phases, (or the accurate values of E(d)) for any particular experiment.

The relative lengths of the phases is of importance in the later chapters. For peripheral fibres, these quantities vary in different specimens, and this is not the place for a detailed study of the experimental results. Inasmuch as our real interest is in interneural junctions, details of the properties of peripheral fibres are important only insofar as they supply a model which the junction may resemble in some respects. Although quantitative data is available as to the relative lengths of the phases in fibres, these data are not directly applicable to our model of the interneural junction.

3/2.2 For the theory of the later chapters, the most important aspects of the excitability fluctuations are the existence of phases /0/ and /2/. The existence of phase /0/, the refractory period, is certain for all excitable tissue, muscle and nerve. For nerve fibres, the absolute refractory period has duration .5 to 2.0 milliseconds, in general. Phase /1/ may last longer.

Phase /2/ plays an important role in the theory, and without it the chapter on the "theory of cycles" would be empty. In the mammalian nervous system three

types, "A", "B", and "C" of nerve fibres are distinguished; of these the "A" and "C" types exhibit a well defined /2/ or /supernormal/ phase. (Fulton 99ff) In the "B" fibres, there is no supernormal phase; recovery is through a continuous subnormal course. However, "B" fibres appear to be confined to the autonomic nervous system, (and there to postganglionic fibres) and it is plausible that the fibres of the central nervous system generally have a supernormal period. This is true of all somatic nerve.

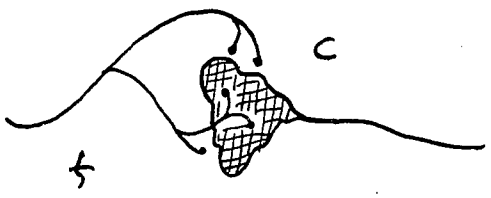
A possibly important feature of the supernormal phase is the fact that if a fibre is stimulated repetitively at very short intervals, the supernormal phase will shorten or vanish. (See again Fulton p 105.) Under the same conditions, the subnormal phases will lengthen.

3/3 Junctions.

If the recovery cycle of 3/2.1 is assumed to reflect a basic process in the surface of the fibre, as is generally believed by those who hold to a "membrane" or equivalent theory of conduction, then it is plausible that the same process takes place at the surface of the efferent cell of the interneural junction. At such a junction, it may or may not be the case that the excitatory stimulus is an electric current (as it was in the experiments on isolated nerve fibres). But in any case it is still plausible that

the interneural junction goes through a similar excitability cycle following stimulation. If we define a transmission probability for interneural conduction, as was proposed in 3/1.3, it is reasonable to expect that the fluctuations of the recovery cycle of the efferent cell surface tissue will be reflected as fluctuations of the transmission probability of the junction. Let us consider a simple case.

3/3.1 Let J be a junction formed by a number of endbulbs from a fibre f terminating on the surface of a cell C. For simplicity suppose that these are the only endings on N.



Then at any time t, the junction so formed will have a certain transmission probability XP(t). It is plausible, by all the arguments above, that under circumstances like those in 3/2.1, the junction J will pass through corresponding phases of excitability, or with the probabilistic interpretation, XP(d) will pass through corresponding phases. We shall therefore assume that if the junction J is fired at t = 0, and that prior to this J was in a "resting state", then its transmission probability XP(d) at times d > 0 will pass through five phases.

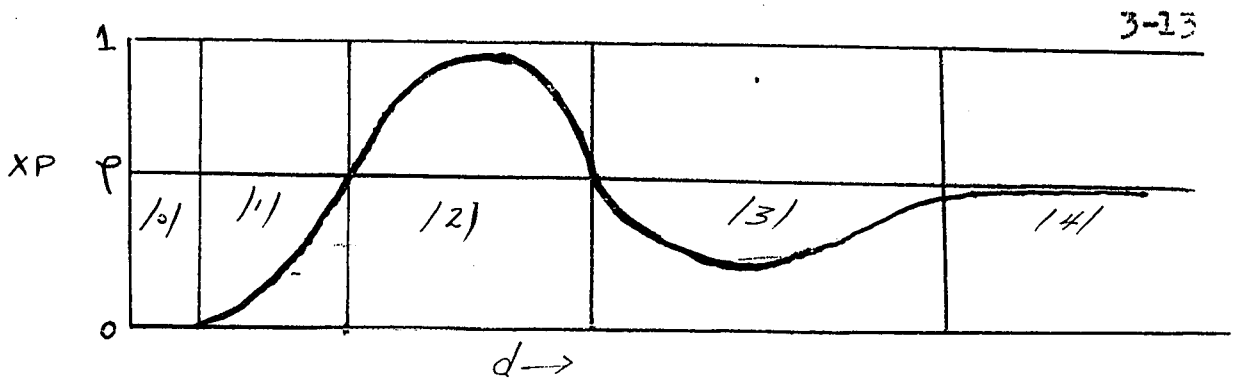


Fig. 3/3.1a

Although the function XP(d) is presumably continuous in d, as indicated in Fig. 3/3.1a, we will generally replace this function by a step function to make certain calculations feasible. For this step function approximation, the phases will be denoted by $/p_i/$ where p_i is, say, some typical value of XP for the i'th phase. We shall let $p_0 = C$, since this is the absolutely refractory period. Since the absolutely refractory period is presumably due to an intrinsic incapacity of the efferent surface to fire during this phase, none of the considerations of 3/1.2 would seem to justify the assignment of even a very small positive XP to the function for this phase. The refractory phase will be denoted by /0/.

The subnormal phases will be denoted by $/p_1/$ and $/p_2/$. The supernormal phase will be called /F/, and the terminal or resting phase is /P/. By definition,

$$p_1 < P,\ p_2 < P,\ F > P.$$

Note: The existence of $/p_2/$ seems to be of minor importance in our theory. Its existence will usually be ignored by the device of

setting $p_2 = p$, and $s' = s''$.

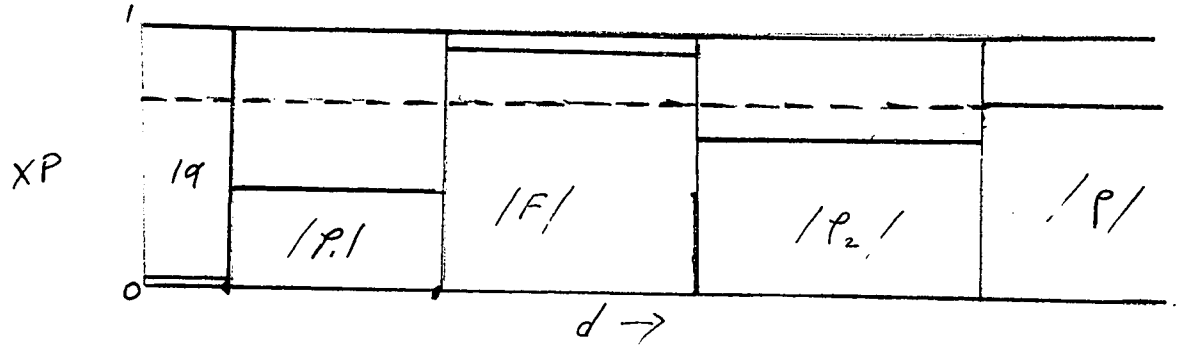


Fig. 3/3.1b

Note: Even when we consider a continuous XP(d), we will use the discrete notation $/p_i/$ for the phases. If we have several cells N_j, then $/P_i/_j$ will denote the $/p_i/$-phase of N_j.

In the chapter on cycles, we shall make some estimates of the duration and probabilities of the phases, using highly heuristic methods. Some supporting biological evidence will be introduced there, also.

3/3.2 If more than one fibre, f_i, has endings on the surface of a cell N, the junction so formed is multiple, and its XP is a function of the stimulus, as well as of the recovery process. There would not be sufficient justification for assuming an explicit form for this function, for little is known about the effect on threshold of so-called "subliminal" stimuli. Pulses which fail to fire a junction may or may not affect its recovery phase, and little is known about this. (For fibres, subliminal pulses lower threshold for

later pulses, if the interval is very short, e.g., 0.5 ms or less.) It would seem safe to assume that if pulses arrive together, or very closely, in any small region, their effect would summate by virtue of summation of the chemical or electric transmitter agent. It also seems fairly safe to assume that (if the recovery cycle is a property of the cell C) when the junction is in phase /F/ due to an earlier firing, that the transmission probability is raised for all stimuli, or at least for the original stimulus and its subsets. Thus a stimulus which ordinarily has only a small chance of firing C, may have a large chance if C (or the part of C's surface concerned) is in phase /F/. Thus, for this kind of junction (a localized region of cell surface with several connections), we will assume that the XP for <u>all</u> (simultaneous and near-simultaneous) stimuli vary together. An analogy in the Pitts-McCulloch model would be the variation of the numerical threshold of the neurons following firing.

It would be convenient if the properties of a localized multiple junction were similar to those discussed in 2/2.5, with a relatively sharp numerical threshold, for then we would have more or less ideal con- and disjunctions. We will have occasion to estimate the effect of assuming sharp thresholds in 5/9.

3/3.3 Synaptic delay.

The time between excitation and firing is called the "synaptic delay". The time quantization axiom would assert that this interval is constant for all junctions and all times. However, this is certainly not the case in the nervous system (Fulton p. 190ff) and if any theorem depended on this assumption, it would be suspect. In accord with the general policy of this theory, it must be assumed that for any given conditions of internal state and excitation, the synaptic delay has a probability distribution with positive dispersion (and, presumably, continuous). The quantization axiom may be approximated by having all such distributions sharply concentrated around unity. However, even disregarding the probabilistic aspect of the synaptic delay for a single junction, it would be reckless to assume that the synaptic delay is the same for different junctions. It is known to vary, in single junctions, with the recovery phase, and for different junctions. Thus any theorem which depends on such constancy must be suspect.

It is known, at least for spinal motoneurons (Fulton 140ff) that synaptic delay is, for a given junction, greater when stimulation is earlier in the recovery cycle, at least in the earlier phases. The fact that synaptic delay is a decreasing function of d(the time between pulses) has an important

consequence in the theory of cycles, in particular on the question of storage of information in cycles.

There are thus two aspects of variation of synaptic delay; that due to individual cellular differences, and that due to the probabilistic fluctuations. It will often be necessary to make the quantization assumption in order to make a typical calculation practicable. In such a case it will be necessary to verify that small changes in timing of the events concerned will not make the calculation invalid.

3/4 REMARKS

There is no justification for choosing any particular distribution to represent any of the phenomena discussed up to now. We will concentrate on theorems which depend only on the form of the distributions as specified already. In particular, the mere existence of the excitability phases / / and /F/ will be the basic condition for most of the results. An attempt can then be made to examine the consequences of assuming particular distributions. When a particular distribution for the transmission probability function is assumed, then any result obtained as a consequence must be regarded only as a model, whose applicability depends on the extent to which it can be safely generalized.

The probabilistic aspects of the behavior of junctions might best be regarded as though thresholds are determinate but that noise is present. Most of the arguments of 3/1.3 lend support to this interpretation.

The description of the junction, up to this point, must be regarded as covering only the "short-term" properties of the junction. In the learning process, it is possible that the junctions also display long-term, and even permanent changes in excitability. We shall take up the theory of permanent changes in chapter 6.

3/5 Global structure of nets.

There is a great deal of available information about the gross structure of brain tissue, but rather little real knowledge about the nature of the connection structure within any small portion of the brain. A microscopic inspection of brain tissue reveals, with certain important exceptions, the following typical picture. (Note: It would be easy to expand the description in much greater detail, even with present information; the description below is a simplification for reasons of brevity. It would be feasible to make a much more detailed analysis of the nets of the brain using only present information, and it is hoped that this will be done presently.)

3/5.1 "Neural cubes".

If certain "specialized" regions of the cortex are excepted, then examination[1] of a small section of cortex gives the following impression.

1. The neurons (i.e. nerve cell bodies) are distributed through the section in a manner showing local irregularities of distribution with a more or less clearly discernable overall structure. In any ~~cube~~ cross-section of cortex there are many different morphological types of neurons; they are distributed in such a way that there is a gross appearance of (six or more) layers, of high neuron density, separated by lamina of relatively fewer cells. However, this laminar structure is far from well defined, in most cases, and it would seem to be most appropriately described by saying that each type of cell has a distinct probability distribution of location, and

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[1] Nervous tissue can be so prepared that only neuronal tissue is visualized. The description omits, of course, the supporting and vascular tissue cells. (This might ultimately turn out to be a serious mistake.)

that the gross laminar appearance of the cortex is a consequence of these distributions. In my opinion, the lamina are sufficiently distinct that it would not be unreasonable to suppose that they have functional significance.

For example, the fact that the motor (pyramidal) cells of the cortex are largely confined to the central layers may be important in that this makes it possible for them to receive impulses from a larger class of cells than would be the case if they were not so centrally located.

If it were not for the laminar distribution, the cortical distribution of neurons might well be called "random". Certainly within any one lamina the distribution appears quite disorderly. (Again, there are certain exceptions.)

ii. The connections between cortical cells also show a partly orderly, partly random, pattern. For the most part, connections show horizontal (parallel to the lamina) preferences, but there is a rich supply of diagonal and vertical fibres as well. The vertical fibres have a very special type of distribution; most of them have

their origin or branches in a few layers, and then run vertically downward directly out of the cortex. In general, the horizontally oriented fibres are rather short, while the vertical fibres which run out of the cortex into the underlying white matter are longer by a large order of magnitude.

iii. The fact that the horizontal fibres tend to be short, together with the fact of the generous supply of vertical fibres entering and leaving the cortex, give the impression that these vertical fibres (tracts) are the primary input-output channels for the sample neural cube. It is my impression, from examination of various preparations, that a pulse entering on an incoming vertical fibre would, typically, pass through only a very few (e.g., two to five) junctions before it would meet an outgoing vertical fibre.

iv. The vertical fibres leaving and entering the cortex may be distinguished into about four groups in the following way:

a. Relatively short fibres running from one side of a convolution to the other.

b. Longer fibres running from one convolution, under the separating fissure, and rising into an adjacent convolution.

c. Still longer bundles of fibres running from one part of the cortex to remote cortical areas in another part of the brain.

d. Long bundles of fibres running between areas of the cortex and non-cortical parts of the nervous system. (Note: The fibre tracts of (a) and (b) may be considered as a slightly larger than "local" feature of the brain. They are uniformly present in the cortex. The tracts of (c) have a different status, as they do not exist between every pair of cortical regions, but have definite "anatomical" locations.)

v. The cortex then may be pictured as a collection of "cubes", each of which is (except for the laminar preferences) a random net, with definite input and output channels (the vertical columns) and which are connected together, through the input and output tracts primarily (the intracortical conduction between cubes may be ignored, or considered as part of (a)) by a set of more or less definite (non-random) bundles. In addition to the intercortical connections are sets of subcortical connections which enter the cubes from specialized midbrain organs. Certain "specialized" neural cubes

receive or send special tracts to and from motor and sensory organs.

3/5.2 In chapter 6 this picture of the brain will be invoked to show how certain processes (in particular certain learning theories) might be realized by the brain. In doing so, there will be an attempt to show which parts of these processes might be realized by certain parts of the brain. It will be necessary then to return briefly to this discussion.

CHAPTER 4 REINFORCEMENT SYSTEMS

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REINFORCEMENT SYSTEMS

4/0 The object of this chapter is to describe a few types of reactive systems (See Chapter 1) and introduce some concepts which will aid in the behavioral descriptions of those systems. The models and descriptions are all related to a general concept of "reinforcement". This concept is very closely related to that used in several current behavioristic models of learning processes of animals. I will define special types of "reinforcement", in connection with the description of particular reactive systems. However, I have found the terms "reinforcement", or reinforcement operator" useful in informal discussion and feel that restriction to a precise definition would result in a loss of conceptual power.

4/0.1 A "learning theory" for an organism, or for a material system, is a theory of those changes in the reactive and/or the internal properties of the object (or the abstract system) which are "due to the effect of the previous 'experience' of the object". ("Experience" here means, generally speaking, a description of the history of the object with special emphasis on the role played by interaction with the environment.) A "learning theory" thus attempts to explain aspects of behavior which

are not known to depend on inexorable internal changes of structure (e.g., "maturation"), or on "built-in" reactive systems.

It is possible to distinguish, in the behavior of many animals, several (apparently) different types of "learning". Many psychological terms suggest such divisions. It is quite possible that these divisions may in many cases reflect the influence of physiologically distinct processes, and it is likely that any satisfactory theory of the behavior of a reasonably complex may entail descriptions of several fundamentally different physiological mechanisms of "learning", and a theory of their interaction. This is the case in the theory of Chapter 6. In that chapter, two types of "learning" are especially prominent; "Reinforcement" and "association"; others also play a role.

4/0.2 An object exhibits "associative learning" if it tends to acquire "related" behavioral roles for sets of stimuli (or "experiences") which themselves are, or have been in the past, related in "experience" (e.g., by temporal proximity, or by virtue of common, or ("previously associated" substimuli). (It would be futile to attempt to use "association" in any precise way; this would only invite misunderstanding and confusion with the many

fundamentally different psychological concepts that parade under that title.)*

4/1 "Reinforcement" is a form of learning in which "an element, X, of the behavior" of an object, M, is caused to become more (or less!) "prominent" in the future behavior of M as a result of the "activation" of a special entity or process, Z, called the "reinforcement operator") within a (relatively) short time after an occurrence of X.

The entity Z is not to be regarded as having any special relation to the particular "behavior element" X. The object (or organism) is to be regarded as actually containing an object Z or at least a distinguished "channel" Z through which reinforcement is effected, and it is also to be implied that Z can "reinforce" ("increase, or decrease, the 'prominence' of") any, or at least a sizeable class of, "behavioral elements".

Two aspects of reinforcement are crucial.

1. There must be available an interpretation of "prominence of X in the behavior of M".

2. In order for a "behavior element", X, to be "reinforced", (i.e., "learned by reinforcement"), it must first occur.

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*Association is defined here only in order to emphasize that "reinforcement" is just one type of "learning theory", and to prevent the misconception that the present empha-

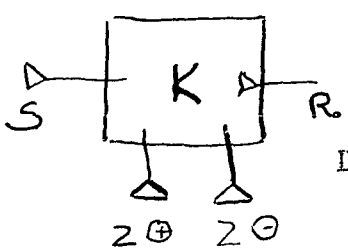
In order to make the discussion reasonably meaningful, we introduce a simple model, and some of its realizations.

4/1.1 A primitive reinforcement system.

Consider the following stochastic-neural-analog net K.

K has four cells: S, R, Z ⊕, Z ⊖.

S, Z ⊕, and Z ⊖ are input cells; R is an output cell. [The following dependency law is not intended to resemble any property of biological cells:



D1: $R(t) \Rightarrow S(t-1)$. (Only the firing of S can produce a response)

D2: $S(t-1) \Rightarrow \text{prob}[R(t)] = f(t)$

$= f[Z\oplus(t-1), Z\ominus(t-1), R(t-1), f(t-1)]$

where f can have any of a sequence of values:

$$\ldots a_{-2} < a_{-1} < a_0 < a_1 < a_2 \ldots \quad [f_1]$$

for which $\lim_{n\to+\infty} a_n = 1$ and $\lim_{n\to-\infty} a_n = 0$.

and for which

$$f(0, 0, x, a_n) = a_n = f(1, 1, x, a_n) \quad [f_2]$$

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sis on reinforcement will lead to a theory of behavior based solely on reinforcement. Certainly a reinforcement theory cannot explain all the behavior of any complicated organism.

$$f(1, 0, 0, a_n) = a_{n-1} = f(0, 1, 1, a_n)$$
$$f(1, 0, 1, a_n) = a_{n+1} = f(0, 1, 0, a_n)$$

Suppose that the net is placed in an environment for which (t)S(t). Then there are only two "elements of behavior" possible: [S(t-1), R(t)] and [S(t-1).~ ~R(t)]. Call these R(ight) and L(eft) respectively. (The system can be regarded as an "ideal" or "Stat"-rat, placed, at each t, at the choice-point of a T-maze. The rat makes the decision to turn right with probability f(t) or left with probability 1-f(t). The event Zθ (t+1) is an ideal "reward" for the decision made in response to S(t); it raises the future probability of that decision. The event Zθ (t+1) is an ideal "punishment" for the response to S(t); it results in a lower future ability for that response.) The joint event {Zθ (t). Zθ (t)} is considered to be without effect.

Of course we know that in real life, and in experimental psychology, "reward" and "punishment" have no such duality. We shall use the terms "positive reinforcement" and "negative reinforcement" for models in which there is such a duality. It is perhaps worth noting that the notion of "reward" is not too inappropriate; the effects of conventional "rewards", or "reinforcers" for animals almost always

causes a future increase in the probability of the corresponding choice. The effects of punishment on animals, however, is often so bizarre that it is proposterous to suggest a duality. The withholding of an "expected", or usual, reward has a more regular effect that "punishment", and it is in the direction of the above "negative reinforcement". Witholding of a reward is, or results in, "extinction"; a primitive model of biological "extinction" can be obtained by providing that if $f(0) = a_0$, before any training has occurred, then $f(0, 0, 1, a_n)$ is between a_0 and a_n. There exist several such stochastic models of simple learning, however, and we do not intend to adjoin one here. The model is intended solely to exemplify def. 4/1.0.

In this model, the conditions (1) and (2) of 4/1.0 are fulfilled by this net: Since the probability of L or R is never zero, either can occur (if reinforcement is withheld long enough), and "f(t)" supplies the interpretation of "the prominence of R"(L)" in the behavior of K. It is possible to "train" K so as to "prefer" either R or L: If one applies Z+ whenever K does the desired thing, or Z- when it does the undesired thing, or both, then condition (f_1) implies that the behavior of K will

approach the desired behavior, with arbitrarily high accuracy, as time goes on.

4/1.2 If the probability of a behavior element were zero, then it could never be reinforced. Although a probabilistic reactive system can most easily satisfy the requirements (1) and (2) for a reinforcement theory, it is perfectly possible to construct determinate processes which have reinforcement. In a later section, it will be seen that the SHARC (4/5) can be so regarded. With this exception, discussion of reinforcement theory will be confined to probabilistic systems.

The definition of 4/1.0 of "reinforcement" is incomplete in one regard. There is no specification as to the effect of Z on the prominence of behavioral elements which did not happen to occur shortly prior to the application of Z. Obviously there must be some such effect, since the prominence (or probability) of an element can be altered only at the expense of others. (In the model of 4/1.1 this effect is, of course, specified.)

Rather than specialize the general concept of reinforcement to some specific description of a behavioral scheme, it would seem preferable to define different types of reinforcement operators, distin-

guishing them by their manner of treating behavior elements which have not occurred shortly prior to the application of the operator. No systematic classification is contemplated here.

4/2 A reinforcement system with an "evaluator"

The following system is constructed in order to present additional concepts which are associated naturally with that of reinforcement. It will be seen that the reinforcement operator of this particular system has a quality of disjointness that certainly cannot be expected in a biological organism. (Nevertheless it may be an instructive oversimplification.) The discreteness can be regarded as a consequence of the extermination of all traces of association theory. In spite of this artificiality, the related concepts are directly applicable to other systems to be analyzed.

4/2.1 i. The construction is started with the introduction of a reactive system M. M has one input cell E and one output cell F. E has m input states $e_1,\ldots, e_m$; F has n output states $f_1,\ldots,f_n$. The system operates in time quantization, and at any time t the dependency law is an m x n matrix $M(t)=([m_{ij}(t)])$, where

$$e_i(t-1) \Rightarrow [\text{prob}(f_j(t)) = m_{ij}(t)]$$

M(t) may be regarded as the internal state of M at time T.

ii. The internal state M(t) of M is under the control of another entity Z. Z has three input cells E*, F* and v. E* has m states e_i*, F* has n states, f_j* and v has three states, called -1, 0, +1. For each input condition [e_i*(t), f_j*(t), v(t)], Z applies to M(t-1) an operator Z_v^{ij} to form a new internal state $M(t) = Z_v^{ij} M(t-1)$. The Z operators are required to have the form:

Z0: $Z_v^{ij} m_{kL}$ is a monotone (↑, or ↓, or constant) function of m_{kL}

Z1: $Z_v^{ij} m_{kL} - m_{kL} = 0 \quad (i \neq k)$

Z2: $\text{sign}\,[Z_v^{ij} m_{ij} - m_{ij}] = \text{sign}\,(v)$

Z3: $\text{sign}\,[Z_v^{ij} m_{iL} - m_{iL}] = -\text{sign}\,(v) \quad (j \neq L)$

It is convenient also to assume that

Z4a: $\lim_{N\to\infty} [(Z_v^{ij})^N m_{ij}] = 1 \quad \text{if} \quad v = 1$

Z4b: $\lim_{N\to\infty} [(Z_v^{ij})^N m_{ij}] = 0 \quad \text{if} \quad v = -1.$

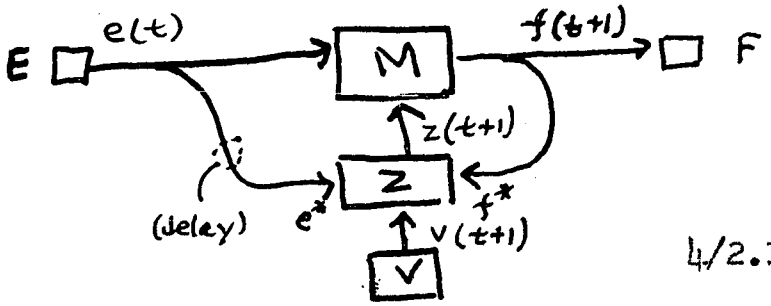
and that for all i, j, t,

Z5: $0 < m_{ij}(t)$.

Z1, Z2, and Z3 imply that application of Z_{+1}^{ij} increases the probability that e_i will be followed by f_j, and decreases the probability that e_i will be followed by any other f_L. Application of Z_{-1}^{ij} has the reverse effect. Z_0^{ij} is the identity.

Z_4 implies that with a sufficiently large number of applications of the operators, "reactions" $[e_i; f_{j(i)}]$ can be "trained" with arbitrarily high accuracy. These reactions are independent in the sense that to each e_i, any f_j can be attached. Z5 is necessary to provide flexibility in the following applications.

iii Z, as above defined, is not a reinforcement agent since it is not connected in such a way that its effect depends on the recent activity of M. This can be remedied by connecting (or identifying) E with E* and F with F*, in



4/2.1

such a way that $e_i(t-1) \equiv e_i^*(t)$ and $f_i(t) \equiv f_i^*(t)$. See Fig. 4/2.1. (The operation may seem simpler if a unit delay is inserted as shown in Fig. 4/2.1 and Z_v^{ij} regarded as a function of $[e_i^*(t), f_i^*(t), v(t)]$ rather than of $[e_i(t-1), f_i^*(t), v(t)]$.) Then Z_v^{ij} becomes a function of the immediately preceding reaction $[e_i(t-1); f_j(t)]$, as well as of the value of $v(t)$. If $v(t) = +1$, then Z acts in such

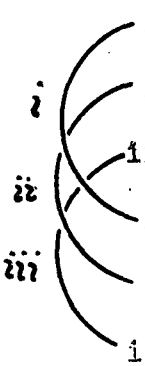
a way as to increase the future probability of the preceding reaction, and to depress other responses to the stimulus e_i. If $v(t) = -1$, then Z operates in such a way as to depress the future probability of that reaction. If $v = 0$, future probabilities are unaffected. In each case, $Z(t)$ affects only the distribution of responses to the particular stimulus e_i which was present at the preceding moment t-1. Hence it does not, in any way, influence the effects of previous "training" of responses to stimuli other than that e_i. For this reason Z may be called "disjoint".

The entity Z, when so connected, is a reinforcement operator. It is said to be "controlled through the channel (or by the cell) v". When v is 1, the reinforcement is "positive" and when v is -1 the reinforcement is "negative."

(Note: Without further elaboration we shall regard it as meaningful to speak of "reinforcement channels" through which one can control the "magnitude" as well as the "sign" of reinforcement, if necessary by the control of continuous parameters of the reinforcement operators.)

4/2.2 It is easy to describe schemes by which sets of reactions can be trained. Suppose that it is

desired to train the reactions $[e_{i(t-1)}; f_{k(i)(t)}]$. Then the function k(i) describes the set of desired reactions. Present successively to ... all stimuli, either in an orderly sequence, or in a time series in which each stimulus occurs with positive density. Any of the following "reinforcement schedules" will train all reactions:



i. v = 1 for desired reactions,
ii. v = 0
iii. v = 1
i. v = 0 for undesired reactions,
ii. v = -1
iii. v = -1

Each stimulus occurs with positive density, and by Z5 each response then has positive probability, at any time. Z1-Z3 then imply that the desired responses never fall below their initial probability, hence the desired reactions always have at least their initial positive density. Then Z4a-b shows that schemata i and iii will raise the desired response arbitrarily close to probability 1.(Z1-Z3 show that the rise is non-decreasing.)

Scheme ii works because no undesired reaction can maintain probability $> \epsilon > 0$. For let $[e_i; f_u]$ $(u \neq k(i))$ be an undesired response. Let e_i have density $d > 0$ in the stimulus time series. If

$m_{iu} \geq e$ for all time, then the reaction e_i-f_u occurs with density $\geq d.e$, and with probability 1 occurs infinitely many times. Each time $[e_i\ f_u]$ occurs, so does Z_{-1}^{iu}. Hence (by Z4b and Z_3) m_{iu} approaches 0 which is a contradiction.

4/2.3 The scheme of 4/2.2 introduced three more entities into the system.

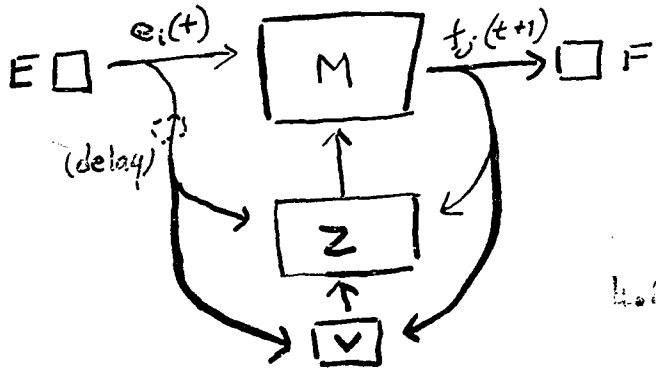
(1). There was a selection of a class of desired reactions, defined in the above case by a function k(i).

(2). There had to be an agency which recognized whether or not the preceding reaction belonged to this class, and then activated the reinforcement control channel v accordingly (in accord with one of the schemata of 4/2.2).

(3). There was a source of stimuli presented to E.

Now (1) and (2) together are equivalent to an agency V which "observes" the reactions of the object M, assigns a numerical "evaluation" to each reaction, and transmits this evaluation to Z through the channel v. In 4/2.2, values were limited to -1, 0, and 1 (and to just two values for each of the

three suggested schemata). Of course with a more flexible reinforcement system Z, the evaluator V could assign a more elaborate signal to each reaction, and the system could assign various degrees of reinforcement to reactions rather than simply categorize them as desireable or undesireable. Fig. 4/2.3 illustrates the form of a realization of the system with V adjoined.



4.2.3

(Again it may be convenient to imagine a unit delay to be present in the E-side of Z and V, to emphasize that those entities respond to reactions [E(t-1); F(t)].

4/2.4 DISCUSSION

It will be noted that now the system has a high degree of autonomy. It can be regarded as a reinforcement machine [M,Z], with an associated trainer V. It is at this level of organization that many individuals assert:

"How can one call this a 'learning' machine when obviously the activity to be learned

is already installed in V? The machine is already 'built' to perform the desired task."

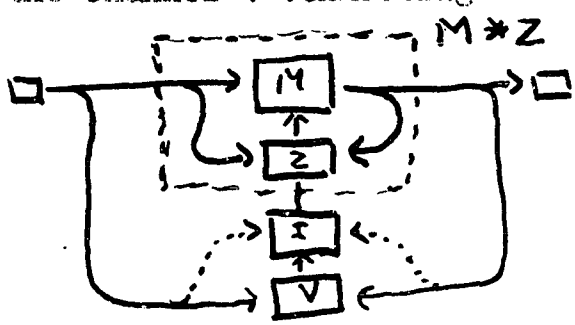
4/2.4.1 It seems that the difficulty lies in the notion of 'built-in', a really deceptive phrase. For in my experience, it has frequently occurred that the questioner is more or less willing to admit that the subsystem [M,Z], with V deleted, appears to be a "learning machine" so long as the channel v is controlled by the human hand, but that in some sense the more complex system [M,Z,V] is acting in a less sophisticated manner. The inquirer has not perceived that there is a difference between an object in which the desired reactions themselves are installed, and the object [M,Z,V] in which information about the reactions is stored. (One might say that in V, only the "names" of the desired reactions occur.) Furthermore, the information in V does not even suggest the manner in which M is to realize the reactions, nor does Z contain such information. We shall see examples in which M can realize a reaction in several ways, and where V does not select among these. Fundamentally [M,Z,V] evolves; M makes attempts and V, through Z, evaluates and selects.

4/2.4.2 Another viewpoint for this question of autonomy is indicated by the notion of "open" and "closed" structure. A "reactive system" is distinguished from other abstract systems not by any intrinsic quality, but by the characterization of certain cells as "observable" and others as "adjustable". A system A will be called "open" to another system B when a large fraction of the cells of A are "observable or adjustable by B", and "closed" to B when this is not the case. (A cell c is "observable" by B when the state of cells in B depends on the state of c, and similarly for "adjustable". We do not need more formality.) A typewriter is quite open to the typist, a desk calculator is substantially more closed. A laboratory animal is rather tightly closed.

Now in the system [M,Z,V] as described in 4/2.1, M was more or less widely open to Z. For the channel from Z to M is sufficiently wide to permit Z to adjust any element in the matrix $((m_{ij}))$ in either of two directions, and (assuming that M is itself not very complex, i.e., that Z acts relatively directly on the cells of M) it would be fair to say that Z has an appreciable direct control over the structure of M. Hence if a (say) human operator has access to the channel from Z to M, i.e., can

select arbitrary operators $Z_v ij$, then it would be fair to say that the system M does not "really learn", but that the human operator is more or less directly adjusting its structure to perform the desired reactions, i.e., he is "building-in" these reactions.

However, when Z and M are connected as in Fig. 4/2.1, the entire width of the channel between Z and M is not really essential, since the variables i and j of Z_v^{ij} are not independent of what is happening inside M. In fact, the values of these variables are accessible from within M. One could therefore construct a new system [M,Z,I] which is externally equivalent to [M,Z] by connecting all the cells of Z whose states depend on i and j to appropriate points within M, or simply define M*Z as the enclosed region of Fig. 4/2.3; and let I be a vacuous Z' with the channel v connecting it with M*Z.



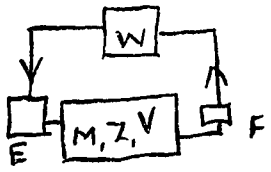
Certainly M*Z cannot be said to be widely open to the trivial object I = Z'. Neither can it be said to be open to V. Thus one cannot assert that the

information in (or the activity of) V acts directly to adjust the structure of M (or M*Z) to perform the reactions.

There is no reason to expect to find a sharp boundary between "learning by experience" and "alteration of internal structure by an outside agency". It would seem that the notion of "degree of openness of A to B" is likely to be more useful than that of "built-in vs. learned", in discussions like the present one. In particular, the greater the extent to which Z*M is open to V, the less natural is the description of the system in the terms of reinforcement theory. To the extent to which the structure of the whole system [M,Z,V] is open to the environment, its behavior is the less dominated by the reinforcement system.

4/3 COUPLING WITH ENVIRONMENTS

In order to provide the system [M,Z,V] with a source of stimuli E(t), it will be embedded in an environment W. Because attention is directed toward the reinforcement system, it will be assumed that the system is closed to W except for the cells E and F, and that W is closed to [M,Z,V] with the same exceptions. Both W and [M,Z,V] will be regarded as reactive systems, with common cells E and F.



4/3.1 If the system $[M,Z,V]$ of 4/2 is taken intact and coupled to W through E and F, the behavior of the resulting system will of course depend on the properties of M, and the behavior of the whole system viewed as a time series may be very complex. However, the behavior of the state $((m_{ij}))$ of M will be, at least in the limit, rather simple, inasmuch as the system is so rigid that each term m_{ij} is monotonic (or constant), if e_k occurs infinitely often then each m_{kj} tends to either 0 or 1. If the behavior is such that e_k does not occur infinitely often, then m_{kj} is, after some time, fixed at some value between 0 and 1. But this value has no influence on behavior since e_k will not occur again! Thus as time goes on, M approaches determinacy in its behavioral properties.

The above argument applies only if V applies reinforcement after each reaction. If V never applies reinforcement after some reaction $[e_o(-t-1);f_o(t)]$, then the response to e_i may remain probabilistic. In any case, because of the monotone behavior of all terms m_{ij}, the state $((m_{ij}(t)))$ of M must approach a limit.

It has been assumed throughout that for a given input e_i, V applies positive reinforcement for not more than one response $f_{k(i)}$. If this is not the case, then the

the above arguments fail because the terms of $((m_{ij}))$ do not necessarily behave in a monotone fashion. In this case, the behavior of $((m_{ij}))$ depends on the behavior of $\mathcal{M}$, and it can be seen that (by providing $\mathcal{M}$ with a history-dependent internal state function $((M^*_{ji}))$, complicated stochastic processes $E(t)$ can be generated.

4/3.2 There is one outstanding feature of the behavior of the system under discussion. The object $[M,Z,V,]$ seems to be more loosely coupled to $\mathcal{M}$ than would seem appropriate for a theory to be applied to brain models. The fact is, so long as reinforcement depends only on pairs $[e(t-1);f(t)]$; the reinforcement system of $[M,Z,V]$ is controlled entirely by the reactions of M. The system will succeed in "learning" the reactions "preferred" by V, but in a sense it does not "learn anything from the environment." The structure of $\mathcal{M}$ tends rather to influence the rate at which V trains M. Even if V has the flexibility suggested in the previous paragraph, the situation is not essentially changed. For it can be shown that the state of M at time t, is independent of all aspects of the time series $E(t)$ other than the simple statistics:

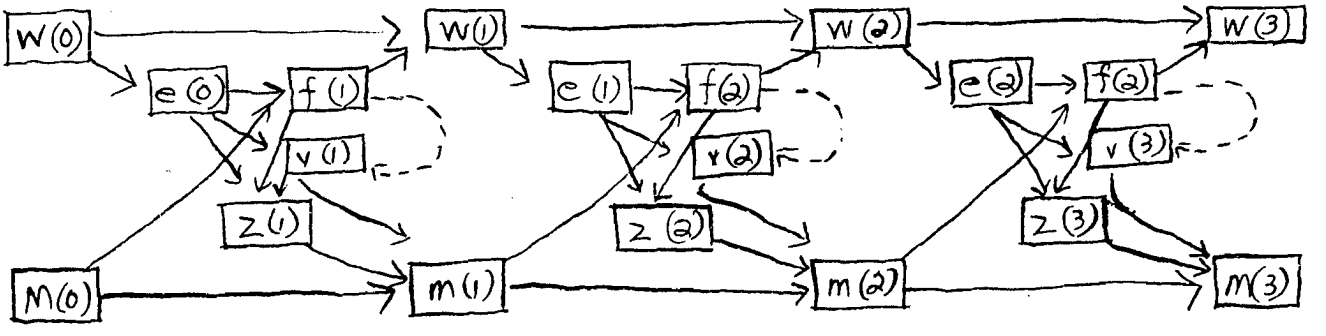
$\#e_i(t_o)$ = the number of times e_i has occurred up to time t_o.*

*This depends on the fact that Z is disjoint. If this were not the case, M would depend on other aspects of the time series $E(t)$. In general, however, this dependence would have little to do with the relation between M, $\mathcal{M}$, and V.

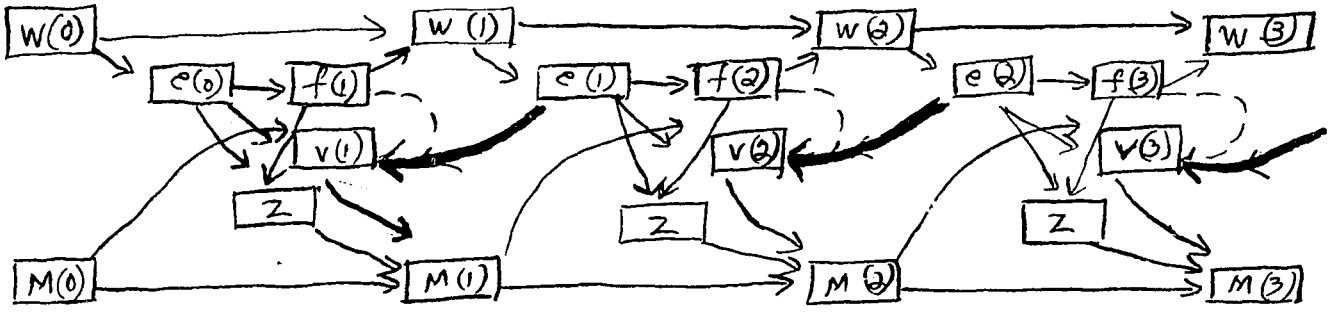
Thus from the point of view of [illegible], [illegible] acts as a source for these simple functions, and all other aspects of the behavior of [illegible] is ignored.

A very small alteration in the structure of $[W,Z,V]$ will alter this situation, and provide an opportunity to introduce a few more concepts naturally related to reinforcement.

4/3.2.1 If the valuation made by V is a function of e(t-1) and f(t) alone, we have seen that behavior of $[W,Z,V]$ depends on that of the environment W in a rather trivial manner. However, if V(t) is made to depend on the values of e and f at other times, then reinforcement will depend more essentially on the behavior of W. Fig. 4/3.1.1 is a diagram showing the dependency relations between the variables of the model of 4/2 and 4/3.1. W(t) is the internal state function of W. M(t) is the matrix $((m_{ij}(t)))$. The changes in M(t) depend on the values of v(t) which in turn depend only on the reactions $[e(t-1);f(t)]$.



Now let the connections be changed so that v(t) depends on e(t-1) and e(t) instead of e(t-1) and f(t):



The behavior of the reinforcement system is now quite different. The activity of V no longer depends solely on the reactions of E, but also on the effect of these reactions on E. Let us first consider the behavior of the system for a particularly simple E.

4/3.2.2 Assume that the state of E is changed only by the action of M, so that if E(t) is the state of E at time t,

$$E(t) = E(\, E(t-1), f(t)\,)^{*} \qquad (1)$$

Assume also that the states of E correspond in a one-one fashion to the possible stimuli e_i.** Then we can write

$$e(t) = E(\, e(t-1), f(t)\,) \qquad (2)$$

The (finite number of) states of E, together with the transitions T_{ij} defined by $T_{ij}e_i = E(\, e_i, f_j\,)$ form

---

*Note that (for convenience) we assume that E reacts to f without the usual delay.

**This corresponds to a model in which the environment is "completely observable" to the system [E,Z,V].

an oriented graph, G. The properties of $\mathcal{F}$ determine the connectivity of G, and the system point will remain in the component in which it originates. We may assume, then, that G is connected. After a finite time, the system point will remain confined to a subgraph each of whose points are accessible from any other, (along a finite oriented chain of transitions) and which is maximal for this property. It will be assumed that G is so connected.

4/3.2.3 Suppose that $v(e_i,e_j)$ has the simple form

$$v(e_i,e_j) = \pm 1 \qquad (3a).$$

Let D be the set of all e_j for which, for some i, $v(e_i,e_j) = 1$. Then

$$\lim_{t\to\infty} [\text{prob}(e(t) \in D)] = 1.$$

Proof: Let e_0 be an arbitrary point not in D. We show that $\overline{\lim_{t\to\infty}}(\text{prob } e_0(t))$ cannot be positive. Now

$$\text{prob}(e_0(t+1)) = \sum(i,k) \text{ prob } (e_i(t)).\ m_{ik}(t)$$

summed over all (i,k) for which $e_0 = v(e_i,f_k)$. Then

$$\overline{\lim_{t\to\infty}} \text{prob}(e_0(t+1)) \leq \sum_{(i,k)} (\overline{\lim_{t\to\infty}} [\text{prob}(e_i(t)).(m_{ik}(t))].$$

If any term (i,k) is positive, then the event $e_i(t).f_k(t)$ must occur infinitely often. But then the reinforcement operator Δ_{-1}^{ik} is applied to m_{ik} infinitely often, and since Δ_{-1}^{ik} never occurs, $m_{ik}(t) \to 0$. But

$$\overline{\lim_{t\to\infty}}(\text{ prob } e_i(t).m_{ik}(t)\ ) \leq \overline{\lim_{t\to\infty}}(\text{prob } e_i(t).\overline{\lim_{t\to\infty}}(m_{ik}(t)) = 0$$

hence $\lim_{t\to\infty}$ prob $e_0(t+1) = \lim_{t\to\infty}$ prob $e_0(t) = 0$. This completes the proof.

Hence, if V operates according to (3a), the system learns to influence the environment in such a way as to bring it to and maintain it in a subclass D of states defined by the values of v.

4/3.2.4 The evaluation function $v(e_i,f_j)$ not only determines the class D of preferred states of W, but it also defines a class P of preferred transitions; namely those T_{ij} for which $v(e_i,e_j) = 1$. If P* is the subset of P for which both e_i and e_j are in D, then it can be shown, by an argument similar to that in 4/3.2.2, that not only does the system point remain in D with probability approaching unity, but that the transitions within (and without) D belong to P* with probability approaching unity. If P* divides D into connected components then the system will tend to remain for increasingly longer intervals in any component.

In general, however, the behavior of the system <u>will not approach determinacy</u>, for it may occur that for some i,j,k with $j \neq k$ both T_{ij} and T_{ik} are in P*. In this case, it may turn out that several distinct responses to the stimulus e_i are positively reinforced,

and the asymptotic behavior may, if Z has the proper form, show a non-trivial probability distribution for the transitions from e_i. (The behavior here will depend critically on the exact values of the arbitrary functions in the definition of the Z operator.)

The important aspect of the system of 4/3.2.2 and 4/3.2.3 is that the function v distinguishes sets of desired states and transitions of W, and the system learns to react in such a way as to bring W into these states and transitions. The role of the individual reactions (e_i,f_j) of M is subordinate to the behavior of W. In fact if a transition (e_i,e_j) can occur through several reactions $(e_i,f_u),(e_i,f_v),\ldots$, the reinforcement process is not basically influenced by which of these reactions actually occur. For positive reinforcement of <u>any</u> of these reactions results in increasing the total probability of the transition (e_i,e_j), unless this probability is already unity. Proof: If m_{iu} is increased, then $\sum_{k\neq u} m_{ik}$ is decreased by an equal amount; and each non-zero term is reduced (by Z3, sec 4/2.1). If m_{iL} is such that $e_j \neq N(e_i,f_L)$ then the total probability of the transition (e_i,e_j) must thus be increased by at least the amount (>0) by which m_{iL} is reduced. Thus, positive reinforcement after a transition always increases the probability of that transition (if $p<1$). (If there is no such m_{iL}, then (e_i,e_j) has probability one to begin with.)

If [M,Z,V] were so "damaged" that one of the alternative reactions for (e_i,e_j) could not occur, the system would remain capable of learning to cause the transition by using the remaining alternatives.

NOTE: It is further possible, by suitable choice of the arbitrary functions of Z, to arrange the reinforcement to so act, that whichever alternative reaction first occurs will with arbitrarily high preassigned probability, remain the reaction through which the desired transition occurs. Referring back to the discussion of 4/2.4, it may be remarked that in the present case, the content of V does not even determine the reactions which M will use to bring about the desired states and transitions. The behavior is even less "built-in" than in the examples discussed in that section.

4/4 Discussion

It would be interesting to examine the transient as well as the asymptotic behavior of the system of 4/3.2 in further detail, and also to study the effects of allowing $v(e_i,e_j)$ to take on the value 0, but this particular system is already too specialized for application in the sequal. There are two important directions in which the model must be generalized.

1. In any real situation W(t) will not be completely determined by the behavior of M, and e(t) will not supply complete information about W(t).

2. The reinforcement processes with which we shall be concerned will be concerned with reactions and valuations that extend over a period greater than one or two time units.

4/4.1 If e(t) does not supply complete information about W(t), i.e., if the state of E at time t is a many-one function of the state of W at time t, then the class D (4/3.2.2) determines a class $\underline{E^{-1}D}$ of sets of states of W, and the class P* determines a class $\underline{E^{-1}P^*}$ of transitions between members of these sets. Neither in reaction or in reinforcement can [M,Z,V] distinguish between elements of the same $E^{-1}e_i$. The reinforcement machine can learn to manipulate (as specified by the values of v) only those aspects of the behavior of M which it can "perceive," i.e., which can be defined in terms of the values of e(t). The events e_i rather than the events W_i are elements of the "perceptual world" of M.

If the transitions between states [e(t);e(t+1)] do <u>not</u> depend to any great extent on the most recent response f(t+1), then the reinforcement system of 4/3 can do little to control W. Such a situation may arise in several ways.

i. The behavior of M, i.e., the values of f(t), might have little influence on W. Then clearly [M,Z,V] cannot exert much control over W (or over E).

ii. If e(t) provides too little information about $\Psi(t)$, or about those aspects of the behavior of $\Psi(t)$ which are affected by M, the reinforcement machine cannot be expected to be able to learn to exert much control over Ψ.

iii. It may be that the values of e(t) do not immediately reflect the changes in Ψ due to the activity of M. Then when a change in Ψ is finally observed by M (through E), and reinforcement applied, this reinforcement will affect only more recent reactions which will not have been concerned with bringing about the change of state observed at E. Hence this reinforcement cannot be expected to alter the probability distribution of that state change in the desired direction.

The limitations imposed by i) and ii) occur when the relation between M and Ψ is "closed" in one direction or the other. (See 4/2.4.2) Under such circumstances M could control Ψ only to the extent that M contains "built-in" information about the behavior of Ψ, since if either the E or the F channel is closed, it cannot obtain such information directly. But M, of course, does not contain such information, initially.

The limitation of iii) is deeper. The system $[M,Z,V]$ as described in 4/3 is, in two respects, incapable of

dealing with events of extended duration. First, the valuation v(t) depends only on the simple transitions of the time series e(t), so that reinforcement cannot be assigned to events of greater duration than simple transitions. Secondly, the Z operator affects only the future distribution of the most recent reaction, so that even if V were able to distinguish special subsequences of e(t), reinforcement could not be applied to influence all the reactions involved in production of particular sequences.

Thus if M exhibits extended reactions to its stimuli, or exhibits special reactions to extended stimuli, [M,Z,V] will not be able to influence these aspects of M's behavior. Any acceptable brain model must be able to "cope with" environments which require sequential activity for their "control." If [E,M] is such that extended observation of e(t) is required to gain information about M(t), then V must be able to respond to special sequences. This can be accomplished in two ways: (1) M can be altered directly so that a stimulus can be a sequence of states of E, or (2) Z can be altered so as to reinforce (as directed by V) all reactions that M has made in some relatively long past interval. The latter scheme (2) will not be very flexible since it means that M learns to react, to an extended sequence of stimuli, with a sequence of

responses each of which is an immediate response to the preceding stimulus. Behavior built up out of immediate responses cannot be trained to make arbitrary responses to different sequences of stimuli which have common elements.

In the models of later chapters, response to and production of non-trivial sequential activity will usually be a property of each net, and the reinforcement systems used will operate on the behavior of appreciable intervals of the past.

4/5 Elementalistic Reinforcement Systems

4/5.0 Discussion

Sections 4/2-4/4 have been concerned with certain properties of the activity of a model reinforcement system [N,A,V]. Each of the objects N, A, and V were regarded as reactive systems, i.e., they were discussed behavioristically, exclusively in terms of the behavior of their input and output channels and of their internal state function. (The internal state function is a behavioristic concept insofar as it is regarded as a description of latent input-output activity and not as a description of the physical internal state of an actual material system.) We now consider the construction of material systems which are required to simultaneously realize a reinforcement theory and a neural network. Again, the models in this section are

constructed only for purposes of illustration; serious proposals will (necessarily) not be formulated until essential results in the theory of cycles have been established (Chap. 5).

4/5.1 "Local" Reinforcement Operators

The term "reinforcement" has been used up to this point in a behavioristic sense, as denoting a process or operator which acts on the (observable) behavior of a reactive system N in the manner indicated in 4/1.0.

A more elementalistic concept will now be introduced, that of "local reinforcement" or "local reinforcement operator" (LRO). It is felt that this notion is sufficiently closely related to that of "reinforcement" to justify the similar terminology. While a "reinforcement operator" acts on the behavior of a reactive system N, a "local reinforcement operator" acts on the elements (abstract or physical) of the structure of N, and influences behavior indirectly. As in the case of "reinforcement," too special a definition would result in a loss of usefulness of the concept, and perhaps invite irrelevant controversy.

Definition

Let N be an object with input and output channels E and F which contains, in its interior, a collection of distinguished objects C_n called "choice cells."

Each C_n is itself a reactive system with input and output channels I_n and O_n, and an internal state function $\ldots_n$ which determines its reactive properties. Suppose that the reactive properties of M are determined, at any time, by the reactive properties, at that time, of the C_n. (It may be assumed that M is a neural-analog network with the C_n being the cells, and the connections of I_n and O_n forming the fibres.) Each reaction of M is supposed to be a direct consequence of reactions of the C_n (responses of the C_n to stimuli from M or from other C_m).

Finally, suppose that with each C_n there is associated a reinforcement operator $\ldots^n_{v_n}$, and that all control variables v_n have at any time t the same value z(t). Let this value be under the control of an external entity $\bar{E}$

Such a $\bar{E}$ will be called a local reinforcement operator.

4/5.1.1 The basic concept is that of an object M whose reactions are consequences of the reactions of some of its parts C_n. During any reaction $[e_i(t),f_j(t+1)]$ of M, some of the C_n "make a choice." Application of a positive value of z results in the increase of likelihood that in the future, under the same conditions those C_n involved will have the same reactive properties. It does not follow, that this must necessarily result in

the increase of the probability that e_i will be followed by f_j in the future, i.e., that local reinforcement implies behavioral reinforcement.

It will nevertheless be necessary, in later examples, to assume that they are, to a large extent, equivalent. When this must be done, plausibility arguments will be provided.

4/5.1.2 As a first example of a local reinforcement system, we will examine a system which may be considered of special interest in that a material realization has been constructed.*

4/5.2 The SNARC machine. A neural-analog net with a local reinforcement operator.

4/5.2.0 The following is a description of a realization of a LRC in a neural-analog net. The machine, being a physical object, and not digital in operation, cannot be described completely; the system below resembles the machine closely, but there are some essential qualifications that must be made. These remarks will be

*This machine is the SNARC (Stochastic Neural-Analog Reinforcement Calculator) constructed by the author with the very generous assistance of Mr. D.S. Edmonds, Jr. at the Harvard Psychological Laboratory in the summer of 1951. Valuable advice and encouragement was provided by Dr. G. A. Miller, formerly of the Harvard Psychoacoustic Laboratory.

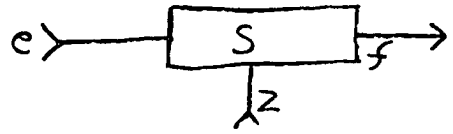
The research was done under contract AF33(038)14343 with the United States Air Force.

enclosed in triple parentheses--(((...))). The basic element of the machine is an electronic unit called a "Snarc" ("Stochastic Neural-Analog Reinforcement Cell"). Each Snarc provides a "probabilistic" transmission characteristic for electric pulses, and provides also a means for altering the transmission probability in a manner dependent on the recent behavior of the Snarc. The remainder of the machine is made up of equipment for interconnecting the Snarcs into nets, and for the introduction and display of pulse patterns for the nets. The "synaptic delay" for the snarcs is uniform, but overall time-quantization is not a property of the machine. Additional apparatus could easily be added to provide for variable synaptic delays and special types of junctions.

Each "Snarc" corresponds to a dis-junction (with any number of input fibres permitted). To obtain conjunctions, or junctions with a numerical threshold, a simple resistance net is attached to the input of a snarc. [Each snarc has a refractory period so that, in principle, any response function can be obtained.] (((In the machine as constructed, the refractory period is very short, and to obtain dependable non-monotone operation it is necessary to add special circuits.)))

4/5.2.1 Description of the individual Snarc.

Each Snarc may be regarded as a three terminal active network element.



In addition, the snarc has two internal state functions, X and K.

X(t) is the transmission probability of the snarc at time t. If a pulse enters on fibre e at time t, then, with probability X(t), it emerges at the fibre f at time t + s (where s is the standard synaptic delay). (((In the actual machine, it is not the original pulse which appears at f; each Snarc regenerates the pulse, just as in interneural transmission. Thus the pulses never lose their original form, no matter how many times they "cross" junctions.)))

K(t) is the internal record of pulse history which must be contained in every "choice element" of a local reinforcement system. In the Snarc it has the following form. Let |t| be the time of the latest firing of f up to time t. (If f fires at t, |t| = t) There is a fixed constant M (representing the "memory span" of the Snarc) and K(t) has the values

$$K(t) = 1 - \left(\frac{t - |t|}{M}\right) \qquad \text{IF } (t - |t|) < M$$

$$= 0 \qquad (t - |t|) \geq M$$

(((Note: it is possible to set the SNARC so that the internal record is of the form

$K^*(t) = 1$ if $(t - |t|) < M$

$= 0$ if $(t - |t|) \geq M$. Furthermore, the value of M is easily adjustable over a wide range.)))

The reinforcement channel Z operates as follows. There are two kinds of reinforcement pulses, z+ and z-. There is an "increment function" I(X) which is positive and can be adjusted by a constant multiplier. (((The actual values of I(X) are determined by a rather complex circuit, and are not as adjustable as might be desired.))) If a z+ pulse enters on the Z fibre at time t, there is a change in the value of X(t) which persists until the next reinforcement. This change has the form:

$$X(t+) = X(t-) + K(t) \cdot I(X(t-))$$

Note: I(X) has the property that $X + I(X)$ is less than 1 and $X - I(X)$ greater than zero, so that X is always between 0 and 1.

On the other hand, if a z- pulse enters at time t, X is changed so that

$$X(t+) = X(t-) - K(t) \cdot I(X(t-)).$$

(((Note, if the function K* of the note above is used, then the reinforcement operator has the form

for z+(t): $X(t+) = X(t-) + I(X(t-))$

z-(t): $= X(t-) - I(X(t-))$ if $(t-|t|) \leq M$

$= X(t-)$ if $(t-|t|) > M$. Here

reinforcement is "all or none" depending on whether or not the snarc has fired within the previous M moments.)))

4/5.2.2 In the machine, the z channels of all the Snarcs are (usually) connected in parallel. Hence the effect of a pulse of either z+ or z- type is that of a local reinforcement operator (as defined in 4/5.1). The machine can be connected so that z is operated manually, or automatically, on occurrence of some event in the net. In either case, z will be controlled by some kind of V-system. The remarks of 4/2-4/4 will apply to the machine to exactly the extent to which this local reinforcement operator acts like a global one.

4/5.2.3 Technical Note.

(((The "probability $K(t)$ of a pulse passing through a Snarc is determined by the instantaneous phase of a multivibrator which gates the input to the Snarc. The value of $K(t)$ is determined by the setting of a potentiometer which fixes the duty cycle of this multivibrator. Several different techniques are employed to insure the incoherency of the multivibrators of the various Snarcs, among these are selection of different average frequencies for the different multivibrators, and changes in the mean frequencies over short intervals. In addition, the phase of each multivibrator is subjected to disturbances

of random magnitude and at random times; the signals for this being obtained from a thermionic white noise generator. Elaborate precautions are taken to minimize coupling between the oscillators. Although no rigorous statistical checks were made on the incoherency, pairs of multivibrators were observed over long intervals, while set at the same mean frequency, and no coupling could be noted.)) ((The reinforcement operator, in the physical machine, consists of the rotation of a countershaft to which the A-potentiometers are coupled through magnetic clutches. The internal memory unit K determines the time through which the magnetic clutch for any particular snarc is engaged. The standard increment I is a function (determined by the circuit associated with the X potentiometer) which depends on the total rotation of the reinforcement countershaft during any reinforcement. The duration of a pulse and its refractory period is of the order of a few milliseconds, making the net operate at realistic (neural) speeds. The reinforcement process, however, being partly mechanical (in the physical sense!), operates over intervals of the order of .5 to 10 seconds, and the internal memory span M of the snarcs is itself of this order. This is also, probably, not an unrealistic figure for simple organisms.)))

4/5.2.4 The SNARC machine was tested in a series of maze-running problems in the following way. First a "random" net of snarcs was assembled on an input-output plug board. Vertices of the maze were represented by distinct stimuli (simultaneous input volleys). Each of several possible responses were interpreted as a decision in favor of some transition to another vertex of the maze. Other responses were interpreted as "no decision" (or "hesitation"). Thus only certain transitions were permitted. The complexity of the mazes was up to ten vertices, and up to four allowed transitions from each vertex. Apparatus was constructed for automatic sequences of the maze-running problem.

Then certain vertices were distinguished as being "bad" ($V = -1$), others as "good" ($V = +1$), and the remainder neutral ($V = 0$). The Δ operator was so connected that when the "rat" arrived at a distinguished vertex, the appropriate z+,z- or no z-pulse was applied. The "training" could also be done manually.

In most cases, the Local Reinforcement Operator of this machine did in fact have the effects of a global operator, and the learning properties were evident to the observer. For certain mazes and nets, the system was unable to accomplish the assigned task because of special combinatorial obstacles. In general, however, the machine displayed gratifying ability. The assigned

goal (the set of "good" vertices; in most cases we used only one such) could be changed during the operation of the machine; in such cases the system displayed an appropriate period of confusion before "learning" the new task. Perhaps most impressive was the fact that at any time during operation, the random net of Snarcs could be rearranged, wires pulled out, tubes removed, and even fuses blown, and yet, if not too many snarcs were inactivated, the machine would, again after a period of confusion, adapt to the new situation and find paths to the preferred vertices. In most cases, the initial setting of the transmission probability of the snarcs had no apparent effect on the asymptotic distributions (if there were such); to determine whether this holds in general will be a subject for later investigation. It certainly is not true for more general nets.

4/6 Local vs. Global reinforcement

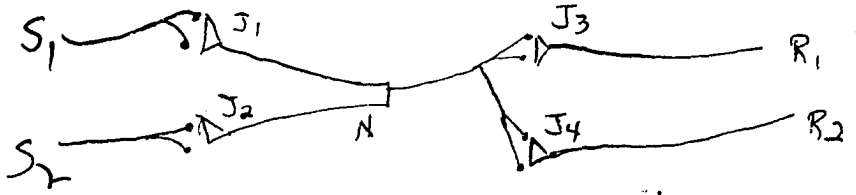
The question of when a local reinforcement operator is equivalent to a global one is a very difficult one. I have made some progress in this direction through the medium of a theory of "selection" or "evolutionary" processes. It was originally intended to include this theory as an additional chapter between the present chaps. 4 and 5, and the decision to omit it was made primarily for reasons of compactness. This theory has been worked out in considerable detail and will be the

subject of a later publication. The theory of selection processes is concerned with the behavior of distributions of points moving in oriented graphs with transition probabilities which depend in a certain specified manner on the past trajectory of the points. I.e., the theory is concerned with a certain class of stochastic processes. The theory seems applicable to the reinforcement problem because both local and global reinforcement have similar representations in this system. For global reinforcement, the points of the base graph represent states of F (see 4/2ff) and the transition probabilities are those of the reactions e_i-f_j. For local reinforcement, however, the graph is more like a phase space, in that the vertices represent the internal states of the whole net, and the transitions are the changes in this total internal state consequent to the application of the local reinforcement operator after different reactions of the net.

Although an adequate theory of the behavior of local reinforcement systems has yet to be developed, some more or less general remarks can be made about kinds of behavior that may be expected from such systems. The following examples illustrate a few features that seem to be important (at least in contrast to what happens in the simpler global systems).

4/6.1 Interference with previous training

The following simple net will occur again and again in any complicated net.



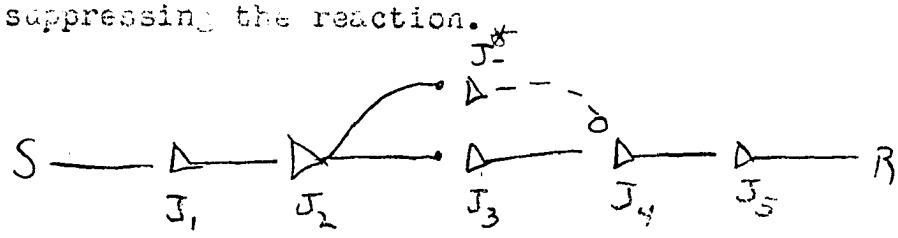
Suppose that an extremely simple LRO is available which when applied to a junction which has recently fired raises its XP from a "resting" value H_0 to a higher value H_1. Now suppose that it is desired to train the response $J_1:R_1$. We need only wait until this response occurs and then apply L+. Then the XP of J_1 and J_3 will be raised from H_0 to H_1. In the future, if S_1 is fired, the response R_1 will occur with reliability $H_1^2(1-H_0)$. (N is a dis-junction with XP = 1.0sec 2/3.2) The response $S_2:R_2$ will have probability $H_0^2(1-H_1)$; if we assume that H_0 is small and H_1 is close to unity, this will be small. Now if it is further desired to train the reaction $S_2:R_2$, and this is done by waiting for an occurrence of that reaction and then applying L+, then all four junctions will have XP = H_1. Thus the reliability of the first reaction $S_1:R_1$ will be reduced from $H_1^2(1-H_0)$ to $H_1^2(1-H_1)$ which is much lower. The second reaction has the same low reliability. This is a simple example of the type of interference of new learning with previous learning that will be found in LRO systems.

4/6.2 "Unlearning"

Consider a situation in which a net has learned (or started out with) a reaction S:R and that it is desired to remove this reaction. If a Σ- operator is available, it is simple enough to await the occurrence of the reaction and then apply Σ-; this will reduce the XP of the junctions concerned in the occurrence of the reaction and, in general, will reduce its probability. (If the undesired response is no response at all, this method will not work, of course.)

However, if the system is not equipped with a Σ- operator (and the important systems of chapter 6, which are proposed as a brain model are not so equipped), then the above routine cannot be used. The only alternative, then, is to wait until an application of S does not cause the occurrence of R, and then apply Σ+. (In chapter 6 a third course, related to "extinction," is indicated in the discussion of 6/1/4 (D3) and 6/1/5.3.) Such a procedure may or may not be successful; if the failure of R to occur is due merely to a failure of one of the links in the establishment of the response, then application of Σ+, is unlikely to depress the future probability of the reaction. (In fact, it will tend to increase it, by raising the XP of those junctions that do fire.) However, if the failure of S:R to occur is due to the inclusion of an inhibition or similar type

of interference, the reinforcement may be successful in suppressing the reaction.



Imagine that the undesired reaction involves the chain $J_1 \ldots J_5$. Then the non-occurrence of the reaction might be due to the firing of a junction (inhibitor, or one which alters the timing of some part of the mechanism for the reaction) like J*. Then reinforcement of the system will reinforce J, and the suppression could become reliable. This mechanism is particularly applicable in the theory of chaps 5 and 6 where the timing of pulses is important and in which extra pulses and paths have a high capacity to suppress other organized activity.

It is important to note that there is a great difference between this process (of reinforcing non-occurrences of undesired responses) and that of applying a Z- operator (to occurrences of an undesired response). In the positive case, along with reinforcement of the "inhibitor" link goes positive reinforcement of these parts of the reaction which still occur. The process thus ought to be called, in contrast to "suppression," "Repression." For if, at a later time, the inhibitor path fails for any reason, the undesired reaction will emerge and with possibly greater reliability than it had before the "repression" was applied.

Further remarks on the local-global problem will occur throughout chapter 6, but no systematic theory is attempted.

CHAPTER 5 CYCLES IN RANDOM NETS

CHAPTER 5 CYCLES IN RANDOM NETS

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5/1 Simple refractory junctions

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CHAPTER 5.

CYCLES IN RANDOM NETS

5/0.0 The model reinforcement systems of chapter 4 each involve operators on the transmission properties of nets. None of these operators have a form which can be realized, in a natural way, by a biologically plausible neural-analog network. The present chapter contains the foundation for a more natural reinforcement theory for nerve nets. In this theory, the "functional elements" of the net will be "circuits"; i.e., subnets which contain a closed "cycle" of junctions, and thus are capable of maintaining sustained activity after cessation of outside stimulation. In the present chapter, attention will be concentrated on the stability and the interaction of such circuits in random nets. The reinforcement theory itself will be presented in chapter 6.

5/0.1 In the first six sections of this chapter, <u>isolated</u> cycles will be examined under various synaptic postulate systems. Following this, we will turn to the examination of cycles embedded in nets. Each synaptic postulate will be a specialization of the general recovery sequence (3/2.3). For each synaptic postulate we will examine the response of simple chains and cycles of length n to a single stimulus pulse, and to stimulation by sequences of

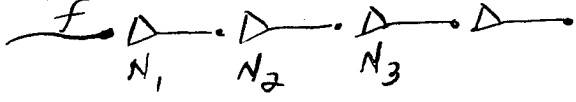
pulses. Discussion of the role of multiple junctions is deferred until section 5/9ff.

5/1.0 We first consider junctions for which

$XP(d) = 0 \quad (d \leq r)$
$= p \quad (d > r)$

r is the length of the "refractory period"; the XP of any junction is p except when the junction is in the refractory phase.

5/1.1 Simple chains Cells: N_i, i = 1,2,... . Connections: $N_i \rightarrow N_i + 1$.



Let a single pulse, f(0), enter on the input fibre f.

Then, $prob(N_1(1)) = p$:

and, $prob(N_t(t)) = p^t$

The probability that N_k is the last cell to fire, i.e., that the pulse dies out at cell N_{k+1}, is $p^k q$. Then the mean distance the pulse will travel, or the expected number of junctions that will fire, is

$$M = \sum_{i=1}^{\infty} i(p^i q) = p/q = p/1-p.$$

In the case that the chain is not infinite, but has just n junctions, the expected number that will fire is

$$M_n = \sum_{1}^{n-1} ip^i q + np^n = \frac{p}{q}(1 - p^n).$$

5.1.2 Repeated excitation of a simple chain.

Let the input fibre f be fired, with probability a,

at each moment t. Each firing of N_1 must be followed by a quiet period of duration at least r. After r moments of non-firing, N_1 will fire with probability p when stimulated (the latter event having probability a at each moment). Hence the mean time T between discharges of N_1 is

$$(r+1)ap + (r+2)ap\beta + \ldots + (r+i)ap\beta^{i-1} \ldots = T$$

where $\beta = (1 - ap)$, and β^{i-1} is the probability that N_1 does not fire in the preceding $i-1$ moments.

Then $T = rap \sum_{i=0}^{\infty} 1 + ap \sum_{i=1}^{\infty} i\beta^{i-1}$

$$= \frac{rap}{1-\beta} + \frac{rap}{r(1-\beta)^2} = r + \frac{1}{ap}.$$ This is just r, plus the mean spacing that would occur if there were no refractory period. After N_1, the pulses are certain to have a minimum spacing greater than r, so that the refractory period has no further effect. The mean spacing of pulses at N_k is, therefore

$$p^{-k+1}(r + 1/ap).$$

Each pulse travels a mean of

$$\left(\frac{1}{rap+1}\right)\left[apq + 2ap^2q + \ldots + jap^jq + \ldots\right] = \frac{ap}{q(rap+1)}$$

junctions. If $r = 0$, this is ap/q, as would be expected from 5/1.1, since then pulses travel independently. If $a = 1$, i.e., if stimulation is regular, then the frequency of firing at N_k is $p^k/(1+rp)$. If r is 0, this is just p^k.

In general nets, of course, it is not true that the

refractory period plays no part after the input cells, since pulses may arrive by different routes to an inner cell, at intervals smaller than r+1. (See 5/7.2.5)

5/1.3 Cycles of circumference n.

If $r \geq n$, then no pulse can travel further than n junctions, and continue to circulate around the cycle. Then the analysis is the same as for the chain of length n.

If $r < n$, then for a single pulse the situation is the same as for an infinite chain, and the pulse will persist a mean of p/q moments, or $\frac{1}{n} \cdot \frac{p}{q}$ "cycles." The cycle can maintain up to $\left[\frac{n}{r+1}\right]$ circulating pulses; each time a new pulse is entered, any pulse that follows within r units will be eliminated when it reaches the junction at which the new pulse entered, since that junction will then be refractory.

5/2.0 Now consider junctions with a supernormally excitable phase in the recovery cycle. The simplest case is

$$XP(d) \begin{cases} = 0 & (d = 1) \\ = 1 & (d = 2) \\ = p & (d \geq 3) \end{cases}$$

or more generally $XP(d) = 0 \quad (d \leq r)$

$= F \quad (r < d \leq s)$

$= p \quad (s \leq d)$

"absolute" facilitation

1. For a single pulse in a simple chain, the behavior is exactly that in 5/1, since each junction has XP = p when the pulse reaches it.

2. If the fibre f (5/1.1) is fired at each moment t = 1, the situation is quite different. N_1 will have XP = 1 until it is fired; thus it will first fire at mean time 1/p. Let t_i be the time of the i^{th} firing of N_i. N_1 will fire first at t_1; it then must fire at all times $t_1 + 2i$ ($i = 1,2,\ldots$). For XP = 1 at each of these times.

It follows that N_2 will be stimulated at all times t_1+2i+1, and after it first fires (at t_2) it must continue to fire at alternate times thereafter. Since N_{i+1} is stimulated at all odd intervals after the first firing of N_i, the mean time $\overline{t_{i+1} - t_i}$ is

$= p + 3pq + 5p\,q^2 + \ldots = (2-p)/p = (2/p) - 1$, which does not depend on i.

It follows that so long as stimulation of N_1 continues, the activity advances down the chain at a (mean) rate of p/(2-p) junctions per unit time. The activity may be visualized as follows: Each pulse of N_1 travels down the chain along junctions which have been left in the facilitated phase by the preceding pulse. This continues until the pulse reaches the junction at which the preceding pulse was lost. From this point on, the given pulse meets ("fresh") junctions with the

normal XP = p. It traverses an average of p/q of these junctions before disappearing.

Except for the transient period (of duration averaging 1/p) before N_1 first fires, a new pulse enters the chain at every other moment. Hence if regular stimulation continues for a period T, then a mean of slightly less than T/2 (almost exactly $\left[\frac{1}{2} + \frac{(T-1/p)}{2}\right]$) pulses enter the chain, and since each pulse ultimately advances the activity p/q junctions along the chain, the activity travels a mean of about $\frac{T}{2} \cdot \frac{p}{q}$ junctions before it ceases. The activity does <u>not</u> cease as soon as stimulation ceases; if at the time of the last input pulse the chain contains K pulses, these pulses will be lost at the rate of R= 1 - p/(2-p) per moment, and activity will persist for a mean of K/R moments.

3. If a cycle, of circumference n, is composed of such junctions, there are two kinds of behavior that can occur, depending on whether n is odd or even. (This is a combinatorial accident, and should not be regarded as holding in more general situations.)

If the number n of cells is odd, and N_1 is stimulated at each moment, then any pulse originating from N_1, will, if it gets as far as N_n, excite N_1 when N_1 is refractory. Thus no pulse can continue to circulate around the cycle, while regular excitation continues.

On the other hand, (with n still odd) when excitation is removed, activity will eventually die out. For;

i. The number of pulses cannot increase after stimulation ceases.

ii. If at any time t_o there are more than $(n-1)/2$ pulses, then at t_o+1 there will be more than $(n-1)/2$ refractory cells, and hence less than $(n-1)/2$ pulses. By i, then there will remain less than $(n-1)/2$ pulses.

iii. Hence, there cannot at any $t>t_o$ be more than $(n-1)/2$ cells firing, or the same number in the facilitated state. Hence there must always be at least one cell in the p-state. Such a cell will remain in that state until it is fired, hence if there are any pulses left in the cycle, at least one of them must meet a junction in the p-phase in each n moments. So pulses will be lost from the cycle at a mean of at least q/n pulses per unit time and all pulses will eventually vanish.

If n is even, then once alternate positions are filled, each pulse will always meet junctions for which $X^p = 1$, and activity will never cease. However, if any pulse is removed, the rest will drop out at a finite rate, because pulses will then meet non-facilitated junctions.

5/3.0 More interesting is the case of non-absolute facilitation, where the M of the facilitated junctions is raised, but not up to unity. It will simplify calculations, without greatly altering the picture, to assume no refractory period. (presence of a refractory period would only add a condition on minimum spacing of pulses.) Assume then, that

$$M(d) = F \quad d \leq s$$
$$\text{if}$$
$$= p \quad d > s$$

For further simplicity, let $s = 1$. Then a junction J is facilitated at time t and only if it fired at time t-1.

We will assume, of course, that $F > p$. and that F is less than unity. Let $q = 1-p$. Also let $G = 1-F$.

5/3.1 Definition:

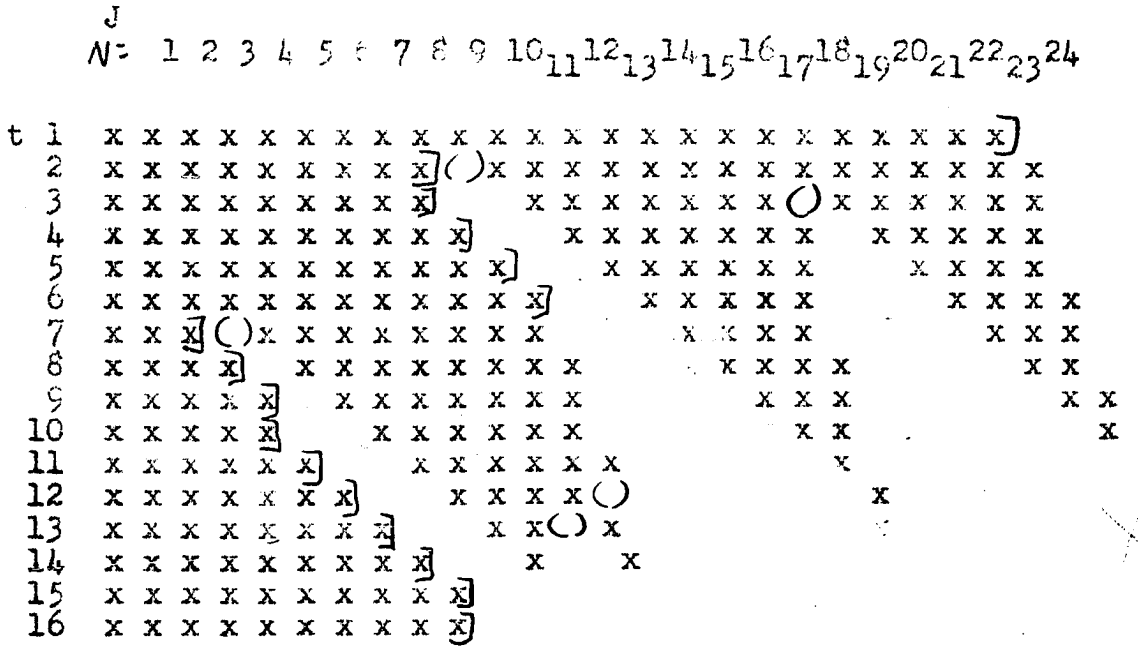
The initial segment $E(t)$, at time t, of the chain is the segment of the chain from f (the fibre at the left) and up to but not including the first junction which does not fire at time t. (We assume that f fires at each moment.)

If at time t_o, the initial segment has length n_o, we will write $E(t_o) = n_o$. We will also say that the chain is in state En_o at time t_o.

If $E(t_o) = n_o$, then at time t_o+1 there are several possibilities. N_{n_o} 1 may fire (probability p), also

N_{n_o+1} N_{n_o-1}

one or more of the N_i ($i \leq n_o$) may fail to fire. In the latter event, if $i < n_o$, the initial segment is said to be "broken," by an "F-failure." (For if an earlier junction failed to fire, it must have failed to fire when stimulated in the /F/ phase. If F is very close to unity, this will be an event special enough to deserve a name.) The following diagram illustrates what happens when the initial segment is broken by an F-failure. (The diagram was obtained with the use of a coin and a table of random numbers, and is based on the values p = 0.5, F = 0.95.)



It is clear that the initial segment tends to grow at the rate p when there are no gaps. Once a gap develops, the segment following the gap is destroyed at a rate of at least (1-p) = q. [Since the application will be to the excitation of cycles, and a cycle excited with a gap will die out at a relatively rapid rate, only the initial segment will be important.]

5/3.2 The states E_n, which are the lengths of the initial segment, form the states of a Markov chain. The system is irreducible since there is a positive probability of going from any E_n to E_{n+1}. (this probability is just $F^n p$) hence all states are in the same component. It is aperiodic since, e.g., there are passages from any state E_m to any other state E_n having both odd and even numbers of steps. This is true because there is a positive probability of remaining in a given state either an odd or an even number of times. Finally all states are non-null recurrent (Feller, p. 322), for state E_0 is recurrent, and in fact occurs every time J_1 fails to fire, an event of probability not less than min(q,G). Since the state E_0 is non-null recurrent, and the system is an irreducible Markov chain, all states have this property. Hence the system is ergodic.

5/3.3 The transition matrix.

Let P_{mn} be the probability of transition from E_m to E_n. Then If $n > m+1$, $p_{mn} = 0$. $P_{mm} = F^m q$. If $r < m$, then

P_{mr} is F^{m}_{r}. These relations hold for m,n,r $= 0,1,\ldots$.

Also, $P_{m,m+1} = F^{m}p$. To summarize,

$P_{m,m+i} = 0$ if $i>1$.
$P_{m,m+1} = F^{m}p$
$P_{m,m} = F^{m}q$
$P_{m,r} = F^{r}G$ if $r<m$

The matrix of transitions is

$$P_{ij}\begin{array}{c} \\ 0 \\ 1 \\ 2 \\ 3 \\ \\ m-1 \\ m \\ m+1 \end{array}\begin{array}{c} \begin{array}{cccc|cccc} 0 & 1 & 2 & 3 & & m-1 & m & m+1 & m+2 \end{array} \\ \left(\begin{array}{cccc|cccc} q & f & 0 & 0 & & 0 & 0 & 0 & 0 \\ Gq & Fp & 0 & 0 & & 0 & 0 & 0 & 0 \\ FGq & F^2p & 0 & 0 & & 0 & 0 & 0 & 0 \\ F^2Gq & F^3p & 0 & 0 & & 0 & 0 & 0 & 0 \\ \hline F^3Gq & F^4p & 0 & 0 & & 0 & 0 & 0 & 0 \\ \vdots & \vdots & \vdots & \vdots & & \vdots & \vdots & \vdots & \vdots \\ F^{m-1}Gq & F^{m}p & 0 & 0 & & 0 & 0 & 0 & 0 \\ F^{m}Gq & F^{m+1}p & 0 & 0 & & 0 & 0 & 0 & 0 \\ F^{m+1}Gq & F^{m+2}p & 0 & 0 & & 0 & 0 & 0 & 0 \end{array}\right) \end{array}$$

The system has an asymptotic distribution; let $m_k =$ prob of being in E_k. The m_k satisfy the linear system

$$m_k = \sum_j m_j p_{jk} \qquad \left(m_k > 0,\ \sum_k m_k = 1.\right)$$

These may be written as

$$m_0 = m_0 q + Gq\sum_{j=1}^{\infty} m_j$$

$$m_k = m_{k-1}F^{k-1}p + m_k F^k q + F^k G\sum_{j=k+1}^{\infty} m_j$$

or $$m_k = m_{k-1}F^{k-1}p + m_k F^k q + F^k G - \sum_{0}^{k} m_j$$

These can be solved successively for $m_0, m_1, \ldots$ but this is very tedious. $m_0 = G/(p+G.)$, $m_1 = m_0\dfrac{p(1+p)}{1+F(G-q)}$

5/3.4 For our purposes, it is important to know, at least approximately, the distribution of the <u>time of the first passage from E_0 to E_n</u>. As will be explained in detail in section 5/6 and ff, it may be assumed, in the applications to be made of the calculation, that F is equal to unity. If this assumption is made, the matrix becomes simply

$$\begin{pmatrix} q & p & 0 & 0 & 0 & 0 & 0 \cdots \\ 0 & q & p & 0 & 0 & 0 & 0 \cdots \\ 0 & 0 & q & p & 0 & 0 & 0 \cdots \\ 0 & 0 & 0 & q & p & 0 & 0 \cdots \\ 0 & 0 & 0 & 0 & q & p & 0 \cdots \\ \cdots & \cdots & \cdots & \cdots & \cdots & \cdots & \cdots \end{pmatrix}$$

The probability that the system first reaches state E_n at the time of the m'th pulse, is the probability that in Bernouilli trials with probability p of success, m-1 failures will be recorded before n successes. For let each unit advance along the chain represents a success. Each failure means that new advances must await a new pulse. Hence m pulses will be required in all, if there are m-1 failures.

The probability of 0 failures before n successes is, of course, p^n. The probability of m-1 failures is

$$p^{n-1}q^{m-1}\binom{n-1+m-1}{m-1}.p.$$ (For the last trial must be a success (by definition), and the earlier n-1

successes and m^{-1} failures may occur in any order.) This is the Pascal distribution; it has mean $\frac{nq}{p}$, which is thus the mean time of the first transit to E_n. The same result may be inferred from 5/1.1; each new pulse meets a chain of junctions in the /p/ phase, and advances a mean of p/q junctions. Hence it may be expected that it would take a mean of nq/p pulses to advance n junctions. (In order that the approximation be meaningful, it must be assumed that F^{n^2} or better, $(F)^{n^2q/2p}$ is close to unity. This would imply that the chance of F-failure, or the chance of transition to an earlier state, is negligible.

5/3.5 The application to cycles is as follows. Let C be a closed chain of n junctions. Let one of the junctions J_0 be stimulated at each moment. How long will it be before the cycle has all junctions firing in the /F/-state? This is $\leq$ the time that it would take for the initial segment to reach the n'th junction in an infinite chain, and the distribution of this was computed in 5/3.4. The mean time of activation of the cycle is, then $\leq nq/p$.

Once such a cycle has been filled with pulses, it will continue to fire until an F-failure occurs. The average time until the first F-failure is easily seen to be (noting that n junctions must fire at each

moment) $T_f = F^n/(1-F^n)$. Under the assumption that (as above) F^{n^2} is close to unity, nG will be small, and expansion of T_f in terms of G yields $T_f \sim \frac{1}{nG} - 1$, providing an estimate of the time the cycle will continue to operate after stimulation ceases.

5/4 F-stability and pulse distributions in cycles.

For the remainder of this study, attention will be concentrated on forms of activity which have appreciable probabilities of persisting for more than a very few moments. In particular, we emphasize the notion of an "F-stable" activity pattern.

Definition: A pattern P of activity in a net is said to be "F-stable" or "F-active" if it is such that until the occurrence of an F-failure in the pattern, each cell of the pattern is in the /F/ phase at each time of stimulation.

(Note: A "pattern" is a distinguished set of pulses and cells. The pulses of an F-stable pattern may meet cells which are outside of the distinguished set of cells; these outer cells need not be in any particular state when stimulated.)

Any F-stable pattern has a definite "half-life" which depends only on the number of pulses in the pattern, in the case that this number is a constant. If m is this number of pulses, then m junctions must

fire at each moment, and the half-life is the L for which $F^{mL} = 1/2$, i.e., $L = \frac{-\ln(2)}{m \ln(F)}$. In the case that the number of pulses is not constant, there is no such simple expression. If time-quantization is invoked, however, then any finite F-stable pattern must be periodic, and the half-life will not be difficult to compute for any particular case.

We will first partially classify patterns of pulses in simple cycles, and establish some theorems which are important in the present theory. The results will then be applied informally to more complex nets, and to the classification of activity in general random nets. It will be seen that while exact calculation is feasible for simple cycles, rigorous extension of the theorems to more general nets would be difficult because of combinatorial difficulties. Qualitative, and topological, arguments will be necessary.

5/4.1 Patterns in cycles of length N.

We now assume that all cells have the same properties, and that they each follow the full recovery sequence of chapter 3. Thus we will have to consider phases /0/;/p_1/;/F/;/p_2/;/p/. However, as we shall concentrate on F-stable patterns, the classification will be simplified and not essentially changed if we set $p_2 = p$. We ask what are the possible patterns in a cycle of

length N, and which of them are F-stable.

a). N e /O/. In this case there can be no cyclic activity at all, since no pulse can complete a circuit.

b). N e /p_1/. This cycle can support at least one circulating pulse, and possibly more. But each pulse must, within N moments of its initiation, continue to meet junctions in either /O/ or /p_1/ phase. Then there are no F-stable possibilities.

c). N e /F/. In this cycle, a single pulse will become F-stable at the time it completes its first circuit. If also $\left[\frac{N}{K}\right]$ is in /F/ then there will be patterns containing 1,2, ... ,K pulses, each of which will be F-stable. And for a given number of pulses, it is possible that there be more than one F-stable configuration, even after identification of time-translated patterns.

d). N e /p_2/ or /p/. Then no pattern with a single pulse can be F-stable but there may be several F-stable patterns with more than one pulse.

Actually, the variety of F-active patterns depends, obviously, on the location and width of the /F/-phase. The classification of F-active patterns can be based on the properties of the interval /F/.

1. If /F/ contains a single integer k, then a cycle

can be F-active if and only if $N/k = x$, with x an integer, and there are x equally spaced pulses in the cycle of length N.

2. If /F/ contains several integers

$$k_0,\ k_1 = k_0 + 1, \ldots, k_m = k_0 + m,$$

there may be many distinct F-active patterns. For to each ordered partition of N into a sum of the k_i's, there is an F-active pattern. For if

$$N = k_{i1} + k_{i2} + \ldots + k_{is},$$

then the pattern of pulses formed by firing the junctions

$$J_{k_{i_1}},\ J_{k_{i_1}+k_{i_2}},\ \ldots,\ J_{k_{i_1}+k_{i_2}+\ldots+k_{i_s}} = J_N$$

is F-active (or will be, once all junctions are fired by the rotating figure).

For example, let N = 10, and let /F/ contain 2 and 3. Then (J_{10}, J_2, J_4, J_7) and (J_n, J_2, J_5, J_8) are distinct F-active patterns. Except for rotation, these are the only such.

3. Exact computation of the number of rotationally distinct partitions of N into integers contained in a given interval would be laborious and (for our applications) uninformative. It is important, however, to have sufficient conditions that a cycle can maintain at least one F-stable pattern:

Theorem: If /F/ contains more than one integer, say s and s+1, then for all $N \geq s^2-1$ there is at least one F-stable state.

Proof: If $N \geq s^2-1$ then N can be written in the form $ps + q(s+1)$ with p and q integers. This is trivial if $N = s^2 = ss$ or if $N = s^2-1 = (s-1)(s+1)$. Otherwise, N can be written (in the s-ary number system) as -

$$N = a_0 + a_1 s + Rs^2 \quad \text{with } a_0, a_1, R \geq 1$$

$$= a_0 + a_1 s + s^2 + s^2(R-1)$$

$$= a_0(s+1) + (s - a_0)s + a_1 s + s^2(R-1),$$

which has the desired form. Thus there is a partition of the type discussed in 2) above, and therefore there is one or more possible F-stable patterns.

5/4.2 A note on the role of time-quantization.

The above classifications and arguments are based explicitly on the postulate of precise time-quantization. If this postulate does not hold, then the conditions for the existence of F-stable patterns will not exhibit such a clear arithmetic quality. Now the classification of F-stable patterns might seem to have a large importance for this theory for more than two reasons:

1) F-stable patterns will play an important role in net activity.

2) The number of possible distinct F-stable patterns that can be maintained within a given cycle C ought to supply a precise measure of the "amount of information" that can be stored by the activation of C in a particular mode.

For a realistic Neurological model, however, it cannot be assumed that time quantization can hold other than approximately, if that; the synaptic delay itself cannot be expected to be perfectly uniform. Hence we may use only those theorems that do not depend on perfect synaptic uniformity.

5/4.2.1 Suppose first that the synaptic delay of a junction, instead of having exact value unity, has instead a probabilistic distribution with, say, mean unity, and, say, the distribution closely, but with positive dispersion, concentrated around unity. This might seem to yield a first approximation to time-quantization.

Now consider a simple circuit, containing two pulses. Let $N = 2k$ with k in /F/, and let J_N and J_k be fired, both at time $t = 0$. If the synaptic delay were constant, then the two pulses would always remain diametrically opposite (assuming no failure), and the circuit would become and remain F-stable. However, with a non-trivial distribution for the synaptic delay, the two pulses under <u>independent</u> variations from unit velocity, and need not remain diametrically opposite. In fact, with

probability 1, the pulses will slip in phase in the manner of a random walk, and sooner or later one of the two circular displacements will become too small to be contained in /F/. Either circular distance can be regarded as the position of a particle taking a one-dimensional isotropic random walk; it has probability 1 of leaving any bounded region.

If this variation of the synaptic delay is appreciable, then F-stability suffers; an apparently F-stable pattern may drift into a non-F-stable one without any F-failure having occurred.

5/4.2.2 There is, however, another effect which may serve partly to offset the random walk effect. It is known,(Fulton p. 140 ff.) that synaptic delay is appreciably greater (at least for spinal motoneurons) when stimulation is earlier in the recovery cycle. Let us assume this to be the case, at least in those regions of the recovery cycle concerned here.

We shall therefore investigate the consequences of assuming that the synaptic delay s is a decreasing function of the recovery phase d of each cell.

In the example of 5/4.2.1 it is easily seen that the negative slope effect of s(d) tends to offset any deviation that might arise from a superimposed added random variation of s. For the closer pulse 1 comes to pulse 2, (measuring along the propagation direction), the larger are the synaptic delays that it meets, and equilibrium occurs only at opposite points.

Of course, if the slope of s(d) were more negative than -1, then the correction, in this example, would be greater than the previous error, and the effect might not be stabilization. However, it is a biological fact that the slope is not that far negative, (except possible for extremely small d, in which case the probability of transmission at all is small), i.e., in general, later stimulation yields later firing, although not so much later. (It must be mentioned once more that there is no direct evidence for any particular synaptic properties within the brain; all is inference from the periphery, or the motor cells.)

On the other hand, it would seem that the negative slope (or, in fact any slope at all) of s(d) would tend to destroy the distinction between different F-stable patterns with any given number of pulses, since it would seem that there would be a tendency toward equidistant spacing.

Hence any information stored in the form of the

spacing of k pulses would seem to undergo degradation, and only the number k would be available for long-term storage of information.

To prove this tendency toward regularization for general s(d) functions seems difficult; however it can be shown for a linear approximation to s(d).

Let us suppose, then, that we have simple cycle of n cells, in which s pulses are circulating. Let the pulses be called p_α ($\alpha = 1,\ldots,s$) with p_{a+1} representing the pulse ahead of p_a. Let the cells of the chain be called N_i ($i = 1,\ldots,n$) but allow i to take on arbitrary integer values with the understanding that $N_i = N_j$ if $i \equiv j \bmod(n)$. Similarly, let α take on arbitrary integral values with the understanding that $p_a = p_b$ if $a \equiv b \bmod(s)$.

Finally, let $t_{a,j}$ be the time at which pulse p_a first fires cell N_j (if $j \leq n$ and $a \leq s$). Also, for $a \leq s$, and $j \leq n$, let $t_{a,j+kn}$ be the time at which p_a fires N_j for the $(k+1)$-th time. Finally, for all a and j, let

$$t_{a+s,j+n} = t_{a,j}. \qquad (1)$$

(Note: this notation represents a corresponding process occurring in a helical covering space, but may be regarded as pure formalism.)

Now we can express the effect of a linear s(d) function:

$$t_{a,j+1} - t_{a,j} = K - p(t_{a,j} - t_{a+1,j+1}). \qquad (2)$$

with $0 < p < 1$.

5/4.2.3 Theorem If $0 < p < 1$, then the pulses of the cycle approach a constant velocity and an equal spacing. This limiting figure is independent of the initial conditions, except for phase. The deviations from this equilibrium are, in an appropriate metric, exponentially damped.

Proof of theorem 4/2.3:

The synaptic delay law, (2) can be written, without loss of generality, as

$$t_{a,j+1} - t_{a,j} = D + p\left[t_{a+1,j+1} - t_{a,j} + D(1 - n/s)\right] \quad (3)$$

where

$$D = \frac{K}{1+p(1+\frac{n}{s})}$$

Define $$d_{aj} = t_{aj} + D\left[\frac{an}{s} - j\right] \quad (4)$$

(Note: it will be seen that D is the asymptotic synaptic delay, and that d_{aj} represents the deviation of pulse p_a at time t_{aj} from its position in the limiting pattern.)

Substituting the d's for the t's,

$$t_{a,j+1} - t_{a,j} = d_{a,j+1} - d_{a,j} + D$$

$$t_{a+1,j+1} - t_{a,j} = d_{a+1,j+1} - d_{a,j} + D(1 - \tfrac{n}{s}).$$

Hence

$$d_{a,j+1} - d_{a,j} = p(d_{a+1,j+1} - d_{a,j}). \quad (5)$$

Note also that

$$d_{a+s,j+n} = t_{a+s,j+n} + D\left(\frac{a+s}{s}\ n - j + n\right)$$

$$= t_{a,j} + D\left(\frac{a}{s}n - j\right)$$

$$= d_{a,j} \qquad (6).$$

Now (5) is a system of difference equations which are linear, and which are actually finite in number because of the modular relations, (6). Assume formal solutions for the system:

$$d_{aj} = C_a\ e^{zj} \qquad (7)$$

Then $d_{a+s,j+n} = C_{a+s}\ e^{z(j+n)} = d_{a,j} = C_a\ e^{zj}$.

Hence $C_{a+s} = C_a\ e^{-nz}$. (8)

Because of (8), we need only solve the system for s consecutive values of a. Choose a = 1, . . . ,s. Substituting the formal solutions (7) into the basic equations (5), we have the linear system:

$$C_a\ e^{x(j+1)} - C_a\ e^{zj} = p\left[C_{a+1}\ e^{z(j+1)} - C_a\ e^{zj}\right].$$

Dividing by e^{zj}, we have

$$C_a(e^z - 1) = p(C_{a+1}e^z - C_a). \qquad (a < s)$$

$$C_s(e^z - 1) = p(C_1\ e^{z(1-n)} - C_s). \qquad (a = s) \qquad (9).$$

or

$$C_a(1-p-e^z) + p\ C_{a+1}\ e^x = 0 \text{ and} \qquad (a<s)$$

$$C_s(1-p-e^z) + p\ C_1\ e^{z(1-n)} = 0 \qquad (a=s) \qquad (10)$$

The coefficient matrix of system (10) is

$$\begin{pmatrix} (1-p-e^z) & pe^z & 0 & 0 & \cdot & 0 & 0 \\ 0 & (1-p-e^z) & pe^z & 0 & \cdot & 0 & 0 \\ 0 & 0 & (1-p-e^z) & pe^z & \cdot & 0 & 0 \\ \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot \\ 0 & 0 & 0 & 0 & \cdot & (1-p-e^z) & pe^z \\ pe^z(1-n) & 0 & 0 & 0 & \cdot & 0 & (1-p-e^z) \end{pmatrix}$$

and the determinant must vanish:

$$(1-p-e^z) - (-p)^n e^{z(s-n)} = 0. \qquad (11)$$

Setting $u = 1 - e^z$, (11) becomes

$$(u-p)^s = (-p)^s(1-u)^{s-n}$$

or

$$(1-\frac{u}{p})^s = (1-u)^{s-n} \qquad (12)$$

Now if u were negative, we would have

$$|1-u/p| > 1 \quad \text{and hence} \quad |(1-\tfrac{u}{p})^s| > 1.$$

On the other hand, with the same assumption,

$$|(1-u)^{s-n}| = |\frac{1}{1-u}|^{n-s} \leq 1$$

since n^{-s} is non-negative. Hence the u is non-negative. Then $1-e^z \leq 1$ and it follows that z is non-positive. Hence the exponents of the system include only 0 and negative value.

Every solution will be a linear combination** of

**For repeated roots, the solution may include polyhomiolo multiplied by exponentials, in which case the exponentials will dominate.

the basic solutions represented by the various exponents. Each of the non-zero exponents determines one (or if the root is multiple, a class of) exponentially damped solutions. The zero exponent determines (see (7)) a constant solution C, with all C_a equal.* This component determines the phase of the limiting pattern. For since all but the constant solutions are exponentially damped,

$\lim_{J \to \infty} d_{aj} = C$ where C is the constant solution associated with the zero exponent. But then by eq (4), we have

$$\lim_{j \to \infty} \left(t_{aj} - \left[C + D \left(j - \frac{an}{s} \right) \right] \right) = 0$$

This means that the firing pattern approaches a pattern which

i. rotates at the rate of 1/D cells per time unit,

ii. has synaptic delay = D

iii. has equally spaced pulses (because of the $\frac{an}{s}$ term)

iv. and whose absolute phase is determined by a constant C, which is the only parameter of the pattern which depends on the initial conditions.

5/4.2.4 I wish to add the conjecture that essentially the same result will hold when s(d) is a function which has slope between and bounded away from 1 and zero.

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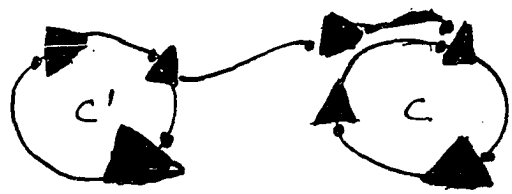
*Differentiation d(12) shows that the o-root is not multiple.

It is worth noting that the tendency toward equalization of pulse spacing which results from a moderate negative slope of s(d) will not tend to destroy the F-stability of any pattern which would be F-stable with the constant synaptic delay. For the limiting (equal) spacing is somewhere between the extremes of the original spacings; since these must be contained in /F/, and since /F/ is an interval, the limiting pattern will be also F-stable.

5/5. Excitation of F-stable patterns. In section 5/3.6 the length of time necessary to establish F-stability in a cycle was estimated for a simple case. It was concluded that for a cycle of length n, [illegible] the mean number of pulses required. $b \pm nq/p$ The same result with the same argument holds in the general case (in 5/3.6 it was assumed that stimulation occurred at each moment). It must be noted, however, that nq/p is the mean required number of pulses; it is not the time required if excitation is not continuous.

5/5.1 Suppose (in spite of the arguments of 5/4) that a cycle C is capable of maintaining two or more distinct F-stable patterns. How can the cycle be stimulated so as to set up a particular pattern in it? If stimulation of each cell is permitted, one has only to fire each dell at the proper time. If there is only one input

available, arbitrary stable patterns can still be imposed. Suppose that there is another cycle C; also of length n, in which the desired pattern is active. One need only connect a chain from any cell of C' to any cell of C.



If we assume time-quantization, then the set of possible pulse spacings in C', modulo rotation, is in one-one correspondence with set of temporal patterns, modulo time shifts of firing, at any one cell of C'. The temporal pattern will propagate down the connecting chain, and around C (at a rate of p/q junctions per pulse) and will set up the corresponding spatial pattern in C. Thus information stored in the form of pulse distributions in simple cycle can be easily transferred to other cycles.

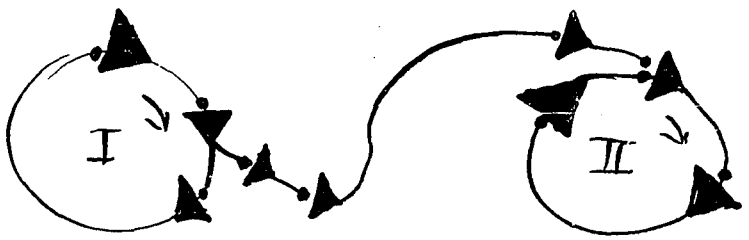
If there is much variation in synaptic delay, the above argument will not be valid, for pulses which have travelled around C may arrive at the input cell of C at times which may conflict with the stimulation at that cell.

If a cycle of length n is excited through a chain of length n as in the figure, the mean time of excitation (until the circuit is P-active) is, by the same argument, at most q/p.

5/5.2 Estimation of phase parameters.

As proposed in 5/0.0, circuits are to play a role as functional units in the behavior of nets. The F-active circuits are to have sufficient stability to act as units. As such, one F-active circuit must be able to remain active long enough to initiate another one, either by contact or through a chain. This assumption makes it possible to estimate plausible values of p and F, with the aid of the knowledge of the duration of the facilitation phases. The latter information comes from neurophysiological studies on nerve fibres, and its applicability to junctions, is of course, speculative.

Consider the network below consisting of two circuits of length n coupled by a chain of length n. If circuit I is F-active at t_o, while the rest of the net is inactive, then if circuit I is to be able to initiate F-activity



in circuit II, it must remain active long enough to deliver 2nq/p pulses. If I contains k circulating pulses, it must therefore remain active for $\frac{2nq}{p} \cdot \frac{n}{k}$ time units.

On the other hand circuit I remains active an average

of $1/kG$ time units (5/3.5). If these quantities have the same order of magnitude, then

$$1/kG \sim 2n^2q/kp \quad \text{or} \quad \frac{q}{p} \sim \frac{1}{2n^2}.$$

5/5.3 The basic time unit (the quantization unit) corresponds to the synaptic delay time. Neurologically, this is probably of the order of 0.5 millisecond. For single impulses, the refractory period is probably short; of the order of 1 to 4 milliseconds, and the /F/ phase appearing 1 or two milliseconds later and lasts perhaps 40 milliseconds (C fibres). However, after several pulses at intervals less than 10 ms, the /F/ phase appears to occur later, and for a shorter time, and higher frequencies appear to cause the /F/ phase to vanish. It seems likely, therefore, that circuits can be F-stable only if pulses occur with spacing at least of the order of a few milliseconds, and relating this to the synaptic delay, n/k is at least of the order of 5 or 10.

We can make an independent guess as to the magnitude of F if we assume that if circuits play a role as elements of brain action, they ought to persist for a noticeable fraction of a second, e.g., of the order of 50 milliseconds. The time of survival of a single pulse in a F-stable circuit is $1/Q$ time units, hence $Q = .02$ or so. It would be foolish to attempt to justify such an estimate at the present stage of knowledge.

However, since in the following sections mechanisms will be described which prevent F-circuits in random nets from surviving as long as they would when isolated. Hence, it is probably necessary to assume that Q is even smaller. Even if Q is as small as .001, a cycle of length 10 (with one pulse) has an expectancy of only 100 circuits, or 1/2 second, taking the synaptic delay to be 0.5 ms. Then (7.4) q/p is of the order of 5, hence p is of the order of 0.2. It will be seen later that the velve of p is not critical for this theory.

5/6 Extinction of F-stable patterns.

F-stable circuits are emphasized here because they supply an adequate source for neural events of more than transient duration. Nevertheless, they are not invulnerable; there are a number of ways in which an F-stable pattern may be extinguished.

i. F-failure, or pulse-loss.
(We will return to the question of when loss of a pulse means extinction of all activity.)

ii. Inhibition resulting in pulse less.

iii. Phase shift (as in 5/4.2.1).

iv. Phase shift due to "subliminal neural activity." (If a cycle is part of a larger net, or in a brain, then the recovery sequence of its cells may be influenced by nearby events. If recovery

is retarded, or hastened, the cycle may leave F-stability.)

v. Introduction of extra pulses.

Surprisingly enough, the effect (v.) on F-stability of extra pulses is so powerful that (v) may well be the most prominent source of extinction of cyclic activity. In order to show why this is so, it is necessary to investigate the problem of which patterns can become F-stable, and the probability of this occurring.

5/6.1 Theorem. Let C be a cycle of length n, and let n be the only integer in /F/. Assume that up to time t = 0 the cycle has been in F-stable activity with one circulating pulse, but that at time t = 0, an additional pulse is introduced. Then the probability that the cycle will ever return to F-stability is less than p^2.

Proof. Let $N_0, \ldots N_{k-1}$ be the cells of the cycle C.

Let the original pulse be described as $N_{-k}(t)$, $(t \geq 0)$, where $N_i = N_j$ if $i = j \bmod (n)$.

Let the extra pulse appear at t = 0 at N_s $(0<s<n)$

Then after t = 0, two pulses will circulate (until one or both fail), and they will be represented as

$N_j(j)$ and $N_{j+s}(j)$.

At each time t>0, one of four events may occur:

i. $E_f(t) \equiv N_{t-1}(t-1).\ N_{t+s}(t).\sim N_t(t)$

i.e. The <u>original</u> pulse vanishes at time t.

ii. $E_e(t) \equiv N_{t+s-1}(t-1).\ N_t(t).\sim N_{t+s}(t)$

i.e. The <u>extra</u> pulse vanishes at time t.

iii. $E_o(t) \equiv N_{t-1}(t-1).\ N_{t+s-1}(t-1).\sim N_t(t) \sim N_{t+s}(t)$

i.e. <u>Both</u> pulses vanish at time t.

iv. $E_1(t) \equiv N_t(t).\ N_{t+s}(t)$

i.e. both pulses are present at time t.

Now let $P_x(t) = \text{prob}(E_x(t))$ $(x = e,f,0,1)$.

We wish to compute the probability with which the system returns to F-stability. It may be seen, by inspection, that if neither s or (n-s) is in /0/, and if $p_1 = p$, then

$$\begin{aligned} P_f(t) &= P_1(t-1)\ pQ \\ P_e(t) &= P_1(t-1)\ qP \\ P_o(t) &= P_1(t-1)\ qQ \\ P_1(t) &= P_1(t-1)\ pP \end{aligned} \qquad (1)$$

where

$P = p$ if $(t<s)$

$Q = q$ " "

$P = F$ " $(t \geq s)$

$Q = G = 1-F$ "

(Note. If n-s is in /0/ then the extra pulse could not have appeared at all. If s is in /0/, then the original pulse must vanish at time s. The new pulse must traverse n-s junctions each in phase /p/ in order to

become F-stable. Since (n-s) is not in /0/, then (n-s) is 2 or greater, so the probability that the cycle returns to F-stability is $p^{n-s} \leq p^2$ for this case. We need not consider it further.)

The system cannot become F-stable until after one of the events $E_e(t)$ or $E_f(t)$ has occurred, i.e. until after there is just one pulse in the cycle. We compute the probability of each event E_e and E_f.

$$P_1(0) = 1$$

$$P_1(1) = pF$$

$$P_1(2) = p^2F^2$$

. . . . . .

$$P_1(s-1) = p^{s-1}\, F^{s-1}$$

$$P_1(s) = p^s\, F^{s-1} p$$

. . . . . .

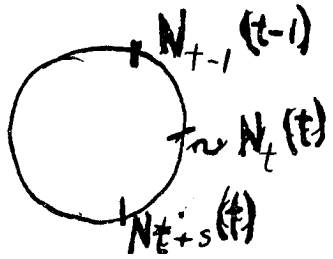
$$P_1(s\,k) = p^{s+1+2n}\, F^{s-1}$$

$$\text{or } P_1(t) = F^t p^t \qquad (t < s)$$

$$= F^{s-1} p^{2t-s}\, 1_{(t \geq s)} \qquad (2)$$

The probabilities $P_e(t)$ and $P_f(t)$ can be easily computed from equations (1), using equations (2).

<u>Let $P^*_f(t)$ be the probability that $P_f(t)$ has occurred, and the system thereafter becomes F-stable.</u>



This means that only the extra pulse is present after time t-1. In order for the

pattern to become F-stable, the pulse must propagate until it meets junctions which were last fired by itself, hence are in /F/ phase. Now

$[N_{t+s+1}, \ldots, N_{t+N-1}]$ were last fired by the original pulse, so the new pulse has probability p^{n-s-1} of reaching N_{t+N}. Also, $N_{t+N+s-1} \equiv N_{t+s-1}$ was certainly last fired by the extra pulse, so it need only return to this cell in order to become F-stable. The phases of the cells in between,

$[N_{t+N}, \ldots, N_{t+N+s-2}]$, or, by definition,

$[N_t, \ldots, N_{t+s-2}]$ depend only on whether or not the extra pulse fired them in the last cycle, i.e. on whether or not

$N_{t+s-N}(t-K)$ occurred. We know that $N_{t+s-K}(t-K) \equiv$ $t \geq N$, since the extra pulse started at $t = 0$.

Hence if $t \geq s$,

$$\begin{aligned} P^*(T) &= p^{N-s-1} && (t \geq s) \\ f &= p^{N-s-1} p^{s-t} && (t < s) \\ P^*_f(t) &= p^{N-t-1} \end{aligned} \qquad (3)$$

If $E_e(t)$ occurs, then only the original pulse is left; it has to reach all N_{t+s+1} in order to become F-stable. A similar argument gives

$$\begin{aligned} (P^*_e(0) &= 1) \\ P^*_e(1) &= p \\ P^*_e(2) &= p^2 \\ &\cdots\cdots \end{aligned}$$

$$P^*_e(s) = p^s$$

. . . . . .

$$P^*_e(s+N) = p^s$$

So

$$P^*e(t) = p^s \quad t \geq s$$

$$= p^t \quad t < s \qquad (4)$$

Now the total probability that F-stability is eventually restored is

$$P_F = \sum_1^\infty P_f(t) \; P^*_f(t) + \sum_1^\infty P_e(t) P^*_e(t) \qquad (5)$$

Or, by (1),

$$P_F = \sum_1^\infty P_1(t-1) \; pQP^*_f(t) + \sum_1^\infty P_1(t-1) qPP^*_e(t). \qquad (5a)$$

where P,Q are defined as in (1).

By (4) and (5), this becomes

$$P_F = \sum_1^{s-1} F^t p^t (pGp^{n-t-1} + qFp^t) + \sum_s^\infty F^{s-1} p^{2t+1-s} (pqp^{N-s-1} + pqp^s)$$

$$= (I) + (II) \qquad (6)$$

$$\text{Now (II)} = \left[F^{s-1} p^{1-s} (pqp^{N-s-1} + pqp^s)\right] \sum_s^\infty p^{2t}$$

$$= \left[F^{s-1} \; . \; . \; . \; . \; . \; . \; . \; . \; . \; . \right] \frac{p^{2s}}{1-p^2}$$

$$= \frac{p}{(1-p)(1+p)} F^{s-1} \left[p^{NH} + p^{2+2s}\right]$$

$$= \frac{p}{1+p} F^{s-1} \left[p^{NH} + p^{2s+1}\right] \qquad (7)$$

And

$$(I) = p^{nG} \sum_1^{s-1} F^t + qF \sum_1^{s-1} (Fp^2)^t$$

$$= p^{n_G} \frac{F-F^2}{1-F} + qF \frac{Fp^2 - (Fp^2)^s}{1-Fp^2}$$

$$= p^n(F-F^s) + qF\frac{Fp^2 - (Fp^2)^s}{1-Fp^2} \qquad (8)$$

Thus, $P_F = (I) + (II)$

$$= p^n(F-F^s) + \frac{q}{1-Fp^2}\cdot F\cdot (Fp^2 - (Fp^2)^s)$$

$$+ F^{s-1} \frac{p}{1+p} (p^{N_H} + p^{2s+1}) \qquad (9)$$

Let $P_{\overline{F=1}}$ be the value of P_F for F=1.

$$P_{\overline{F=1}} \quad 0 + \frac{q}{(1-p)(1+p)}\cdot (p^2-p^{2s})$$

$$+ \frac{p^2}{1+p} (p^N + p^{2s})$$

$$= \frac{p^2}{1+p} (1 + p^N - p^{2s-2}(1-p^2))$$

$$P_{\overline{F=1}} < \frac{p^2}{1+p} (1+p^N) < p^2 \qquad (10)$$

Now F occurs in the expression P_F only to take account of the possibility of an F-failure of the original pulse in the first s-1 moments of time. $P_{\overline{F=1}}$ is the probability of F-stability being vested on the conditional hypothesis that no such failure occurs. Let this, latter hypothesis have probability H_1. The contrary hypothesis, (that such an F-failure does occur) is $H_2 = 1-H_1$.

In the event that an F-failure does occur in the first s-1 moments, the only way in which F-stability

can be restored is if the extra pulse crosses <u>at least</u> n-s junctions in /p/ phase, so the probability of F-stability being restored, on hypothesis H_2, is $\leq p^{n-s}$.

Then $P_F \leq H_1 \cdot P_{F=1} + H_2\, p^{n-s}$.

Since n-s is not in /0/ (see (1)),

$n-s \geq 2$, and

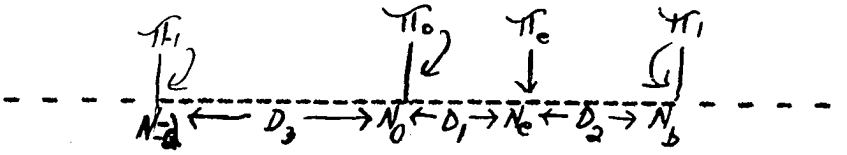
$P_F < H_1 p^2 + H_2 p^2 = p^2$.

Q.E.D.

5/6.2 Theorem 5/6.1 is not sufficiently general for the applications intended. It is restricted in two ways; the cycle contains just one pulse, and /F/ contains a single integer. An exact computation of P_F for the general case would be very tedious, but fortunately an informal argument can establish the desired results. It is to be shown that <u>if /F/ is relatively short compared with the earlier intervals of the recovery sequence</u>, then a <u>random extra pulse</u> will, with high probability, extinguish the activity of the cycle. We assume that $F \neq 1$. See 5/6.2.3.

Consider an arbitrary F-stable cycle with two or more pulses. Suppose that at time $t = 0$ an extra pulse π_e is introduced. Let N_0 be that cell of the cycle which is fired at $t = 0$ by the pulse immediately behind π_e, and index the other cells by the integers modulo the

length of the cycle. Let N_b be the location, at t = 0, of the pulse ahead of π_e, and N_{-a} that of the pulse behind the pulse at N_0. Let N_e be the cell fired by the extra pulse at t = 0. Let $D_1 = e$, $D_2 = b - e$, $D_3 = 0 - (-a) = a$. The pattern at t = 0 then resembles the figure:



(Note: There is no restriction that N_{-a} and N_b be distinct. We do assume that N_0 and N_b are distinct.)

Because, prior to t = 0, the cycle was F-active, we knew that D_3 is in /F/ and

$D_1 + D_2$ is in /F/. There are several cases, depending on the location of N_e and the limits of the interval /F/.

I. $D_1 \in /F/ \;.\; D_2 \in /F/$.

Then the circuit will be F-stable with π_e surviving.

II. $D_1 \in /F/ \;.\; D_2 \notin /F/$.

In this case, π_e will continue to meet junctions not in /F/ until it drops. The remaining pulses are not affected, and the circuit will return to F-stability.

IIIa. $D_1 \notin /F/ \;.\; D_2 \in /F/ \;.\; (D_1 + D_3) \in /F/$.

In this case, π_e will remain, since it meets

cells in /F/-phase. Then π_o must drop, since it will meet junctions not in /F/-phase. But since $D_1 + D_3$ is in /F/, π_1 will continue to meet cells in /F/-phase, and the circuit will remain F-stable.

IIIb. $D_1 \notin /F/ \cdot D_2 \in /F/ \cdot (D_1 + D_3) \notin /F/$. Then π_e will stay, π_o will drop, and then, since the circuit will contain a gap larger than any number in /F/, the circuit cannot recover F-stability and will be extinguished.

IVa. $D_1 \notin /F/ \cdot D_2 \notin /F/ \cdot (D_1 + D_2 + D_3) \in /F/$. Then $(D_1 + D_3)$ must be in /F/ (since /F/ is an interval). π_o must drop. The circuit will recover F-stability whether or not p_o is also dropped.

IVb. $D_1 \notin /F/ \cdot D_2 \notin /F/ \cdot (D_1 + D_2 + D_3) \in /F/$. π_e must drop. If π_o also drops the circuit will be extinguished; if π_o survives until it passes the point at which π_e dropped, the circuit will recover F-stability. This is the only indeterminate case; the probability of recovery must be computed:

The probability that π_e last fires cell N_{e+t} is $p^t(1-p)$. The probability that pulse π_o survives to fire this cell is p^{t+1}, for π_o is certain to fire cells $N_0, N_1, \ldots, N_{e-1}$, and it will

have probability of p of firing each of the remaining $t+1$ cells. The total probability that π_0 will pass the point at which π_ϵ drops is then

$$\sum_0^\infty p^t q p^{t+1} = qp \sum_0^\infty (p^2)^t$$

$$= \frac{qp}{1-p^2} = p/1+p.$$

5/6.2.1 The results of the case analysis fall into three groups.

A). (Cases I,II,IIIa,IVa). In each case F-stability is recovered. In order that one of these cases arise, the proposition

$\left[D_1 \in /F/\right]$ or $\left[D_2 \in /F/ \text{ and } D_1 + D_3 \in /F/\right]$ or

$\left[D_1 + D_2 + D_3 \in /F/\right]$

must hold.

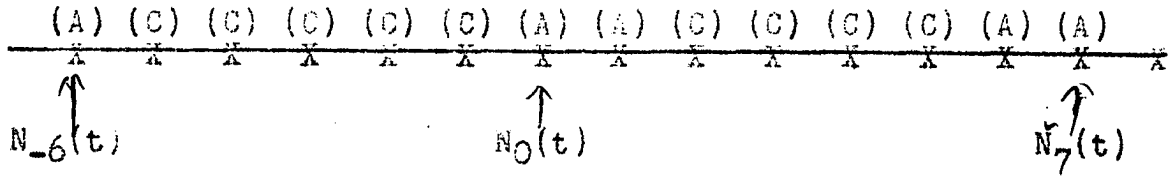
B). (Case 3b) Here extinction is certain. The condition is

$(D_1 \notin /F/).(D_2 \in /F/).(D_1 + D_3 \notin /F/)$

C). (Case IVb) Here extinction occurs with probability $1-(p/1+p) = 1/(1+p)$. The condition is that none of D_1, D_2, or $D_1 + D_2 + D_3$ be in /F/.

The occurrence of each case depends on the location of the extra pulse and on the extent of /F/. The former determines the values of D_1 and D_2. D_3 is determined

by the location of the pulse π_{-1}. For a given /F/, and a particular F-stable circuit, each location (modulo rotation) may be said to have a <u>vulnerability index</u> A,B,or C(or 0,1,1/(1+p), the probability of extinction if an extra pulse were introduced at that point). For example, if /F/ contains only the integers 6 and 7, then the figure shows the vulnerability index at each point:



(Note that if the left hand pulse were moved back to N_{-7}, then the vulnerability index of N_1 would change from (A) to (B).)

Now if we introduce a <u>random</u> extra pulse into an F-stable circuit, then we can compute the probability with which the circuit will be extinguished <u>if</u> we can compute the relative numbers of points having each vulnerability index. In fact, if p^A, p^B, and p^C are these relative numbers, the probability of extinction will be just $p^B + p^C/(1+p)$.

However, if there is a refractory period, then it is not possible to introduce pulses into every position; cells are blocked for an interval to the left of each pulse. The condition (of A) that $D_1 \notin /F/$ implies that the extra pulse is close to the left of a pulse of the

cycle, so that the refractory period tends to prevent extra pulses from entering at A-points, so that the presence of a refractory period will not greatly decrease the extinction potential of a random extra pulse.]

5/6.2.2 Let f_1 be the first integer in /F/, and f_2 the last. Let $N_a(0)$ and $N_b(0)$ be consecutive pulses in an F-stable circuit. Then the condition $D_1 \in /F/$ will be satisfied by the f_2-b+a cells to the left of N_b, hence they will be "A-cells." The condition $D_2 \in /F/$ will be satisfied by just the b-a-f_1 cells to the right of N_a. These may or may not be A-cells, by case IIIa. Now let us assume that $f_2 < 2f_1$. If this holds, then no condition of the form $[D_1+D_2+D_3 \in F]$ can hold, since this would require that the sum of two integers of /F/ be also in /F/. Then no other cells can be A-cells than those mentioned above, except the cells N_a and N_b themselves. Now suppose that the cycle has length n, and that the F-stable pattern has k pulses. Adding all around the cycle, the total number of left A-cells is $kf_2 - n$. The number of right A-cells is less than or equal to $n-kf_1$. Adding the k A-cells which carry the pulses of the pattern, we have

Theorem 5/6.2.2 If $f_2 < 2f_1$, i.e., /F/ contains no pair(r,2r) of integers, k is the number of pulses in the cycle, n is the length of the cycle,

Then the number of A-cells is less than or equal to

$k(1+f_2 - f_1)$, hence $p^A \leq \frac{k}{n}(1+f_2-f_1)$

Furthermore, since the space between pulses must be at least f_1, we have $\frac{k}{n} \leq \frac{1}{f_1}$ and it follows that

$$\boxed{p^A \leq \frac{1+(f_2-f_1)}{f_1}} .$$

It follows that if /F/ is relatively short compared with /0/+/p_1/ (the two earlier phases of recovery), then p^A is small. The probability with which a random pulse will extinguish activity is better than

$$\frac{1-p^A}{1+p.}$$

This extinction efficiency may therefore be assumed to be of an order of magnitude not less than 50%.

5/6.2.3. Another viewpoint toward the result of theorem 5/6.2.2 is the following:

An extra pulse can fail to extinguish ~~an~~ F-stable pattern only if it can set up another F-stable pattern, or the original. If /F/ is narrow, then in most cases an extra pulse will inteffere and eliminate the pulse following it. In this case, it will no longer be possible to reestablish the original pattern. But as was seen in 5/4.1.e, if /F/ is narrow there will be only a few

possible F-stable patterns, hence it is that much the less likely that the circuit can recover.

The value of F was assumed to be unity in this section. If F is less than unity, then in every case, the probability a circuit will be extinguished shortly after introduction of an extra pulse can only be increased, hence this assumption could not weaken our results.

As pointed out in 5/6.2.1, the presence of a refractory period makes it less likely that a random pulse can enter certain positions of the cycle, and hence may reduce the facility with which cycles can be extinguished by noise. These points form an interval to the left of the pulse points, and hence overlap the interval of "left A-cells" of 5/6.2.2. It can be seen, therefore, that if /O/ is itself short in duration, or of the same order of length as /F/, that this effect will not be marked.

5/6.2.4 Returning to the discussion of 5/6.0, it appears that when F-stable cycles are embedded in nets in which there is additional activity, or "noise," the extraneous-pulse effect will be a major contributor to extinction. We will assume that the relative durations of the recovery phases are such as to favor this effect. There is however, one further observation on extinction,

or related to extinction, that will tend to moderate the necessity to impose strong conditions on the recovery sequence.* It may be noted that in cases I, IIIa, and IVa, while F-stability is restored, it is not in the original pattern. In many of the applications, a change of pattern may be equivalent to extinction. In cases I and IVa the number of pulses may be changed (certainly in I). In this event, the alteration will not be reversed by the effect of theorem 5/4.2.3; in case IIIa, where the position of a pulse is simply shifted, that effect will tend to restore the equilibrium pattern.

5/6.2.5 Suppose that /F/ varies from one cell to another. Let $/F/_i$ be the /F/-phase of cell N_i. Let I_j be the intervals between pulses in the cycle. Then it is easy to see that the pattern can be F-stable only if, for each j,

$$I_j \subset \bigcap /F/_i$$

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*Note. Neurophysiologically, for widely spaced firing, it is not the case that /r/ is short as compared to /0/+/p1/. However /F/ does become short (and may even vanish) after a few closely-spaced pulses, and it may be expected that this will occur shortly after initiation of F-activity. Furthermore, during the same "fatigue" effect, the length of /0/+/p+/ is increased, further improving the $\frac{f_2-f_1}{f_1}$ ratio.

The fact that /R/, the "subnormal" phase, is quite long in most nerve increases the probability of extinction in case IVb, and improved our result. A further effect of this property of nerve is that if a cycle is extinguished, then it will be exceptionably difficult to start it again for an appreciable fraction of a second.

In particular, if $\bigcap_i /F/_i$ is empty, no F-stable pattern is possible. If the intersection is narrow compared to $\max_i (f_1)_i$, then few F-stable patterns are possible, and by the argument of 5/6.2.3, extraneous pulses are effective in extinguishing F-stable patterns.

5/7. F-stability in random nets.

5/7.0 In this section we consider certain aspects of the behavior of random nets, with particular emphasis on the forms of F-stability. The statistical parameters of the underlying nets are, so far as possible, not specified. Instead of attempting to describe the behavior of any particular net or type of net, the attempt will be made to describe various kinds of events that may occur in any (sufficiently complicated) net. Knowledge of the complete structure of a net would make it possible, in principle, to make a complete catalog of its behavior, and to assess the prominence in behavior of the types of activity which are emphasized here. However, in any non-trivial net, such computations would be, in general, enormously complicated. In my opinion, detailed analysis of a reasonably complicated net could be made practical only through the use of modern high-speed calculators, and the capacity of present-day machines would be strained by a net of more than a few hundred cells. Specific questions about special forms of behavior might be more easily answered

by computation; when it becomes clearer what questions are to be asked, this form of attack may seem more reasonable. For the present, existential and classification arguments will have to suffice.

5/7.1 Let N be a random net composed of cells N_i and connections which will remain unspecified. We will <u>not</u> assume that each N_i has the same recovery sequence.

Assuming that F-stability will be of primary interest, the following approach might be attempted: The net can be regarded as supporting a set S of potential F-stable cycles, i.e.,

$C \in S$ if $C = \left[Nc_1, \ldots, Nc_{i_c}\right]$ is a cycle, and

$$\bigcap_{s=c_1}^{c_{i_c}} /F/_j \neq 0. \quad \text{(See 6/2.4)} \qquad (1)$$

One could then attempt an algebraic theory of the relations between the elements of S. Conceivably, this could lead to another network theory, in which the <u>cells</u> are <u>the F-stable cycles of N</u>. This would be very desirable, insofar as the F-stable cycles are in a natural way functional elements of the behavior of N, e.g., if attention is to be concentrated on sustained forms of activity.

In my opinion, such an algebraic (or set-theoretic) approach would not be feasible in nets whose connections are predominantly of the multiple-threshold type. In such nets (and presumably the nervous system is of this

type) the elements of sustained activity, which may well be F-stable patterns, will not be simple cycles in general, but will include more general forms of oriented graphs, and the relations between these graphs will be exceedingly complex. The graphs themselves can be examined by qualitative methods in many cases, and some approaches are indicated below.

5/7.2 Suppose that the net N is quiet for $t<0$. Choose an arbitrary cell K as an input cell, and let K be fired (from outside the net) at times $t_1, t_2, \ldots$

K sends fibres to other junctions. Not all of these can necessarily be fired by K, for the other junctions may require multiple simultaneous stimulation. However, if J is a junction that <u>can</u> be fired by K alone, <u>then</u> it will be fired, eventually. J will fire <u>regularly</u> in response to the stimulation from K only if some condition like

$$n>n_o \Rightarrow \left[(t_{n+1}-t_n) \in /F/_J\right]$$

is satisfied.

In such a case, we shall say that "J is recruited into F-activity by K." J and K in turn may recruit more cells, and a regularly firing pattern may grow out from K. The statistical parameters of the net will determine the behavior of this pattern.

5/7.2.1 Definition: Suppose that at time $t = t_0$, the properties of every cell are changed so that, after t_0, $p_1 = p_2 = p = 0$, and $F = 1$. i.e., the transmission probabilities are zero except when the cells are in /F/, and then the probabilities are 1. And let the outside stimulation be continued. Then the subnet which remains active will be called the "F-graph of N at time t_0," or "$F(t_0)$-graph of N." In the present case, where only the cell K is stimulated, we will refer to this subnet as $F^K(t_0)$.

This definition is far from satisfactory; the term "remains active" is not well defined. [If K is stimulated at a fixed frequency, then at some time after the above change in cell properties, the activity will become periodic, and this periodic pattern might be a good referent of $F^K(t)$]. In any case, $F^K(t)$ is supposed to represent a measure of the extent of F-activity in the net at time t. (The formal alteration of the transmission properties is not an operation actually proposed for nets; it represents a formal operation of "freezing" just that activity that resembles F-stability, for definitional purposes.)

5/7.2.2 Let K be fired at times t_i (as in 5/7.2). Then each cell of $F^K(t)$ must satisfy a condition like that

of 5/6.2.4. If $I_j = t_{j+1} - t_j$, then for each j, $I_j \in /F/_{N_i}$ must hold in order that N_i be a member of $F^K(t)$. Thus each stimulation spectrum (the set of intervals between consecutive pulses of the stimulation) determines a subnet of cells for each of which, /F/ contains the spectrum. And each $F^K(t)$ must lie in such a subnet.* If /F/ is narrow for each cell, then for different stimulation frequencies, the domains of possible F-activity will be distinct.

NOTE: The nets of the brain contain many morphologically distinct types of neurons, in a common (spatially) net. It is likely, then, that the brain contains cells with widely varying recovery sequences. For different stimulation spectra, more or less distinct subnets will become F-active, and it is conceivable that these subnets might function more or less independently, as "sub-brains." This idea will be further developed in chapter 6, where a reinforcement theory for F-activity is introduced. In this theory, it is possible to apply reinforcement independently to these subnets. When any given subnet is active, the activity of the others cannot interlock with it in a regular pattern, and each will be a source of noise to the others. Since, in the theory of chapter 6, control of noise will imply control of reinforcement, this means that our random

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*Exceptions to this are noted in 5/7.2.6.

nets contain, in principle, the basic materials for highly complicated interdependent reinforcement systems.

5/7.2.3 The growth of $F^K(t)$ proceeds through the "recruitment" into F-activity of cells on the fringe of the pattern at any time. Only cells whose /F/ phase contains the stimulation spectrum can be recruited. While cells may be added to the periphery of $F^K(t)$, other cells already in $F^K(t)$ may drop out of the pattern. There are a number of ways in which parts of the F-active pattern can be lost.

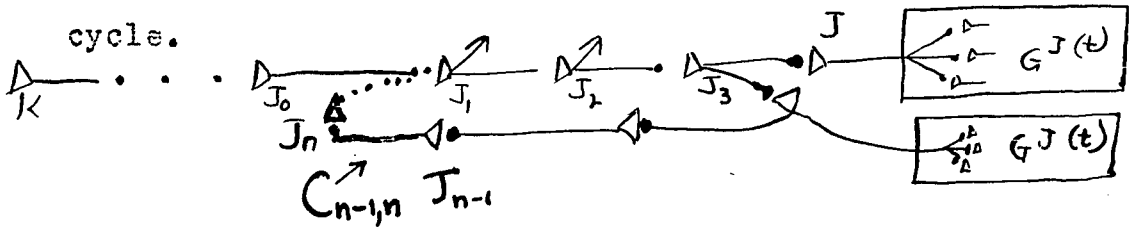
1). Cessation of stimulation. If K is prevented from firing, then all cells of $F^K(t)$ will drop out of the pattern except these which remain activated by F-stable cycles, if there are any in $F^K(t)$, or if any form before activity ceases.

2). F-failure. If F is not unity then parts of the pattern are certain to fail eventually.

[In a theory of F-activity in nets, a notion of dependency can be defined; a part P of an F-active pattern can be said to depend on a cell C of the pattern if F-failure of C causes the loss of the part P. Similarly, each location of the pattern may be said to have a vulnerability index with respect to P, representing the probability that P will fail if an extra pulse is introduced at C.]

3). Effect of extra pulses. The growth of $F^K(t)$ may cause parts of itself to fail by direct interference. The next few paragraphs are examples.

5/7.2.4 As new cells are recruited into the F-graph of K, the connectivity of the pattern may change. It would seem that whenever an increase of connectivity occurs the probability of loss of parts of the pattern is high. Consider, e.g., the effect of the formation of a simple cycle.



Let $F^A(T)$ have the form shown in Fig. 1, where each depicted cell is in $F^A(T)$ except the cell J_n. Then when (and if) J_n is recruited into the pattern one of the following will result. (Assume that F=1 for simplicity; the effects of an F-failure are obvious.)

I. If the pulses arriving at J_1 from the loop are in coincidence with the pulses arriving from J_0 at all times, no part of the pattern will be lost. In fact, the parts of the pattern previously dependent on J_0 will no longer be so; failure of K or of any part of the pattern before J_0 will not cause the part after J_0 to be lost. Hence such a loop will tend to increase the stability of the part dependent on J_0. Note however, that the condition for this to happen is rather

strong; K must fire at regular intervals, or in a sequence periodic with period n.

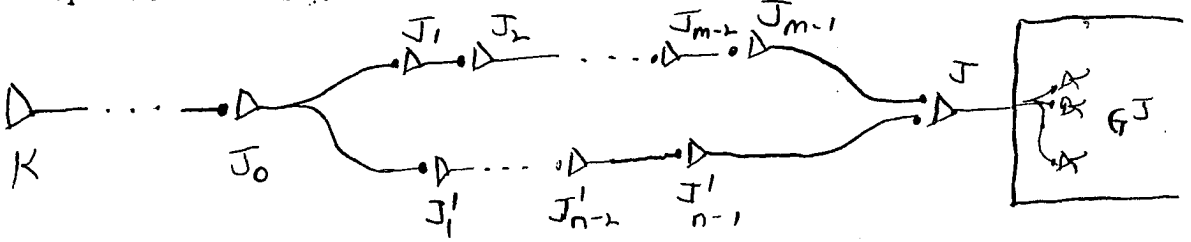
II. If the pulses arriving at J_1 from J_n are <u>not</u> coincidental with those from J_0, there are several cases. If the pulses from the loop arrive during the refractory period of J_1 they will have no effect, (although they might serve to stabilize the system in the case of later J_0-failure). If they arrive at any other phase of J_1 they will have a marked effect on the system. For if they arrive in /pl/, they will act as extraneous pulses, and as a steady supply of them! Hence both the loop, and everything dependent on it, including G^J and $G^{J'}$, will be lost, (with probability 1, since extra pulses are injected until failure occurs).

On the other hand, if the loop pulses arrive when J_1 is in /F/-phase, this does <u>not</u> imply that the system is undisturbed. For the activity of the pattern previously dependent on J_0 is then dependent on the autonomous cycle. And the stimulation arriving at J_1 from K via J_0 plays the role of extraneous pulses, and is likely to extinguish the cycle, since the extra pulses again are supplied until (and after) extinction occurs! Even if the cycle and the stimulation are able to return to synchrony at a later time, there will be a transient disturbance that may cause the loss of G^J and $G^{J'}$ if J and J' have short /F/-phases, or if G^J

and $G^{J'}$ themselves contain autonomous cycles which are synchronous with the stimulation. If the stimulation and the cycle have independent frequencies, then the extra pulses will appear at all phases of the recovery cycle of J_1. Even if failure of the cycle does not occur immediately, J_1 will be alternately dominated by cyclic and by stimulatory pulses, and each time the transition occurs, a transient disturbance will be transmitted to G^J and $G^{J'}$.

Thus it is seen that the incorporation into $F^K(t)$ of a subnet which, in itself, is potentially F-stable, does not imply that $F^K(t)$ has itself become less fragile. Such an inclusion may provide a continuous source of disturbances to itself, and to other parts of $F^K(t)$, and in general would seem to reduce the stability of the total pattern.

5/7.2.5 Another way in which connectivity may be increased is depicted in Fig. 2:



If m and n are the lengths of the two paths from J_0 to J, then $J(t) \Rightarrow J_{m-1}(t+m-1)$. $J'_{n-1}(t+n-1)$. If $m \simeq n$ there is no effect on G^J when the second path is recruited

into F^K (t). Otherwise, in general, extra pulses will be introduced into C^J, and loss of that pattern may occur. (Also, in general, the interval spectrum of activity in the net will be broadened.)

5/7.2.6 If stimulation of K is regular, with intervals d, and if it happens that $m - n \equiv d/2 \mod (d)$ then pulses will arrive at J at regular intervals of d/2. In this case, J may initiate another pattern of F-activity in the net, $F^J(t)$ which operates at the doubled frequency, and is composed of cells from a domain different from that of $F^K(t)$ (See 5/7.2.2). Although $F^K(t)$ and $F^J(t)$ may not be able to share the same cells, they may each act as a noise source for the other, and perhaps interfere until one is destroyed. It is through events of this type that activity may arise in a spectrum domain without prior stimulation by patterns within that interval spectrum.

5/8.0 MULTIST BLE PATTERNS

Although the addition of potentially F-stable patterns to an F-graph does not necessarily raise its stability, as was illustrated in 5/7.2.5, there may be special cases in which coupled circuits may attain a high degree of invulnerability to extraneous pulses.

5/8.1 Consider the case of two cycles with a common chain. (Fig. 5/8.1)

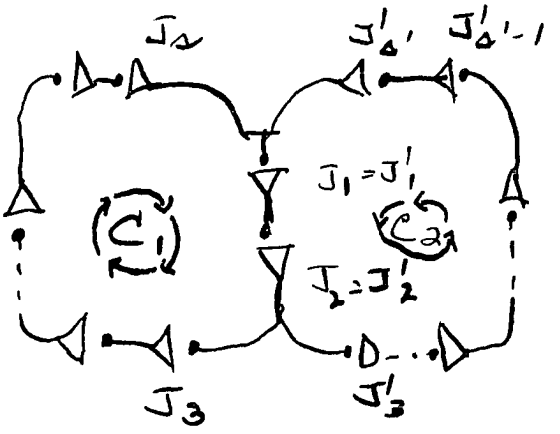


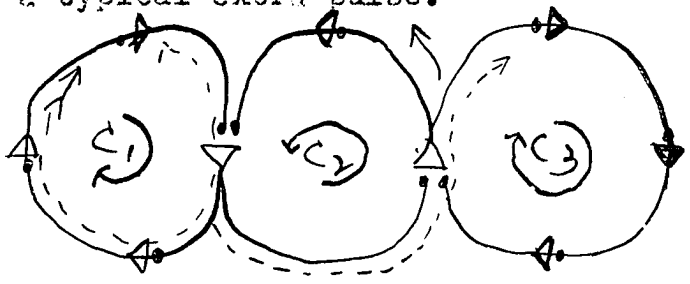
Fig. 5/6.1

There are various conditions under which such a net can become F-stable. The simplest condition is to let the activity be symmetrical about the common chain. Now consider the effect of introduction of an extra pulse. There are two cases:

i. If the extra pulse is introduced into a cell of the common chain, or if it is introduced into a side chain, but reaches the common chain before it disappears, then there is a high probability that all activity will be extinguished, as if extra pulses were introduced into separate cycles. (The probability of complete extinction is not, however, a simple function of the probability in the single cycles case.)

ii. If the extra pulse is introduced into one of the side chains, and does <u>not</u> reach the common chain, then the other loop will remain F-active, and may reactivate the loop in which failure occurred. A long 131-phase /Pc/ would make this less likely. See footnote (5/6.2.4).

5/8.2 If a circuit contains several loops, the conditions under which a single extra pulse will cause total extinction become more restricted. (So do the conditions on the net for F-stability to be possible.) In the case (Fig. 5/3.2) of three cycles with two common chains, an extra pulse would have to propagate through a distance of the order of a full cycle in order to enter all three loops. (The dotted lines represent the trajectory of a typical extra pulse.



5/8.2

It is interesting that total extinction in such a net would seem to be favored by relatively high values of transmission probability in phases other than /F/, in order to facilitate the entry of an extra pulse into each loop of the circuit.

Even if the extra pulse does not propagate into other loops, if loop 1 recovers F-stability in a different pattern, it is likely to interfere with and extinguish the adjacent loop.

5/8.3 With small values of $/p_1/$ and /p/, it might seem a circuit with many coupled loops might be so stable that it might become a relatively permanent part of a net, or even that a random net might assemble itself into a

collection of stable subnets. (Such a net would not be useful in this theory.) Such an eventuality does not seem too likely, for side-chains will tend to grow out from any F-active circuit, and if a side chain becomes re-entrant, it is more likely to be destructive than stabilizing. (In section 5/9 the growth of side chains is further examined.)

However, it would seem that the extent of the growth of autonomous F-stable subnets will depend greatly on the statistical parameters of the underlying nets, and it is not impossible that in particular nets, such "Multi-stable" patterns might tend to grow and dominate the net with useless massive periodic activity. If this happens to be the case for the random nets of the brain, then it is necessary to provide a dependable way of removing such massive activity. The following proposals suggest ways in which this could be done. They are based on the provisional hypothesis that the cortical nets are such that there is a tendency for multistable patterns to become extensive. If this is not the case, then the proposals and interpretations are not necessary. (The behavior of the cortex in epileptic (and perhaps migraneous) seizures may indicate that the cortex has the potentiality of operating in a persistent massive synchronous manner under some conditions, although these patterns might also derive from abnormal subcortical activity.)

5/8.3.1 Junctional fatigue." Persistent synochronous activity could be eliminated if an additional synaptic postulate of the following nature were added: "The /F/-phase of any junction becomes shorter, or vanishes, after a sufficiently long period of frequent transmission." (For present purposes, an equivalent postulate would have the value of r become smaller, rather than have the duration of /F/ become shorter.)

Such a postulate has a definite analogue in the known behavior of real nerve fibres. The supernormal period is known to shorten and vanish after sufficiently long periods of stimulation at sufficiently high frequencies. (See e.g., Fulton p105, fig. 32.) and this effect is observed at frequencies whose period lies in the (original) F-phase, so that the analogy seems plausible.

5/8.3.2 "External asynchronous stimulation." A multistable pattern will necessarily operate in a narrow pulse interval spectrum. An extinction process could operate through the simple medium of a massive discharge into the cortex at a different frequency, or even a single occasional blast of noise.
(It is conceivable that extinction processes are not always adequate in the brain, and that in some brains, the parameters of the net are (or become!) such that growth of multistable subnets become a serious

pathological problem. The effect of medical "shock" treatments might conceivably be a result of extinction of these subnets; in the case of electrical shock by introduction of a massive discharge of extraneous pulses; in the case of chemical shock, by the alteration of the synaptic parameters in such a way as to change the excitability properties and make the pathological multistable patterns untenable.) Any noise level sufficient to extinguish multistable patterns will also be sufficient to extinguish ordinary F-stable cycles. Anticipating the arguments of chapter 6, if memory is, in part, due to the consolidation of previously F-active circuits, then extinction as a result of massive discharges, or "shock," would also tend to produce the "retrograde amnesia" which is a familiar sequel to electric or traumatic shock.

5/8.3.3 Methodological note. It is frequently asserted that it is premature, and hence undesirable, to speculate about the nature of psychological and psychopathological phenomena on the basis of an unestablished theory. (This attitude represents, in my opinion, a reaction against the wave of inadequate "mechanistic" theories of the early twentieth century. It is unfortunate that this attitude should persist in the present time, for "mechanistic" theories are in the process of becoming, in my opinion, vastly more sophisticated than the

"dynamic" theories of present-day psychology). In all branches of science, it is important to examine the postulates of a theory. But even when they have not been proven, it is customary to entertain hypotheses provisionally if they show promise that, once established, they will provide convenient explanations of the behavior of the system concerned. In the particular example at hand, the pathological processes that are conceivably attendent on the uncontrolled growth of multistable patterns, and their domination of activity in a net, depend on postulated synaptic properties which at present have not been verified. Suppose, for example, that there is a pathological syndrome X which tends to cause general broadening of the /F/-phase. It would seem almost certain that this syndrome would cause the extension of multistable patterns, and cause a behavioral (or "affective") disorder. If "X" were by nature an "observable" lesion,i.e., if changes in the junctions are observable by laboratory methods, the disease would be considered "organic." If no "changes" were observable, the disorder would be considered "functional." There seems to be a tendency among many psychiatric workers to regard this distinction as basic in some way, although many recognize the notion of "biochemical lesion" and the possibility that a psychopathological state may be a consequence of an

unobservable, yet "organic," disorder. I feel that it is particularly important to point out that it is extremely probable that brain function is greatly dependent on synaptic properties, while our present-day methods of studying synaptic properties in the brain are inadequate (to say the least). Thus it is particularly dangerous to imply that on the basis of no "observable organic pathology," a certain condition is "functional" in nature. The methodological error would not have serious consequences, perhaps, if there were not an associated tendency to treat "organic" conditions by "biological" methods whenever possible, but to treat "functional" disorders with "psychotherapeutic" methods. I fail to see why it is less significant that a "depression" may in some cases be relieved by certain drugs, then that the psychological manifestations of a meningitis may in some cases be relieved by the use of antibiotics.

However, there is a sense in which the use of the term "functional" would be appropriate in the classification of behavioral disorders. It is easy to imagine (although I am not at present prepared to construct) nets in which the a priori probability of occurrence of a gross, autonomous synchronous pattern is low, but in which such patterns are possible. In this case, the pathological behavior that occurs as a consequence of

the occurrence of the multistable pattern may be called "functional," in sense that no "organic," including synaptic, changes have occurred.

It is important to add that in systems which have extensive learning properties, the definition of "pathological" behavior pattern must always be carefully formulated, for evident reasons.

5/8.3.4. If, as suggested in 5/8.3, the nets of the brain require an additional extinction mechanism, it might be proposed that the large electroencephalographic potentials are due to massive synchronous discharges, which, whatever their nature, serve as extinction sources. However, it is equally easy to suggest that these potentials are due to the synchronous activity of large multistable patterns. Under this hypothesis, the different frequencies observed might be associated with the different spectrum domains proposed in 6/7.2.2. Or these potentials might also be associated with reinforcement (chapter 6), With the evidence that some of the rhythms are associated with sensory mechanisms, etc., it is more than likely that the "brain waves" do not represent any one process.

The necessity for periodic sleep in the highest animals may be associated with a need for an occasional clearing out of multistable patterns. Sleep might be the period in which subcortical structures, e.g., the

hypothalamus, control the removal of organized activity from the cortex. The occurrence of dreams during sleep is not particularly incompatible with this proposition, since (1) there is no necessity to suppose that the disorganization must be complete, and (2) dreams have a notable quality of disorganization, notwithstanding the fact that the "elements" of a dream may have significance.

5/9. MULTIPLE JUNCTIONS AND F-ACTIVITY.

5/9.0 Multiple junctions, discussed at length in chapter 2, have been suppressed up to this point in order to develop the notion of F-stability without becoming involved with other aspects of random net activity. It is almost certain that the majority of certral nervous system junctions require more than one pulse for excitation (at least during ordinary states; see 5/9.1 below), and some of the consequences of this fact are examined in this section.

5/9.1 Recovery sequences of multiple junctions.

Let J be a multiple junction, i.e., one for which the transmission probability is (normally; in phases /p_1/./p_2/, and /p/) low unless a certain number, n_j, of pulses arrive at J in a very short interval. In view of the arguments of 3/2.3/2 (see also Fulton; pp 138-140) the following postulates would seem to make a

biologically plausible, if idealized model.

J-1: After J is fired it passes through the recovery phases as described in 3/2.1 and in the present chapter. For each possible stimulus, the transmission probability depends on the recovery phase at the time.

J-2: The transmission probability, for stimuli containing less than n_j (the threshold number) of pulses, is (very) low except when the junction is in /F/-phase. But in the /F/-phase the transmission probability is of the order of magnitude of F, at least for stimuli containing n_j-1 pulses.

Postulate J-2 suggests that in the supernormally excitable phase, the numerical threshold is lowered, in addition to the raising of the transmission probability.

The postulates for multiple junctions are idealized in the sense that the recovery cycle is not explicitly dependent on the manner in which the junction is fired, and that the recovery cycle is the same for each type of stimulus. Also, the "fatigue" postulate suggested in 5/8.3.1 is not included. The following chapter will introduce further modifications. The important notion here is the reduction of numerical threshold during supernormal phases.

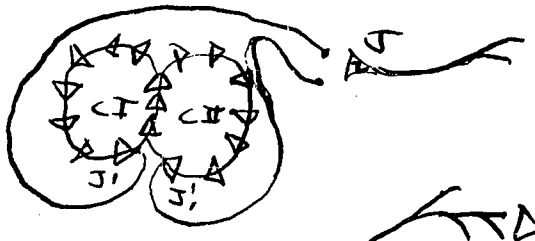
5/9.2 Growth of F-graphs in nets with multiple junctions.

If the underlying net is composed predominantly of junctions with multiple thresholds, then the growth of F-active patterns is contingent on pulses meeting the stringent conditions for firing a new junction, as well as on the new junction meeting the appropriate stimulation interval spectrum conditions. Once a new junction is fired the condition on the number of pulses required is relaxed, by J-2 of 5/9.1, so long as the intervals between stimulations continue to lie within /F/. There are two ways in which a new cell (junction) may be recruited into an already F-active pattern:

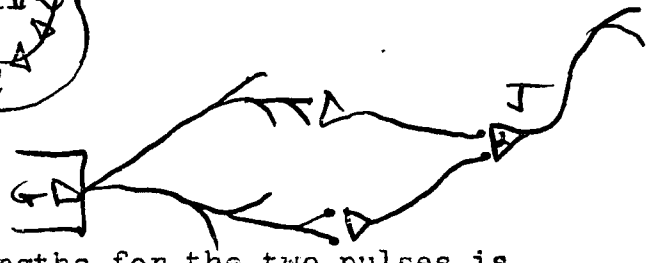
5/9.2.1 I. Pulses from within the existing F-graph may satisfy the normal threshold for the new junction. Three examples are

Ia

Ib



Ic



In each case the path lengths for the two pulses is the same, assuring that the threshold condition of two pulses is met. (In Ib, the two cycles are symmetrical so that the pulses arrive at J simultaneously.)

Assuming that J has an appropriate /F/-phase, once J is fired, it will be recruited into F-activity.

The effect of the postulate J-2 can be seen in the response of the net of Ib to an extra pulse. If an extra pulse is introduced into one of the cycles, say CI, and causes failure of that cycle, (and assuming that the extra pulse does not go as far as J_1) then J_1 will be lost from the F-activity, and J will receive only one pulse at each stimulation. Nevertheless, J will remain in the F-stable pattern. The same would be true for Ic if in some way one of the intermediate cells were inactivated. Thus the conditions for maintaining a multiple junction in an F-stable pattern may be less stringent than the conditions for recruiting it. On the other hand, a single extra pulse can extinguish a multiple junction from a pattern only if it is able to fire the junction. J-2 does not specify whether the transmission probability remains of the order of F when the stimulation is well below the normal threshold.

5/9.2.2 II. An extra pulse, plus pulses from the existing F-graph may combine to satisfy the firing condition of the new junction. But then, by J-2, the junction can remain F-active without the repetition of the extra pulse.

It is important to note that, without the extra pulse, it might not have been possible for J to have been recruited into the pattern. Thus the presence of "noise" in the net may stimulate growth of F-activity, as well as its destruction.

5/9.2.3 The type of growth indicated in 5/9.2.1 will be influenced by the statistical parameters of the net. If the threshold is high, growth will be slow. Both the growth rate and the destruction rate will increase with the amount of F-activity already in the net, since the recruiting condition requires multiple coincidences, and as in a large net, large parts may be destroyed with a single extra pulse.

For a net composed essentially of n-ary junctions, with a complete statistical homogeneity, growth rate might tend to depend on the n^{th} power of the amount of activity at the time, since recruiting of a new junction requires n simultaneous pulses. No such assertion could be made within a net with significant spatial preferences of connection, without extensive examination.

The type of growth indicated in 5/9.2.2 is due to the presence of "noise" in the underlying net. For the applications of chapter 6, it will be convenient to summarize the effects of this "noise."

5/10. Given a net N and an F-active pattern G, "noise with respect to G," or "G-noise" can be defined as "all activity of N which is not a direct consequence of the activity of G." The presence of G-noise has been shown to have two effects on G.

1). Noise, in the form of the "extraneous pulses" of this chapter, is an agent which plays a large part in the destruction of F-graphs.

2). Noise makes possible the incorporation into F-graphs cells (junctions) which would otherwise be accessible with very low probability.

Both effects are in the direction of changing the existing pattern of F-activity.

On the other hand, assuming that the net is normally connected to a source of noise, then <u>removal of noise tends to stabilize the present pattern in two ways</u>:

1). Removal of noise removes the extra pulses which destroy F-activity.

2). Removal of noise tends to slow the process of recruiting new junctions into the F-graph.

(There is a second-order effect in that removal of noise tends to slow the formation of side chains, and the especially destructive effect of the closure of a non-synchronous side chain.)

In the applications, it will be assumed that

i. The effect of noise is relatively large, at least in its role as extinguisher.

ii. In the absence of noise the stability of F-active circuits is high, and the growth of side chains slow, with the reverse holding in the presence of adequate noise levels.

Each of these propositions will be favored if

A. F is close to unity

B. p_1, p_2, and p are small, and their phases are broad.

C. Multiple junctions are preponderant.

Verification of these propositions for a reasonably large net would require an enormous number of calculations. It is hoped that in the near future it will be feasible to study the problem with the aid of a high-speed digital computer.

CHAPTER 6

A REINFORCEMENT THEORY FOR CYCLES AND SOME APPLICATIONS

CHAPTER 6 A REINFORCEMENT THEORY FOR CYCLES AND SOME APPLICATIONS

6/5 Internal reactive properties of nets

6/5.1 Intercepting surfaces. "Pseudo-stimuli."
6/5.2 "Pre-motor" activity

6/6 "Contiguity" of stimuli

6/6.1 Simultaneous stimulus pairs
6/6.2 Sequential stimulus pairs

6/7 Prediction theory

6/7.0 The prediction process
6/7.1 Motor gates
6/7.2 Location of the predictor net
6/7.3 Behavior of the system with a fixed random predictor net
6/7.3.1 Actions with dependable sensory consequences
6/7.3.2 Prediction of environments of the form of 4/3.2.2
6/7.4 Control of the motor gate by the secondary net

CHAPTER 6.

THE REINFORCEMENT THEORY OF CYCLES AND CERTAIN APPLICATIONS

6/0 Introduction

This chapter begins with the proposal of a possible neural basis for reinforcement. This proposal is then followed by various applications to more or less special assemblies of random nets with the objective of indicating the possibility of obtaining higher forms of behavior.

Unfortunately, I have not, as yet, found convincing support for many of the propositions in this chapter. While many of the statements of chapter 5 were formulated in such a manner as to be invariant of the statistical parameters of the underlying random nets, this method does not seem to extend to the basic processes of the present chapter. This is perhaps an inevitable consequence of the fact that these processes are concerned with larger patterns of activity, e.g., control of one net by another. Still, it would be equally unsatisfactory to confine the arguments to any narrow class of nets at the present time, for the purpose of the chapter is to present several behavioral concepts and to make plausible the possibility that they can be realized in an assembly of random nets, rather than to describe the behavior of a particular net or class of nets.

Not entirely out of choice, then, we will take a middle course. For the main part, the discussion must be informal and existential, rather than formal and constructive. It is hoped that the interest of the propositions will, in some measure, compensate for the weaknesses of the arguments.

✳ As part of the supporting structure for the main propositions, it has seemed necessary to analyze a few subsidiary notions, and to draw on some biological analogies. Many of the subsidiary arguments are themselves weak, and I do not want to give the impression that I intend them to be entertained too seriously. (Accordingly, certain section numbers will be starred.)

6/1 A reinforcement theory for cycles.

6/1.0 Cycles as "functional elements" in the behavior of nets.

Much of the discussion of this chapter will be based on the assumption that in the nets under consideration, F-activity of cycles plays a dominant role in behavior. There are several motives for this viewpoint.

1. The results of chapter 5 on F-activity hold in a wide class of random nets for which the junctions exhibit the recovery sequence of chapter 3. The emphasis on F-activity is partly due, it must be admitted, to the fact that I have not been able to

develop a correspondingly advanced analysis of random nets in terms of other abstract behavior patterns. The theorems on F-activity are to a large extent independent of the statistical parameters of the nets, and I have been reluctant to restrict the class of nets in order to obtain other types of behavior.

2. A source of sustained periodic activity is an essential for any theory of the brain. At least in motor and sensory domains, single occurrences of pulse patterns are not likely to have much significance. A motor act requires a sustained flow of pulses to each muscle involved, there certainly must be mechanisms for extended persistence of various internal state conditions.

3. A technical problem arises in the consideration of any large net without cycles. If a typical path from sensory to motor channels involves more than a few junctions, then (in nets composed primarily of multiple junctions) the propagation of pulses through the net will tend to exhibit chain reaction properties. There will be a very narrow range between the minimum stimulus magnitude necessary to obtain any motor response at all, and the magnitude above which the entire net will explode into activity. In a net dominated by F-activity, this type of explosion is not

likely to occur, as the competition between cycles prevents excessive pulse densities in the net. (Another way of limiting activity would be to provide an appropriate distribution of inhibitor fibres within the net.) For a given type of net, it might not be too difficult to calculate the level of activity under various stimulus conditions; this will be attempted when the time comes to specify net parameters.

4. For the following reasons, it is not entirely unnatural to consider the F-active behavior of a net as distinguishable from other combinatorial aspects of the net's activity, with other activity regarded as noise for the F-activity. For an F-active pattern can be reliably initiated only through extended periodic stimulation, and such stimulation can be supplied most conveniently from other F-active patterns. Other sources are external sensory (an unlikely source of periodicity, and periodic patterns generated by or synchronized with autonomous periodic phenomena within the brain, (which, in turn, may be associated with F-activity elsewhere in the brain). In a deep net (see (3) above) single pulses crossing unfacilitated junctions must have a low survival probability, or the whole net would explode into activity, but the system of F-active patterns can drift about within their

spectrum domains with relatively high survival probabilities, so long as there are not too many of them. Furthermore, while extinction of an F-active pattern by a single random pulse is only probable, mutual interference of two F-active patterns is <u>certain</u> to destroy one, since the other acts as a continuous source of extra pulses. Thus the behavior of the F-active patterns of a net can be described to some extent in terms of these patterns, and the theory of F-active patterns has some degree of autonomy as part of the complete theory of the net.

6/1.1 <u>Local Reinforcement Operators on cycles</u>.

A given, potentially F-active, subnet N can be regarded as a miniature reactive system. Different stimuli may cause different reactions, and among the possible reactions are <u>establishment of F-activity</u>, and <u>destruction of F-activity</u>. Suppose that we require an operator L which can raise the "F-excitability" of the subnet; i.e., if a given stimulus S originally induces an F-active pattern B(S) in N with probability pB(S), then application of L raises that probability. One way of doing this would be to have application of L cause a rise in (the resting transmission probability) p for each junction of N which is active in the pattern B. (Note that raising the value of F would

not have any such effect on the excitability of the pattern B; it would only make B more stable and less liable to F-failure like being established, once initiated. If L is also to satisfy the definition of a local reinforcement operator, it must be such as to have this effect selectively on the junctions of recently active F-active patterns.

6/1.2 The operator L of 6/1.1 will be a local reinforcement operator if F-active patterns are regarded as the elementary reactive systems of the definition (4/5.1). Of course, the underlying random net does not split disjointly into potentially F-active patterns; two cycles may, in general, contain common junctions. Thus, if L is to reinforce a given collection of patterns, it will in general interfere with other previously reinforced patterns, and not only recently active ones. It will simply have to be assumed that the cross-effects are not catastrophic.

6/1.3 The remainder of this section is devoted to description of such an L. This L will operate entirely through neural channels. There is a basic problem involved: If L is to operate only on junctions which have recently been involved in F-activity, how is it to distinguish such junctions? We cannot expect to find an L which operates bodily on cycles, or expect to find that a junction J contains information about

which F-active patterns it has been recently involved in. However (I), it is not unreasonable to expect that a junction would contain information indicating whether or not it has recently participated in some F-active pattern, for this fact can be determined (with high reliability) from examination of the pulse history of the junction. This idea will be examined in 6/1.7. Also (II), if a junction has recently been involved in an F-active pattern, the probability is greater than chance that it is so involved at the present time, since F-active patterns have an extended lifetime. This will be especially significant if there has been a recent reduction in net noise, since this increases the average lifetime of F-activity. In order to construct an I using these principles, we require an additional axiom about synaptic behavior; this is D-2 of the following section. (6/1.4)

6/1.4 The "density axioms."

The following "axioms" are a series of proposals about the behavior of interneural junctions. We do not assume that they all hold; the discussion is rather an investigation of the consequences of assuming each one. Each proposal states that if activity at a junction satisfies a certain firing "density" condition, then the transmission properties of the junction undergoes

certain changes. These changes are analogous to those in the recovery phase sequence of chapter 3, where if a junction satisfies the elementary "density" condition of firing once, then the transmission probability passes through the excitability phases /0/, /p_1/, /F/, /p_2/, /p/. This property will be considered as axiom D-0.

AXIOM D-0: If a junction J is fired once, it passes through the recovery cycle as depicted in 3/2.3 and 5/9.1.

AXIOM D-1: Let |1| be a condition which is satisfied, at time t, if in the interval of length P_1 prior to t the junction has fired at intervals not larger than some ϕ_1.

Then D-1 states that as soon as a junction J satisfies condition |1|, the junction proceeds to pass through an extended recovery cycle of the following form. The junction retains the properties of axiom D-0 except that for a period P_1 after condition |1| is satisfied, the value of the "resting probability" p (the "p" of phase /p/) is raised to a significantly higher value p(|1|). The period P_1 is assumed to be considerably longer than that of the whole recovery cycle of D-0. It is further assumed that the transmission probabilities in the phases /0/, /p_1/, and /F/ remain substantially unaltered during p_1. (The effect is not really assumed to be in the form of a step function over P_1, but, as in 3/2.3.1, this is a convenient approximation.)

AXIOM D-2: Let |2| be a condition (T_2,ϕ_2) like |1| which

is satisfied when J has fired at intervals not greater than ϕ_2 for an interval T_2 with $\phi_2 \leq \phi_1$ and $T_2 > T_1$. Then D-1 states that if |2| is satisfied, the junction experiences a rise in 'p' which is (subject to the qualifications of D-3 below) permanent. It is further supposed that successive applications of |2| cause further rises in 'p' and that the other remarks of D-1 apply.

AXIOM D-2': Let |2'| be a condition which is satisfied if condition |1| is satisfied in a certain number of disjoint intervals all contained in an interval T_2'. Then the same conclusion as in the case of D-2.

NOTE: AXIOM D-2' is an alternative form of D-2 and leads to essentially the same results. It will not be considered separately and is exhibited only to suggest another acceptable possibility.

In a complete theory, it will be necessary to provide some means by which the resting probability can be reduced for long intervals. Axiom D-3 assumes that this can occur as a result of a junction satisfying some (unspecified) density condition |3| and also that:

AXIOM D-3: If p is reduced as a consequence of the junction satisfying |3|, then the value of p can be restored to its previous value by

(1) satisfaction of some density condition weaker than |2|, or alternatively,

(2) application of fewer occurrences of |2| than would be necessary for a junction for which p had <u>not</u> been previously reduced as a consequence of |3|. A possible form of |3| might be: J is fired frequently in an interval T_3, but not so frequently that |2| is satisfied.

6/1.5 <u>Motivation for the density axioms</u>.

6/1.5.0 Axiom D-0 has been discussed at length. It is the basis for the theory of F-active patterns, and has a plausible biological basis. (Chap. 3)

6/1.5.1 Axiom D-1 is significant, not so much in the reinforcement theory aspects of net behavior as in behavior viewed as a stochastic process over short intervals. Suppose that for a given net N, the interval T_1 (of the condition |1|) is of the order of magnitude of the duration of a "typical" F-active pattern in N <u>in the presence of ordinary noise levels</u>.* Suppose also that the frequency condition ϕ_1 of |1| includes frequencies consistent with F-activity in the net, i.e., some numbers in the interval /F/ are less than ϕ_1. Call this condition $|1|^N$. Then if D-1 and $|1|^N$ hold, the behavior of the net will exhibit some coherency over extended periods (of the order of magnitude of small multiples of P_1), because cycles which have been F-active

*Note: The interval T_1 must be longer than the subnormal phase $/p_2/$; cf. footnote page 5-46.

within a past interval of length F_1 will have a greater than a priori excitability. The behavior will tend to be composed of recurring elements in any such interval. If the net is viewed as a reactive system, the occurrence of a particular reaction S_o-R_o raises the probability that R_o will recur in a short time, even as a response to a random noise input. Internalistically, if it were desired to reactivate recently active patterns, this could be done with greater than chance reliability by simply introducing noise into the net. This might provide a basis for a "short memory."

6/1.5.2. The application of axiom D-2 requires the following condition:

Condition $|2|^N$: We suppose that the interval T_2 (of condition $|2|$) is of the same order of magnitude as that of the duration of a typical F-active pattern in the net N <u>in the absence of external noise. We suppose further that the duration of a typical F-active pattern in the net N in the presence of "ordinary" noise levels exceeds T_2 with insignificant probability</u>. Also, it is assumed that the frequency condition of $|2|$ can be satisfied by the junctions of F-active patterns, in N, i.e., that numbers $\leq \phi_2$ are contained in /F/, for most junctions of N.

It should be noted that $|2|^N$ is a condition not on junctions alone, but on the connections within and external to the net N as well, and that $|2|^N$ can be satisfied only if the conclusions of chapter 5 apply to N.

If D-2 and $|2|^N$ hold, then it follows that:

> If, at any time, t, the noise level in the net N, is greatly lowered for an interval of the order of magnitude of T_2, then there will be a (long duration) rise in the resting transmission probability 'p' of each junction of many of the patterns that are F-active at time t, hence of many that were F-active shortly prior to time t. (It is not possible to estimate how "many," at this stage.)

For if the noise level is cut at time t, then by $|2|^N$, each of the patterns which are F-active at that time will have a significant chance to survive through the interval of length T_2, hence their junctions will have a chance to satisfy condition |2|, and axiom D-2 implies the consequent rise in the value of 'p' for those junctions.

It follows that if D-2 and $|2|^N$ hold for the net N, then <u>the operation of greatly lowering the noise level in N for a sufficiently ($\geq T_2$) long interval has the effect of a Local Reinforcement Operator L.</u> We will return to this discussion in 6/1.6.

6/1.5.3 An axiom like D-3 will undoubtedly be necessary for any adequate ~~learning~~ theory of learning for if only increases in synaptic transmission probabilities were admitted, the nets would probably become "saturated" and inflexible. The particular form of ⟨3⟩ proposed in 6/1.4 is suggestive of the phenomenon of "extinction" in which repeated evocation of a learned or conditioned response without reinforcement leads to weakening of the reaction.

6/1.6. The conclusion of 6/1.5.2 states that reduction of noise in a net for which D-2 and $\langle 2 \rangle^N$ hold has the effect of a LRO. The possibility of performing this operation will depend on the connections of the net N with the remainder of the brain model. If noise is defined as that part of the net activity which is interfered with F-active patterns (thus one F-active pattern can be noise relative to another) there are several ways of controlling it.

1. Removal of sources of pulses external to N, by direct means.

2. The removal of internally generated noise is more difficult; presumably it cannot be done directly from outside the net without prejudicing the survival of the F-activity of the net. Internally generated noise is due to (a): Interference between essentially

independent F-active patterns through contiguous and common connections, and (b): Propagation of pulses through non-F-active pathways. Noise of type (b) will depend on multiple junction conditions, and will be reduced when external noise is cut. The noise of type (a) is more serious since it cannot be directly removed without interference with the F-active patterns. But this can be done indirectly: if the net is normally operated in such a manner that the number of simultaneous active F-active patterns is kept small, then there will remain little type (a) noise when the external noise is out. But this can be done simply by keeping the normal noise level (in non-reinforcement intervals) at a high level. Since this condition is probably necessary anyway in order that $|2|^N$ hold, it costs little more to assume that it will hold in our nets. If normally there is high external noise level, a reduction in this level will cause an associated drop in the internal noise of both types, and the external noise reduction will have the effect of a LRO.*

(A slightly different technique that might work if external noise were not normally high would be to precede the drop in noise by a brief increase. It seems plausible that a very short burst of noise would effectively destroy some F-active patterns, but would be unlikely to initiate any. The operation would destroy

* Noise cannot be withheld too long, or an excess of F-patterns will form.

some of the F-activity, but it would give the remainder a better chance to survive long enough to satisfy condition |2| and be thus reinforced. This technique does not seem promising as compared to the normally high noise level technique.)

The purpose of the second provision of condition $|2|^N$ is designed to prevent the (random) reinforcement of F-active patterns in the absence of an overall noise reduction. The alternative condition of |2'|, on the other hand would imply that patterns which are F-active sufficiently often would be facilitated by virtue of that fact alone, without the necessity for application of a reinforcement operator to the whole net. It is conceivable that this would be a desirable property of a brain model, but on the other hand, it is not difficult to imagine ways in which such a type of "learning" might be antagonistic to attempts to organize the net by other means. D-2' is mentioned only as an example of how a synaptic property might be reflected in net behavior; we will not assume it for the nets of this theory.

6/1.7 Biological aspects of the density axioms.

6/1.7.0 The evidence for the normal recovery sequence of axiom D-0 is reviewed in chapter 3. I have made no systematic effort to assemble evidence for the validity

of any form of axioms D-1 - D-3 in the nets of the central nervous system. The following arguments are intended only to show that these axioms need not be regarded as a large number of arbitrary assumptions about the behavior of interneural junctions, but may instead be regarded as the single proposition that, in a special way, the interneural junctions exhibit a set of properties which are more or less universally observed in living tissue.

6/1.7.1* It is frequently the case that when living cells or tissues are exposed to an "irritating" stimulus, their responses exhibit the following form. I-0: If the "irritation" is transient, the tissue responds with a transient change in properties, either morphological or biochemical.

I-1. There is a "density" condition $\{I_1\}$ for which, if the "irritation" is intense and prolonged enough to satisfy $\{I_1\}$, there is a change in the properties of the tissue of relatively long duration, with eventual restoration of the original behavioral properties.

I-2. There is a density condition $\{I_2\}$, stronger than $\{I_1\}$, for which, if the irritation satisfies $\{I_2\}$, there is a more or less permanent change in the properties of the tissue.

(I_3) If through some process, a tissue which has been exposed to irritations satisfying $|I_2|$ but have later been restored to their original properties, subsequent application of an irritation of type $|I_2|$ causes a greater change in the tissue than would otherwise be the case, i.e., apparent "healing" may be incomplete, or the tissue may have become "sensitized," in some way.

A typical example is provided by the classification of the sequalae of chemical or thermal trauma into "first" (I_0), "second" (I_1), and third (I_2) degree burns. In the third degree case, the permanent change is exemplified by the (morphological) conversion to "scar" tissue. Other examples are available, but there is little point in cataloguing them. In certain cases, the (biochemical) process of "sensitization" to foreign substances may proceed along these lines.

For the case of interneural transmission, the axioms D-0 - D-3 may be regarded as examples of I_0-I_3 if the following provisions are made:

1. The transmission of a pulse at a junction is regarded as an "irritation" to one or both of the afferent fibre ending or the efferent cell surface.

2. The changes in the properties of the tissues involved are such that the transmission probabilities of the junction is raised in the appropriate intervals.

6/1.7.2* The following is a proposal of a simple mechanism through which D-0 - D-3 could be realized in the nervous system. It is proposed only to show that D-0 - D-3 need not be necessarily regarded as a large number of independent hypotheses about interneural transmission.

A frequent form of response to irritation is a local edema or "swelling." This is often the case for thermal, electrical, chemical, or even mechanical irritation. Suppose this were true for the surfaces involved in the "irritation" of interneural transmission. Such a local edema or swelling will bring the active surfaces closer together. But then, on the basis of either an electrical or chemical agent transmitter theory, it would be natural to expect a rise in the transmission reliability, since in either case the concentration or field strength of the transmitting medium will be increased at the efferent surface. D-2 suggests that if irritation is sufficiently prolonged, the geometric changes are relatively permanent. The other axioms have an obvious analogy.

6/1.7.3. Chemical or "humoral" reinforcement theories.

The noise operated LRO of 6/1.1-6/1.6 has the feature that in a real sense it is contained entirely within the network structure of the net. It is possible to describe alternative models in which the reinforcement control channel is not a part of the net proper, e.g., as in the SNARC machine. If such a model is

proposed as a brain theory, the most natural correspondence to the extra-neural reinforcement control channel would seem to be "humoral" or vascular distribution of a biochemical reinforcement agent. Such a "reinforcing substance" would have to selectively alter the transmission properties of interneural junctions which have recently been active, or have recently satisfied a density condition. It is not difficult to envisage a mechanism through which this could occur:

It might be proposed, for example, that one of the effects of the "irritation" of interneural pulse transmission is to render the boundary membranes of the junction surfaces permeable to penetration by the circulating reinforcement substance. Thus the substance would selectively enter recently active junctions and produce the appropriate alteration of their properties.

There are limitations to such a "humoral" theory of reinforcement. First, the time of circulation and diffusion of such a substance through the brain might be too slow to be adequate for some types of learning. Also, it would be difficult to localize the domains of the brain in which a particular reinforcement act is applied, without having to introduce a large class of different agents, or postulating their injection into specialized vascular channels.

A proposal which might be a good compromise between

the humoral theory and the completely network theory is the following: There are many morphologically distinct types of neurons (and other cells!) in each subnet of the brain. It is not inconceivable that there are associated biochemical distinctions, and that the discharge of some particular type T of cell causes the release, into the surrounding interstitial fluid of the brain tissue, of the hypothetical chemical reinforcing agent. It need only be further assumed that the cells of type T are widely distributed, but can be selectively fired through one or more central channels, which thus become the reinforcement channels for the brain. Such a system would be acceptable for the theory of cycles with firing of the control channels of T replacing the reduction of noise in the role of the reinforcement operator. However many of the mechanisms of the remainder of this chapter would be difficult to realize in a simple way with this local chemical type of operator. It may be noted that this local chemical system would be much faster than a vascular system in its action, and that it is closer to being a part of the underlying net. It is perhaps worthy of mention that the fact that no "reinforcement substance" has been discovered is not good evidence with which to attack such a chemical theory of reinforcement. For in this model, the reinforcement substance is released directly into the

interstitial fluid of the brain. If, like most substances, it does not penetrate the blood-brain barrier, it would never enter the general circulation, and would not be detected there; similarly it would not have any effect when injected into the general circulation, since it would not have access to the interneural junctions. (Finally, it would have to have a short lifetime in the brain tissues if it were to provide an acceptable reinforcement operator.)

Regardless of these possibilities, we will investigate primarily the noise controlled reinforcement operator of 6/1.6. Whether or not a process of this kind has a realization in the brain, it is a convenient basis for the operation of some systems of non-trivial behavioral complexity.

6/1.8 Summary.

The arguments of this section combined with those of chapter 5 show that it is plausible that if the conditions D-2 and $|2|^N$ are satisfied for a net N, and if N is normally operated at high (externally generated) noise levels, then the act of sharply reducing the (external) noise level has the effect on N of a local reinforcement operator.

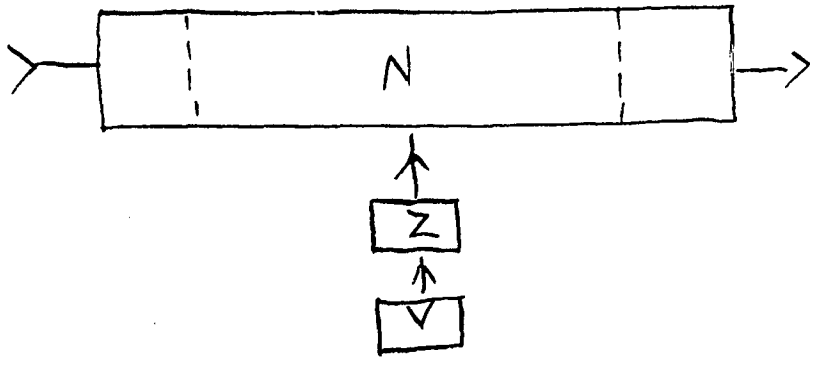
It seems likely that only computational, i.e., "experimental" techniques could determine whether this

is the case for any particular net N_0. It would also seem that only such methods could verify whether or not the resulting local reinforcement operator would have, to an adequate extent, the properties of a global reinforcement operator. (See 4/5.1). It seems advisable to characterize nets for which these conclusions hold by a special notation; we will call them **||-nets**. Most of the nets in this chapter will have these properties.

6/2 Neural primary reinforcement theories.

6/2.1 In this section we discuss the possibilities of realizing, within a neural-analog network, systems of the form (M,Z,V) (see 4/2ff), where the Z is a local reinforcement operator.

6/2.1. We first consider nets of the following form.



The main net N-I is assumed to be a (random) neural-analog net provided with a local reinforcement operator Z. [illegible]

The operator may be regarded as operating on either the principles of 6/1.6, or those of 6/1.7.3, or on any other workable principle. As was pointed out in 4/3, for global reinforcement, the behavior of the system, as related to an environment, depends strongly on the nature of the information used by the V to control the application of reinforcement. This is equally true for local reinforcement operators. We shall examine some possibilities, with attention to the biological applicability of each model.

6/2.2 In simple organisms, learning must be directed in such a way as to promote survival. (To put it somewhat less teleologically, it can be expected, that with probability much greater than chance, the process of evolution will tend to provide those organisms which have an M,A,V system with a V which tends to promote behavior which is pro-survival.) It seems to me that in those lower animals which have an appreciable learning capacity, this is accomplished in the following way. Each organism may be described as having an "INTERNAL PHYSIOLOGICAL STATE." It would be quite futile to attempt a precise specification of what I mean by this expression; I mean to indicate an "imaginary") catalog of the instantaneous metabolic and mechanical condition of each tissue of the body, excluding

specifically those aspects of the state of organs and channels which can naturally be regarded as information theoretic (e.g., the pulse state of the nervous system, or hormonal concentrations). Let us suppose that this internal state can be described by a finite number of distinct real parameters (which are supposed to represent things like concentrations of nutrients in the blood or interstitial fluids, or the mechanical forces at work in specific organs). There will be a subset of these parameters whose values must be maintained within some "normal range" if the organism is to be able to survive (or at least to be "normal"). Examples are (i): the concentration of circulating oxygen (or of CO_2), (ii): the concentration of "essential" metabolites like glucose, (iii): the internal temperature of the body, (iv): the relative and absolute concentrations of electrolytes in the local and general circulations.

Now, of the "critical" internal physiological parameters, some will be maintained within their "normal range" by means of "built-in" internal regulation mechanisms ("homeostasis"). Such mechanisms may or may not involve neural or mechanical adjustments. Glucose concentration is partly regulated by such a built-in mechanism utilizing a hormonal channel. Respiration, similarly, is partly regulated by

neuromuscular built-in channels. So is internal temperature and water distribution. However, the built-in regulators are not, in general, capable of complete stabilization. For many of the physiological parameters, some form of interaction with the external world is essential for maintenance within normal limits. In some cases, the form of such interaction may be such that again a built-in mechanism will suffice.

In the case that the environment is not sufficiently dependable, or equivalently, if the organism is to be able to survive in a wide variety of environments, then evolution may fail to be able to produce a sufficiently versatile collection of built-in mechanisms, and another method will be required. Consider the case of thermal stabilization. The internal regulators have a limited success in regulating the body temperature, but if the level falls too far, it is necessary for the organism to find a way to make the environment provide heat. It would be valuable for such an organism to be able to learn how to recognize and exploit sources of heat. It would also be essential for the organism to be able to learn to do this only when it "is cold"; we will call this "motivated" or "drive-motivated" behavior.

6/2.3 This type of learning can be exhibited by an (M,Z,V)

system equipped with an appropriate V system. The most elementary scheme that seems acceptable is that of "drive-reduction reinforcement." Suppose that V is so constructed that the reinforcement system is activated (V is operated) whenever a physiological parameter that has been outside the "normal range" is returned to normal level. (This of course can equivalently be restricted to those parameters which do not have complete built-in regulators.) Then any reaction which precedes the restoration of normalcy will be reinforced, and this will tend toward the learning of "adaptive" behavior. (It will, of course, not always do so; preceding reactions may not have had anything to do with the restoration.)

Such a system has at least two possible defects:

(1) "Motivation" is absent; the above organism will, perhaps, learn what to eat when it is hungry, but there is no provision to make it stop eating when it is not hungry. This is a serious consideration, since when it overeats, another internal parameter will go out of range, and, by a similar process, it will learn to avoid eating. Then there will be a conflict.

(2) In many cases, e.g., in that of <u>eating</u>, the restoration of the physiological parameter to its normal value may be delayed until long after the "adaptive"

reaction has occurred. This would inactivate the learning process in systems where reinforcement applies only to very recent reactions. For a carbohydrate-dependent animal, the V-system must be activated long before the food has been absorbed into the circulation, since this may take minutes or even hours. One solution, in animals, is simple; the taste receptors of the mouth have V or reinforcement potency; the reinforcement is not of the direct "drive reduction" type of the first paragraph.*

6/2.4 The motivation problem (1) can be solved by the device of providing the main net with information about the state of the "critical" physiological parameters in the form of stimulus. Consider the effect of adding such a channel.

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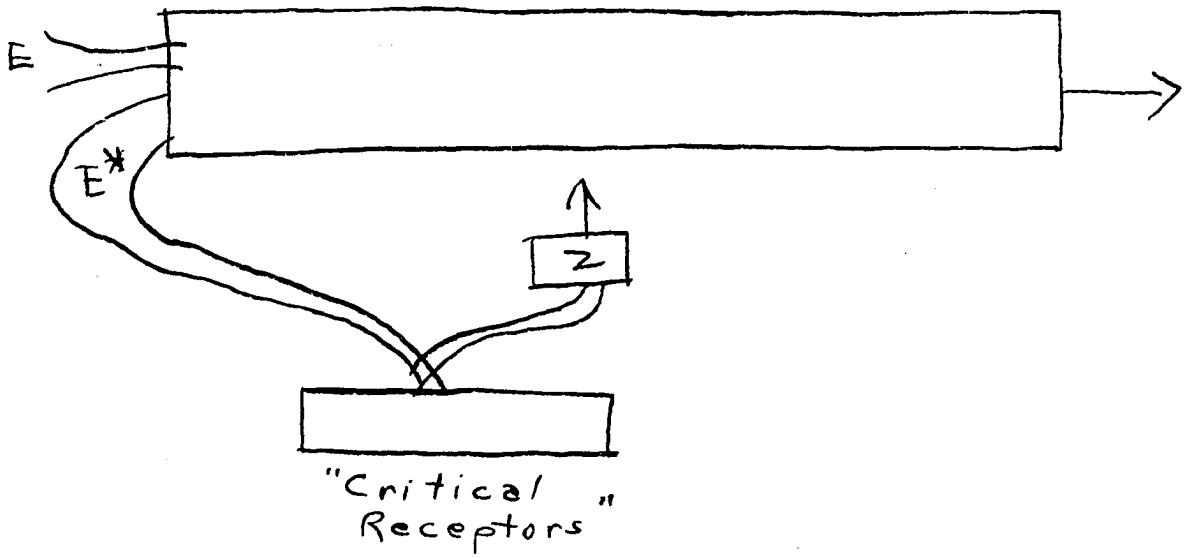
*An amusing result of the attempt to correlate physiology and learning theory is provided by the following observation. It has long been known that the salivary fluid contains a starch-hydrolyzing enzyme. The "function" of this enzyme has been long in question, since the amount of digestion that it can accomplish in the mouth is negligible; it is quickly inactivated in the acid pH of the stomach, and there is a vastly greater supply of such enzymes in the intestinal tract. What use, then, is there in having such an enzyme in the mouth? From the point of view of reinforcement theory the answer seems evident: it is there to make starchy foods taste sweet! More precisely, it is known that sweet taste, i.e., excitation of the "sweet" taste receptors of the mouth, has reinforcement power in (hungry) animals. Unhydrolyzed starch does not excite these receptors (presumably because it does not penetrate the limiting membrane of the taste receptors). But the sugar molecules which result from the hydrolysis have the power to excite these receptors!



For this system, a "stimulus" at the input of the main net will always be the sum of two components; one component S is that which comes from the environment through the "exteroceptors" of the organism, the other, S*, contains information about the internal state of the organism. Then a reaction, which <u>externally</u> appears to be of the form S_0-R_0, is(when the organism is in an internal state giving rise to the stimulus component S_0*) really a reaction with the stimulus S_0*$\cup S_0$. Thus, if this reaction is reinforced, the response R_0 will not be associated with S_0 unconditionally, but only when the organism is again in an internal state represented by S_0*. The organism will tend to use behavior which has, in the past, been successful in normalizing states like its <u>present</u> one.

The selection of critical channels for quick reinforcement is largely a matter of evolutionary

development. If the taste system is to have reinforcement capacity, and still be compatible with the "motivation" channel, then the taste system must tend to be active as a reinforcement activator only when there is a "need" for nutrients. We may expect each organism to contain a moderate number of such special devices; however for complex and sequential learning, this type of reinforcement control cannot be adequate. Reinforcement systems in which the content of V is "built-in" will be called PRIMARY reinforcement systems.

6/3 Primitive Organizations

*6/3.0 There are certain aspects in the behavior of random nets with primary reinforcement operators that may be of crucial importance in brain theory. This section will discuss these aspects informally. Unfortunately, they have not yet been analyzed in abstract form. The central idea is that of "vocabulary," or the notion that the overt part of behavior can be analyzed into a relatively small number of elements.

*6/3.1. Motor Vocabularies.

Let the model of 6/2.4 be drawn so as to represent explicitly a set of exteroceptors which have reinforcing potency.

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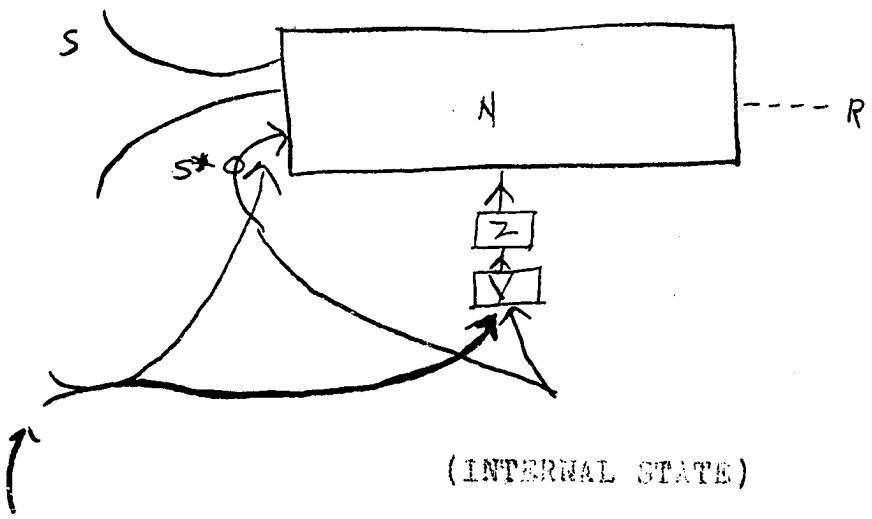
*See (*)6/1.



Consider the behavior of the system in the early stages of training; before the net contains highly organized and dependable reaction pathways. Suppose that the net is "long," i.e., that most paths from the sensory end to the motor end have a fairly large number of junctions. (Note: this assumption may have to be relaxed, perhaps by having a short net in parallel with the long one during "infancy" of the system). Then the initial S-R behavior of the net will tend to be quite random.

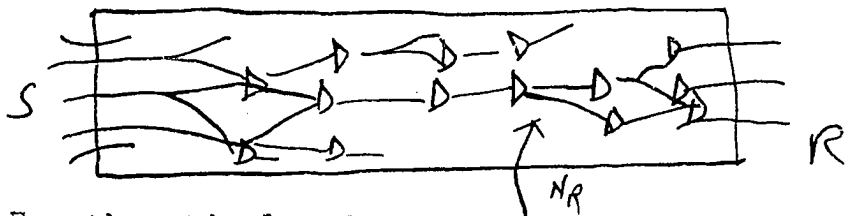
Suppose that an early reaction is S_1-R_1 (where S_1 is a pair S,S* as defined in 6/2.4).

Suppose, also, that the result of this reaction is such as to cause it to be reinforced.

In a "long" net, a single reinforcement of a reaction is not likely to make that reaction dependably reproducible. For if the reaction is mediated by relatively long chains, the failure of any junction

in the chains can cause failure of the reaction to occur. Until the net contains a collection of well-established paths, little dependability can be expected from its reactions.

In general, the pathways mediating S_1-R_1 may be expected to have the following appearance:



For the stimulus S_1 sets up activity which tends to branch and die out randomly. Looking backward from the R end of the net, it may be expected that the response R_1 can be attributed to the firing of a relatively small number of intermediate cells such as the N_R of the diagram. Each of the N_R cells will connect to the output cells through a (small) net, and these net will be reinforced when S_1-R_1 is. It is these small nets in which we are interested.

What are the conditions under which a reaction S_1-R_1 is reinforced? The basic fact must be that <u>within a short time of the occurrence of this reaction, something must occur in the environment which cause the activation of the V-system</u>. My assertion is that there is greater than chance probability that the response R_1 (to stimulus S_1) had something to do with the environmental

event which led to reinforcement. (For if this were not the case, then the motor behavior of the system would be generally incapable of influencing the environment in such a way as to bring about "desired" types of events.) To put it another way, there is greater than chance probability that the response R_1 is an action on the part of the system which can be used to manipulate the environment in a desirable way (from the point of view of V). This is a non-trivial statement. Consider, e.g., a response which consisted in the equal contraction of a pair of antagonist muscles. Such a response is not likely to cause a significant change in the environment, and is not particularly likely to be associated with reinforcement. On the other hand, a manipulative response, such as grasping an object, is likely to result in a significant environmental change. If nothing the organism does is likely to have much effect on the environment, then these arguments will not hold, but then there is little chance of the organism being able to acquire any adaptive behavior.

Now if the stimuli presented to the system are complicated, as in a retinal image, but if it also is the case that in such a complicated stimulus, only a small part is significant (i.e., that part which provides information about that part of the environment which the organism can immediately influence, e.g.,

nearby objects rather than the color of the sky) then even if the response R_1 has manipulative significance, it is not particularly likely that the pathways of the complete reaction S_1-R_1 have such significance. For many of the paths of the reaction may be excited by meaningless parts of the stimulus S_1. The organization which is reinforced at the S end of the net will tend to be rather accidental.

But the paths which get reinforced toward the motor end of the net will tend to be "meaningful." The patterns which get reinforced at this end are such that proper introduction of a few pulses at locations like N^R will cause a motor act which has manipulative significance for the environment-V combination. Thus the motor end of the net will tend to become a sort of "keyboard," where entering pulses will tend to produce "coordinated" movements. (This is the sort of activity actually encountered when the brain is stimulated close to the motor area of the cortex.)

Following the establishment of the above elementary "motor vocabulary," it can be expected that the cells of form N_R (the motor "keyboard") themselves become organized into trees whose excitation causes even more highly organized motor acts. Thus one may expect to find that a little further away from the motor end, stimulation causes more highly organized actions.

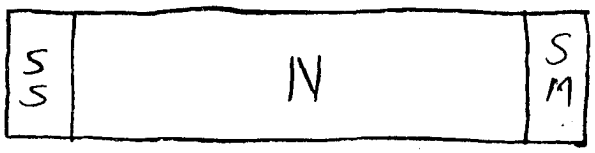
*6/3.2. In a "long" net, of the type considered above, it seems to me that until such a motor vocabulary is established, no dependable reaction structure can be developed. For without such a dependable reproducibility of simple action elements, an organized reaction would require complicated pathways extending across the net, and such a structure would be required for each reaction. Once a motor vocabulary is constructed, a few independent chains running across the net would make dependable reactions practical. I believe it could be shown that a "sensory vocabulary" will then develop, although my present arguments are weaker than those for the motor vocabulary. (The latter, I believe, could be rigorously demonstrated under general conditions. I have no doubt that formation of an "adaptive" or a "manipulatively significant" motor vocabulary occurs uniformly in the training of neural-analog nets with local reinforcement operators.)

An argument for the establishment of a sensory vocabulary can be formulated in a way parallel to the main argument of 6/3.1. Each stimulus can be separated into an "adaptively meaningful" component and an "adaptively meaningless" one. Now a given reaction S_1-R_1 is more likely to be reinforced if S_1 contains an appreciably meaningful component, because Reinforcement will occur at a slightly later time only if

something adaptively significant has occurred recently. Thus reactions are more likely to be reinforced when there is an adaptively significant event in the sensory field. The parts of reactions which depend on responses to non-significant parts of stimuli will get, almost by definition, much less regular reinforcement than responses to the significant components, and this will hold true also of the paths in the early part of the net which mediate these responses. Thus one may expect the consolidation of trees in the early part of the net also. (The arguments here are somehow weaker than those for the motor case. The above argument does not indicate that the sensory vocabulary must post date the construction of the motor vocabulary, but I feel that a more detailed analysis would show this to be the case. Weak arguments can be formulated which indicate the opposite, however.) The fact is, that stimulation of the cortex near the primary sensory areas in a developed animal indicate the presence of a sensory vocabulary. It is easy to see that there is no reason why a meaningful scheme of localization of this vocabulary should be expected, and it is not found. (In the motor areas, point stimulation yields organized movements of muscular groups associated with nearby pyramidal connections. In the areas peripheral to a primary sensory area, there is no clear and constant relation between

the location of a point and the sensation which arises on stimulation there (except as to modality!); this is not surprising; the motor areas send their output to the primary motor cells and their geometric arrangement; the sensory areas project into the entire brain, and away from the geometric arrangement of the primary sensory cortex.

6/3.3 While on the subject of sensation, it should be noted that it is almost certain that much of the so-called "sensory" and "motor" regions of the cortex is constructed in ways that cannot be assumed to satisfy the notion of "random net." It is highly probable that these areas are, ab initio, organized to some extent, and that the organism is given an important "head-start" toward the development of adaptively significant perceptual and motor organization. I had intended to devote an entire chapter to sensory special mechanisms, but this has been omitted to reduce the bulk of this paper. The presence of such special mechanisms will be represented in the diagrams by special strips at the input and output extremities of our main nets.



The special sensory (SS) strip may be regarded as con-

The special sensory (SS) strip may be regarded as con-

taining, for example, mechanisms such as those proposed

(McCulloch-Pitts 1947) for visual and auditory abstraction, and retinal contrast mechanisms, etc. The special motor (SM) strip may be considered to contain such mechanisms as the cortico-spino-cerebellar postural stabilization circuits.

Perhaps the most important consequence of the development of a pair of adaptively significant vocabularies is that behaviorally complicated reactions may be mediated through relatively small numbers of junctions. The association areas of the cortex do not need to be concerned with the control of individual muscle movements (and indeed are actually incapable on initiating such in many cases) and the information required to perform a complex act may be much less than might be expected from observation of the organism from outside.

6/4 Secondary reinforcement mechanisms.

6/4.0 All the systems treated up to now have involved a fixed, given, V-system. It is abundantly evident that no such system can ever be adequate for a theory of activity of higher animals; the class of events which cause reinforcement in higher animals is not fixed, and, in fact, definite methods are available for altering this class for most organisms.

A fairly general observation is this: A stimulus

which is frequently present at, or shortly before, the time of reinforcement itself acquires reinforcement potency. In an orderly environment, such a mechanism has great adaptive value. For those objects which are often and hence acquire reinforcement value, are likely to be just the objects which are responsible for the occurrence of reinforcement. Thus, to some extent, the objects which acquire reinforcement value, as stimulators to the exteroceptive senses, will be those which in some immediate way can satisfy the needs (as determined by the primitive, given, V-system) of the organism.

The operation of such a mechanism can transform the behavior of the system (M,Z,V) from its rather crude reactive behavior to more elaborate sequential and "partial-goal-motivated" patterns. Consider the case of eating. With a primitive V-system involving only the less discriminating senses, only actions that lead immediately to the ingestion of food are reinforced (in the hungry animal). With a "secondary reinforcement" mechanism operating, it is fairly certain that the sight of food will acquire reinforcement value, and hence the pursuit (if successful!) of food will be reinforced (again for the hungry animal! if the connections of 6/2.4 are included). If this "secondary motivation" ever becomes detached from the original internal drive,

then patterns such as "hoarding" of food can appear.

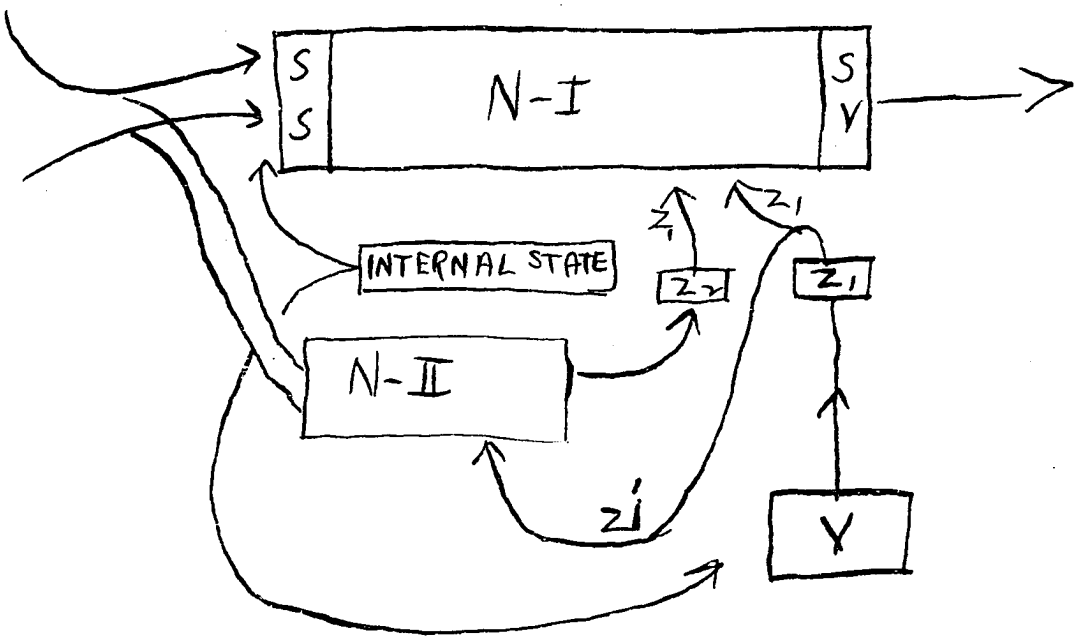
If the system is further constructed so that after a certain period (which might correspond to "infancy") the original V-system loses control over reinforcement, then we have a very flexible system. For the "secondary V" system established in this period then becomes dominant. During this period, the content of the secondary V system can be dictated from outside by appropriate systematic training, to a large extent, and the trainer thus has wide control of what will be "motivation" of the system after the "infancy" period. This systematic training can be of the general form: whenever primary reinforcement occurs, a certain object A is placed in the sensory field. Then A acquires reinforcement potency. Now the choice of A is quite arbitrary; it need not be of any value in, say, the stabilizing of the internal physiology of the system. Nevertheless, any behavior which leads to the occurrence of A in the sensory field will be reinforced (assuming that the internal state (6/2.4) at the time is one of those which occurred at times during the secondary training of A). The system will have A as a goal; pursuit of and collection of things sufficiently like A will be part of the behavior that the system will tend to acquire at later times.

A great deal is known about the establishment and

extinction of secondary reinforcers, and I will omit further discussion of its role in higher animal behavior. Let us instead see how secondary reinforcement may be incorporated into random neural-analog nets.

6/4.1 A first attempt.

Consider the system below (fig 6/4.1).



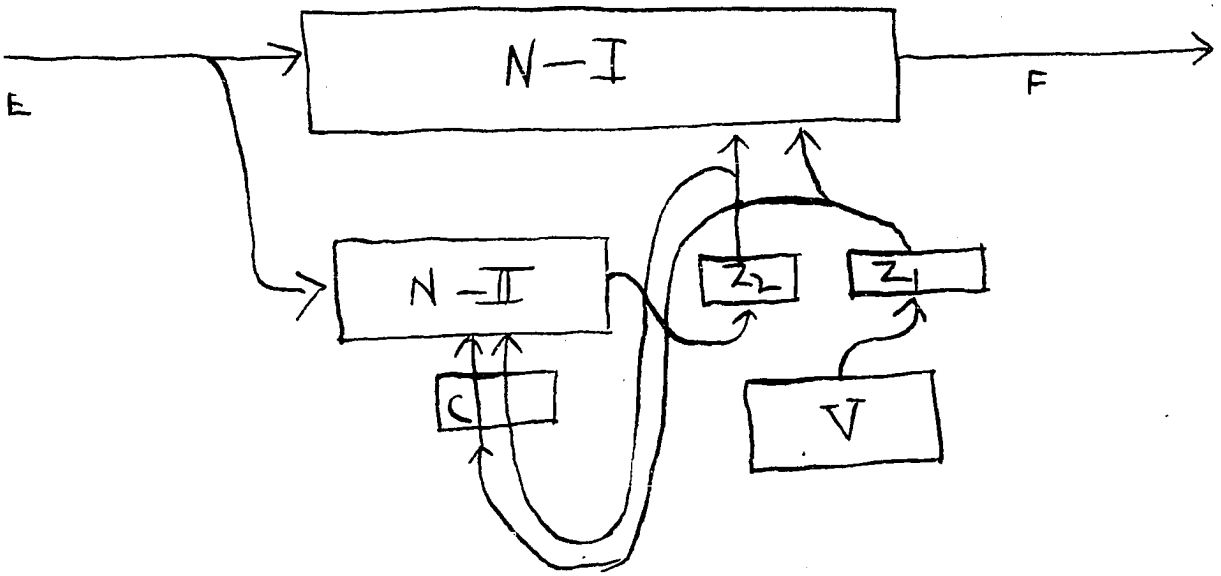
This system comes close to being a secondary reinforcement system. Stimuli present at or shortly before times of primary reinforcement cause Net N-II to react in some way, and this reaction is reinforced. Because of the way in which the output of N-II is connected, its reactions are potentially capable of controlling a second reinforcement operator (Z_2) for N-I. If the way in which Z_2 must be controlled requires non-trivial activity on the part of N-II, however, there is no guarantee that the reactions of N-II will be such as to exert control over Z_2.

6/4.1.1 There is a system of the above sort in which it is fairly certain that N-II will exert control over Z_2. Suppose that Z_2 is activated only when N-II produces at its output a signal of large (relative to its usual) magnitude. Suppose further that N-II has the property: If N-II is on many occasions presented with some stimulus S and then reinforcement is applied to N-II, <u>regardless of the particular responses of N-II to the stimulus S</u>, then the magnitude of the response of N-II to S is much increased. (It is, in fact, difficult to construct a net with a local reinforcement operator for which this is not the case.) Then it is clear that stimuli (like the above S) which frequently accompany reinforcement (by Z_1) will tend to acquire control over Z_2, and hence acquire secondary reinforcement value.

If Z_2 is of the (noise-cancellation) nature of the operators of 6/1ff, then Z_2 could be realized by a net which has the property that it emits noise <u>except</u> when it receives a large stimulus from N-II. This would not be particularly difficult to construct, especially if it were admissible to make the tract from N-II to Z_2 out of inhibitory connections.

6/4.2 A somewhat more complex, but much more general, way of insuring that N-II will learn to control Z_2 can be provided by adding a <u>feedback</u> channel. This will be

important in the event that Z_2 requires more delicate control than that suggested in 6/4.1.1.

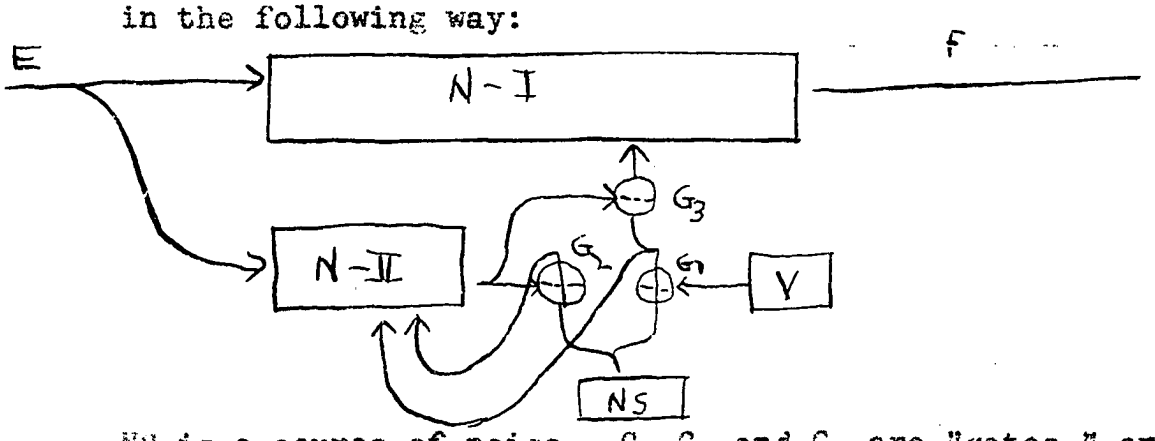


N-I, Z_1, and Z_2 are supposed to be such that application of either Z_1 or Z_2 will reinforce N-I. However, N-II, Z_1, and Z_2 are assumed to be related so that reinforcement of N-II requires the (coincidental) application of <u>both</u> Z_1 and Z_2. The square marked "C" is included to provide for this coincidence-conditional behavior in case a special device is needed.

The "feedback" connection of Z_2 with N-II has the following effect. Whatever the input requirements to activate Z_2 may be, N-II will be reinforced if and only if its reactions, during a period of concomitant primary reinforcement, are such as to meet these requirements. Hence, if there is a possibility that N-II can properly operate Z_2, the reinforcement structure, as applied to N-II itself, is such as to train N-II to

exercise such control, and to operate Z_2 as a reaction to stimuli which have been temporarily associated with primary reinforcement. ~~The net N-II may be regarded as the main net of an E,Z,V system itself; the "V" component of the reinforcement system for N-II is the assembly~~ Z_1, Z_2, V, G.

6/4.2.1 The above system can be realized using |-| nets (6/1.8) and a noise-reduction reinforcement operator L in the following way:

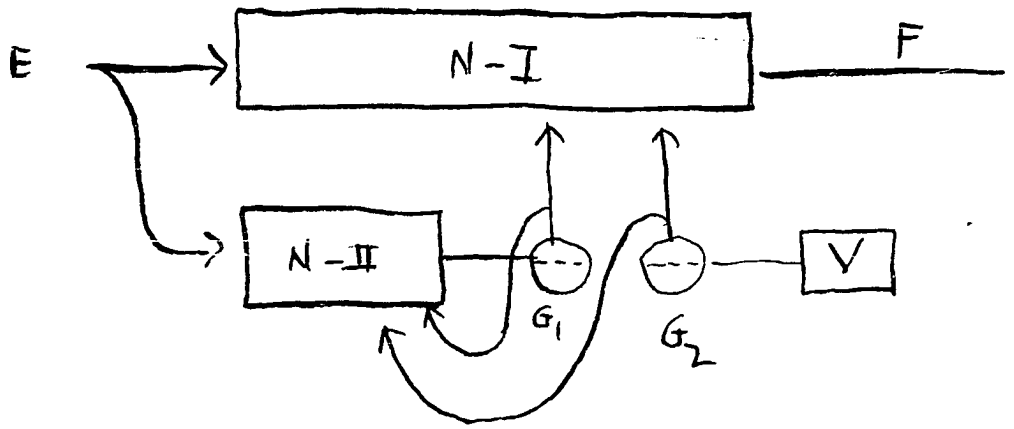


NS is a source of noise, G_1, G_2 and G_3 are "gates," or devices by which the noise channels can be interrupted. If G_2 and G_3 are operated by the same signals, then if N-I and N-II are |-|-nets, the system is equivalent to that of 6/4.2.

The system may be simplified by replacing G_1 and G_2 with units which contain their own noise source; then NS can be eliminated. NOTE: By "NOISE" we mean, of course, ANY form of activity which is efficient in extinguishing F-active patterns in |-|-nets.

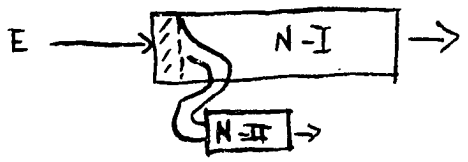
If N-I is such that relative reduction of noise acts

as a local reinforcement operator, then the system may be simplified further (provided that N-II requires greater noise reductions than N-I does):

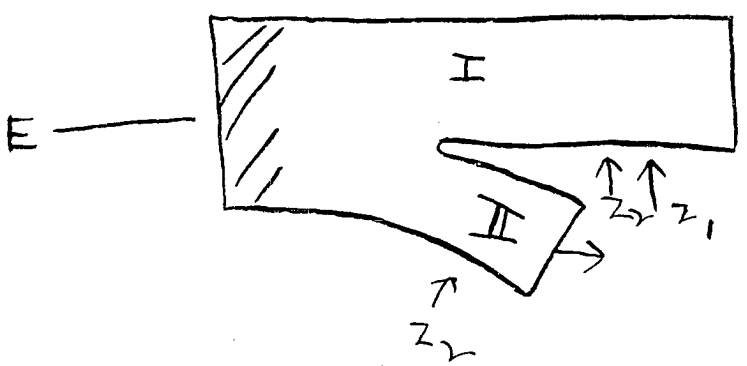


6/4.3 Content of the secondary net.

The connection schemes of the systems of 6/4.2 are inefficient to the extent that N-II does not receive the special sensory stimuli available in the early part of net N-I. It would be unlikely that the secondary reinforcement mechanisms of organisms have their own exteroceptive processing mechanisms, and a more plausible connection scheme would be:



In fact N-II could be contiguous with N-I:



In any case, we will assume that N-II has access to the same external sensory devices as does N-I. Assume, for the moment, that N-II has no motor connections.

The behavior of N-II will then be composed of simple reactions; for certain stimuli, N-II will activate Z_2. The class of such stimuli is important, for it defines the V-system for the secondary reinforcement. There are a number of important questions related to this content of N-II; the answers will be different in different systems, but some general remarks can be made.

1. Is it possible to determine the content of N-II by direct observation (behavioral) of the system?
2. Is it possible for the "goals" of the V-system directed by N-II to be contradictory to those directed by the primitive V-system?
3. Can the machine as a whole gain control over the content of N-II (in some non-trivial sense) and thus direct its own evolution?
4. What behavioral disorders would be consequences of injury to N-II?

6/4.3.1. If the only connections to N-II are those specified in 5/4.3, then the content of N-II could be determined only by prolonged observation of the behavioral time series of the system. No form of direct interrogation is possible.

If, however, the application of reinforcement is associated with any behavioral peculiarities, then a routine procedure for examination of the content of N-II should be possible. If the application of Z_2 produces distinguishable neural activity in N-I, then it might be possible to train the system so that N-I reacts to this distinguished activity as a stimulus, and the reaction response to this stimulus would be a behavioral indication of the occurrence of Z_2. In any such interrogation, motivation conditions within the organism would have to be considered, and it might be very difficult ti find out exactly which external stimulus was responsible for occurrences of Z_2.

6/4.3.2 It is easy to devise schemes in which the values of N-II are highly "non-adaptive." The trainer can establish a "dangerous" object as a secondary reinforcer by preventing injury to the system during the training. When released from the supervision of the trainer, the behavior would become non-adaptive. Any higher animal can be "domesticated" into behavior patterns absolutely incompatible with those of its primitive "drive-reduction" reinforcers (or which would be so if the organism were returned to its traditional environment.)

6/4.3.3 If N-I is able to react to the occurrence of Z_2, then N-I should be able to learn for itself the content of

N-II, if it is "sufficiently intelligent." In principle, N-I could also discover the theory of secondary reinforcement, and thus gain control over N-II, IF THERE REMAINS ANY WAY TO CHANGE THE CONTENT OF N-II after the period of "infancy." It may be the case for some organisms that secondary values acquired early are difficult to alter by any ordinary form of activity. This would be true if the original primitive Z operator were to disappear after infancy.

If a system like that of 6/4.2 were considered as part of human neural organization, it might be necessary to provide that after infancy the channels of primitive reinforcement become weak and that the secondary values dominate adult behavior.

6/4.3.4 Injury to N-II would have the interesting result of NO effect on overt behavior in any short interval. If N-II were destroyed at time t, the behavior patterns which had been acquired by N-I under its influence would remain. But no new secondary-motivated reactions could be learned thereafter.

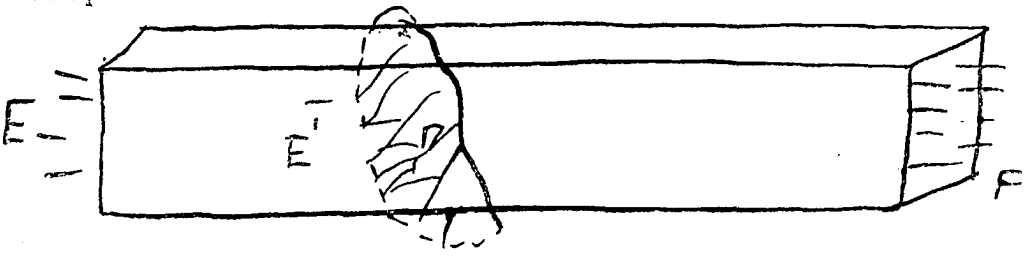
Another important question is whether the system can establish higher order forms of reinforcement, i.e., chains of values. There is no provision for this in the system of 6/4; the simple expedient of connecting Z_1 to N-II as well as to N-I will make it possible for

stimuli to acquire reinforcement value by being associated with a secondary reinforcement. Or one could add nets N-11, N-12, etc. with appropriate connections.

6/5 Internal reactive properties of nets.

6/5.1 Consider a net N, such as that of 6/2.1. This net has been regarded as a reactive system with respect to a set E of input cells, and a set F of output cells. It might be convenient to regard the net as reactive with respect to some other pair of sets of cells, even though under ordinary conditions (within some specified brain model) these sets of cells would be considered to be "inner cells" in so far as all pulses arriving at these cells come from within the net.

In particular, suppose that a surface D is passed through the net so as to separate the normal input and output channels of the net.



Let E^D be the set of fibres and/or cells that intersect this surface. Then any form of motor activity that can be induced (at F) by ("normal") stimulation at E can be evoked by removing the part of the net to the left of D and introducing the appropriate pulse pattern at E^D. If, for example, a reaction S-R can be evoked in the intact net, then the response R can be obtained

from the mutilated net by introducing at E^D a stimulus S' where S' is the activity pattern that appears on the surface D' when (in the intact net) the stimulus S is presented at E.

If it so happens that (in the intact net) the fibres crossing D all conduct in just the direction from E to F, then R can be produced by introducing S' at E' in the intact net. Thus, in the case of a net in which conduction is unidirectional, for each input stimulus S, there is, at each cross-section level D, a "pseudo-stimulus S^D which can produce the same activity to the right of D as does S. This will not, in general, be the case for nets in which conduction is not unidirectional. In particular, to the extent that secondary sensory cortices have a one-way connection from their primary sensory cortices, it should be possible, in principle, to manufacture a "pseudo-stimulus" which when applied to the secondary cortex will produce the same reactions in the rest of the system that would arise from a given external stimulus. The more the surface is crossed by loops, the less likely it will be that a pseudo-stimulus can be found for a given external stimulus.

6/5.2 Similar remarks hold for surfaces passed between the primary motor cortices and the rest of the brain. To the extent that conduction is unidirectional from the

"premotor" to the primary motor cortex, arbitrary motor acts could be induced by direct stimulation at the motor cortex. The patterns of activity at the premotor cortex could be considered to be "pseudo-motor" responses.

This discussion will be continued in 6/7.

*6/6 "Contiguity" of stimuli.

Any brain model must include a provision for some kind of "associative" learning. An analysis of this aspect of animal behavior would be out of place here; to do justice to present knowledge about the subject would require a large volume. The nets of the type considered here would seem to show some types of "associative learning," e.g., the acquisition of similar responses to "pairs of stimuli which occur together frequently."* An informal discussion of how this may come about is appropriate here, if not for its own sake, then to make more plausible the realization of the important system to be described in 6/7.

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*The term "stimulus" has been used in all previous sections in such a way that the expression indicated by quotes would be meaningless, for "stimulus" has been used to denote the total input pattern to a system, and in this sense, two "stimuli" cannot occur together. In ordinary parlance, one talks about sets of simultaneous stimuli, each originating from some part of the "total sensory field," and expressions like "total environmental situation," etc. are used to denote the total input to the system or organism. We have used capitals to denote total inputs; lower case letters will be used to denote "partial inputs" henceforth.

6/6.1 "Association" of simultaneous stimulus pairs.

Suppose that in the past experience of the system, a certain pair s, s' of stimuli have frequently occurred together. Assume also that reinforcement has been applied frequently following the stimulus, so that some response R (or better, some "sensory vocabulary element" of the sort conjectured in 6/3.2) is a dependable consequence of the stimulus s+s'. Now, assuming that the net is composed essentially of multiple threshold junctions, as is probably the case in higher animals, it is likely that at first evocation of the response R requires pulses derived from both s and s'. However after extensive reinforcement, the numerical threshold of the multiple junctions will be lowered, and s or s' separately will have greater than chance probability of evoking R.

From the point of view of association theory, it may be better not to consider the relation of s and s' to any particular R, however. Instead, consider an intercepting surface D of the type considered in 6/5.1. Let D be so close to the sensory input end of the net that pulses traverse only one or two synapses before crossing D into the main net. Now if two stimuli s and s' were such that they produce the same activity at the surface D, then their "consequences" (the distribution of internal responses of the system to the stimuli) will

be the same. It seems safe to assume that in general, the more similar are the patterns evoked at D by s and s', the more similar will be the consequences of these stimuli, and that this ought to provide some measure of a kind of "association" between s and s'. Now if we assume that a large proportion of the junctions between the input of the net and D are of high numerical threshold, then the activity evoked at D by presentation of the compound stimulus s+s' will be largely derived from junctions which require pulses from both s and s'. If s+s' occurs frequently followed by reinforcement (without reference to any particular response!) then these junctions will have lowered numerical numerical threshold and will become excitable by both s alone and s' alone. Thus (even with a random reinforcement schedule) the activity evoked at D by s and s' will become more and more similar if s+s' occurs frequently.* (The activities at D will not, in general, approach identity, however.)

6/6.2. In 6/6.1 it is indicated that stimuli which occur together may tend to acquire similar net consequences

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*It is not difficult to establish quantitative measures of the "similarization" effect discussed here; the theory has been omitted for brevity. A basic result is that the higher the numerical threshold of the net junctions, the more marked is the similarization effect, in simple nets containing one or two layers of unidirectionally oriented, but otherwise random connections.

(and in some sense should accordingly acquire related "meaning"). Another form of association that might be desirable is the following: suppose that s is frequently followed by s', and with a constant delay. Then, s' will often occur at times when a pseudostimulus s^D (6/5.1) of s is in the interior of the net. Then by a mechanism parallel to that of 6/6.1, s' and the pseudostimulus s^D of s will tend to acquire similar consequences and in effect s^D will become like a pseudostimulus of s'. This "sequential association" will be discussed further in 6/7; it will be seen that the occurrence of s will, in a certain sense, set up an "expectation" of s'.

6/7 Prediction

6/7.0 A stochastic neural-analog net is, in general, capable of many responses to a given stimulus, depending on its internal state, and beyond this, on a probabilistic basis. The adaptive capacity of a system depends on its ability to exploit this reactive flexibility. The following scheme seems (introspectively) an important aspect of higher intellectual processes:

> Given an environmental situation, one "contemplates" a particular "potential" action. One then, in some way, makes an estimate of the consequences of that action, i.e., of what changes would occur in the

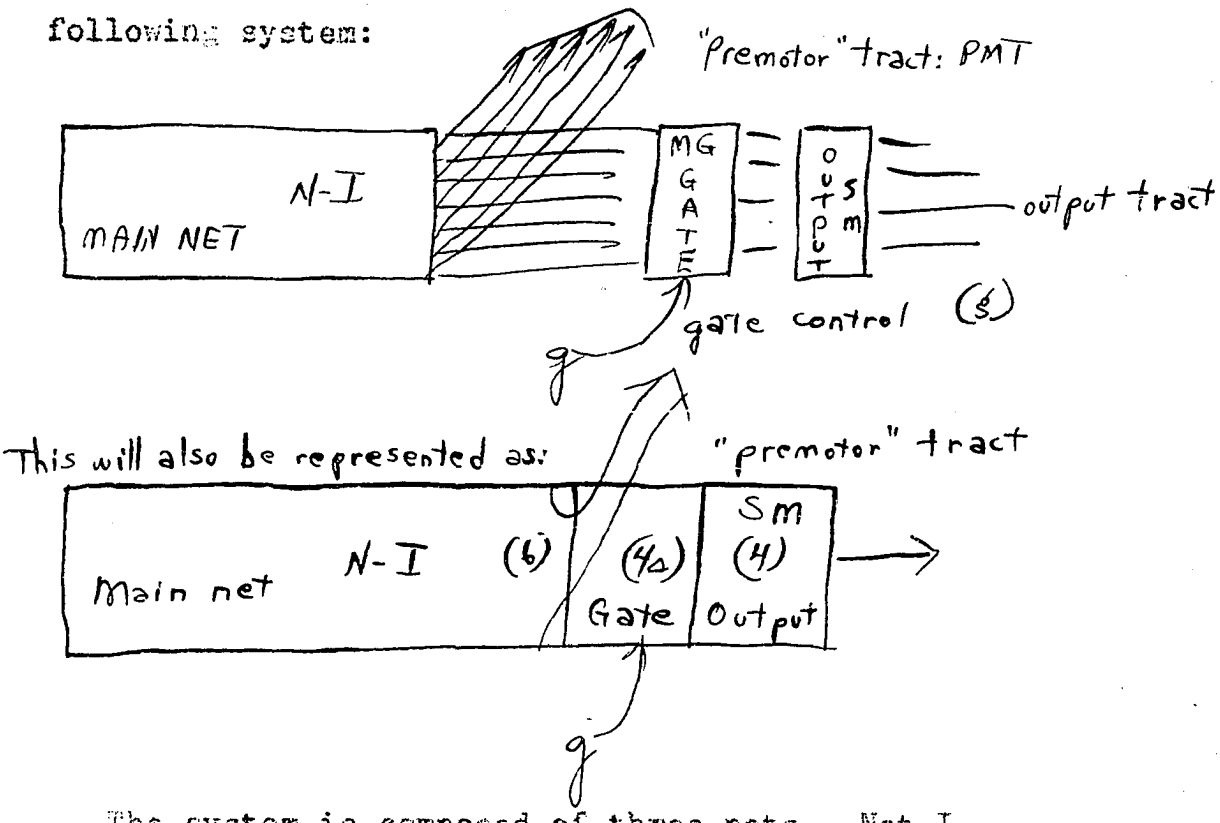
external situation were the contemplated action made overt. Then a valuation is made on this estimate; if the estimated consequences are deemed acceptable, the action is performed, if not, the action is rejected, and then a different action is contemplated.

In this section a system of random nets is described, and an attempt is made to show that the behavior of this system will conform, to some extent, to the above scheme. (It is necessary to enlist the sympathy of the reader in connection with the use of some terminology of introspective psychology. If the reader rejects the possibility that a net may have an attribute like "foresight," let him interpret the arguments as attempting to show merely that the system may act "as though it has this attribute.")

6/7.1 Motor gates.

The first property required of the system is that it have in some sense the capacity to "consider" a response before actually translating it into action. How this can be done is indicated briefly in 6/5.2. A surface is drawn cutting across the net near the output tracts. If this surface is replaced by a "gate" which can be opened and shut to pulse patterns, then this gate can be used to control whether or not an overt

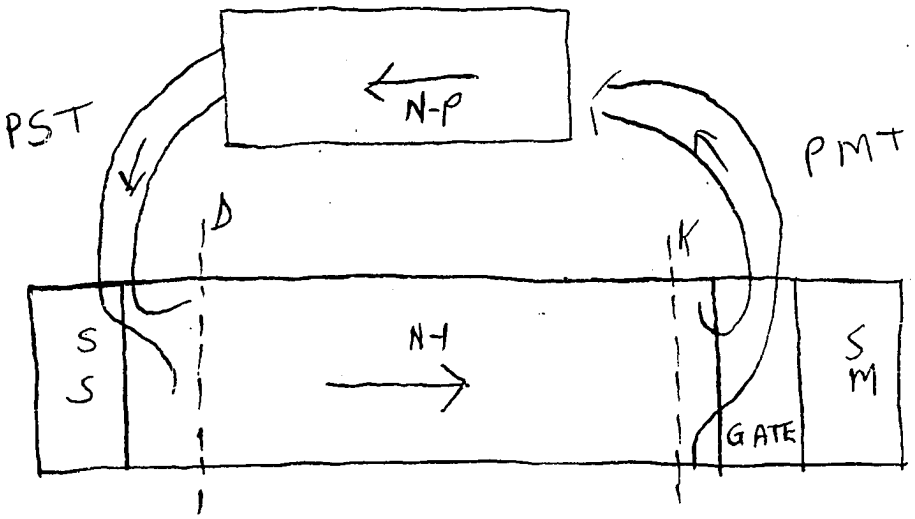
action will take place at any time. Consider the following system:



The system is composed of three nets. Net I corresponds to the N-I of earlier sections of this chapter, except that the final output tract is removed. Two tracts emerge from the right of Net-I; one of these, PMT, will be unterminated for the moment, the other goes into a special net which we call the "motor gate" MG. (The two tracts emerging from the right of N-I are supposed to have the same origin, and contain equivalent information.) MG is, as its name implies, a device which can either stop pulses, or transmit them to the final "output net." The gate is controlled through a channel G by a signal 'g'; if g(t) = 1 the gate is open (transmits), if g= 0 the gate is closed (blocks pulses).

Now if the channel from N-I to MG conducts only in that direction, then every motor act must be preceded by activity at the right end of N-I, and information about this activity will be carried along the accessory tract PMT. If the gate MG is closed, then there can be no overt motor action, but activity may still appear at tract PMT. Such activity may be regarded as potential or non-overt action, which, if the gate were opened, would become overt. If the scheme of 6/7.0 is to be realized, there must be some way of using the output of PMG as an input to some kind of PREDICTOR mechanism which is to make an estimate of the consequences of the potential action.

6/7.2 The predictor is to make an estimate of the changes that would result in the external situation if the potential action is performed. It would seem natural to arrange the system so that the output of the predictor produces a pseudo-stimulus (6/5) representing the changed situation. (Presumably, the pseudo-stimulus would be incomplete and/or the system would have other ways of knowing that the action had not yet been performed.) Thus the predictor could be inserted as shown (Fig. 6/7.2)



The predictor net has PMT as input; its output tract, PST, enters into the main net close to the input end of that net and spreads out over a region D of the sort discussed in 6/5.1. The job of the predictor net N-P is to construct a function which, given the "contemplated" action at PMT as domain, has as its range a set of patterns which act as pseudo-stimuli for the consequences of these actions. This may seem like an unattainable requirement, since it may seem to imply that N-P contains information about the reactive properties of the environment. (Note: it is true that N-P may have to be supplied with information about the present state of the environment; this can be included by allowing PST to transmit information in both directions. Nevertheless, it appears that it may be sufficient to let N-P be a random net, and PMT and PST random connections to the appropriate regions of N-I!

6/7.3 Behavior of the system with a random net for N-P.

6/7.3.1 Consider first the case of actions which usually result in definite sensory consequences. Now it is clear that in order for a particular action (i.e. pattern of muscular movement) to have a definite sensory consequence, there must be a corresponding regularity in the reactive properties of the environment. There is one class of such regularities quite independent of most ordinarily variable features of environment: if we regard the physical body of an animal to be part of the environment of its nervous system then all actions which produce stimuli by interactions between parts of the body have highly regular consequences. A simple example in man is that the act of contracting the flexor muscles of the hand will (almost always) cause a tactile stimulation of receptors in the palm and fingertips. (Any muscle contraction will produce dependable excitation of related joint and tendon receptors.) What does this mean in terms of the internal behavior of the system of 6/7.2?

Let R* be any activity pattern at K (in the "premotor" area) which, if the gate were opened, would result in closing of the hand. Suppose that R* occurs often, and is followed by reinforcement, and that the gate is in fact opened on many of these occasions. Now R*(t) is an input to N-P, hence (assuming that N-P does not have any

learning properties!) N-P will react producing at PST a pattern (or more likely a probability distribution of patterns) P(R*)(t+1). (The time quantization is used for convenience only). Now let s be the stimulus representing contact with palm and fingertips. Then s and P(R*) occur together frequently, and will be "associated" in the manner described in 6/6.1. It may then be said that the net N-P becomes modified in such a way that P(R*) becomes a pseudo-stimulus for s. In this simple manner, it may be said that N-P becomes a predictor. For even if the gate is closed when R* occurs, the pseudo-stimulus P(R*) goes through, and a "pseudo-sensation" of hand-closing is experienced by the system.

The same arguments apply to more complicated environmental regularities; motion of the hand will become associated with the visual stimulation consequent to the motion of the hand across the visual field, etc. It seems to me that these regularities consequent to physical body structure are rather well-distinguished, by their dependability, from other interactions with most environments, and that through such a predictor mechanism a "body image" can be constructed.

6/7.3.2 The system as described is not adequate for prediction of environmental events which do not depend only on the

immediate motor activity of the system. The system should be able to predict the behavior of the environment W where W has the form indicated in 4/3.2.2 or exhibits regularities of this form. (i.e., where the change of state of W depends on the activity of the system even if the state itself does not.) To some extent this can be accomplished by allowing the tract PST to transmit information both ways, or otherwise providing N-P with an input from the sensory end of N-I. Then stimuli for N-P will have the form $s^* R^*_i$ where s^* is the image at D of a stimulus s, and R^*_i is one of the set of possible responses at K to the stimulus s. If the gate is open, R^*_i results in an overt action which induces a transition in the state of W and produces a new stimulus s'_i $W(s,R^*_i)$ (See 4/3.2.2, (1) and (2) for notation, and overall system. The time indices have been dropped.) This s'_i evokes an image s'^*_i at D, and this is accompied by the arrival from N-P of the stimulus $P(s^* R^*_i)$. If this occurs frequently with reinforcement (random reinforcement will do) then, if the contiguity mechanism operates correctly, $P(s^* R^*_i)$ will become a pseudo-stimulus for s'_i, and N-P becomes a predictor for this type of environment.

6/7.3.3 Allowing N-P to have learning properties yields a much more complicated system. Superficially, it might

appear that "good prediction" would tend to lead to positive reinforcement situations, and hence the learning capacity ought to tend to improve N-P in its prediction role. However, there is the danger that the prediction capacity of N-P is too delicate to tolerate internal alterations, and that if N-P is alterable, it might become absorbed into the simple reactive structure of N-I.

On the other hand, it is perhaps worth noting that <u>if</u> N-P can have the same kind of structure as does N-I, then there is no need to delineate N-P as a distinct structure. For in this case N-P may be nothing more than a distinguished subnet of N-I; for example N-P might be simply the subset of cells and fibres of N-I which conduct pulses primarily from right to left!

6/7.4 The discussions of 7/3 indicate how the system may "estimate the consequences of an action"; it does not complete the scheme of 6/7.1. Nothing has been said about closure of the gate, and what is to control this. Now this gate is to be controlled by a device V* which acts by evaluating the output of N-P but this output is meaningless except in relation to the pseudo-stimuli it produces at D. Hence, it would seem that the evaluator V* has to have its input taken from D or later in net N-I. If this is the case then the evaluator cannot discriminate between real and pseudo-stimuli. This means

that if a certain action consequence is estimated, action taken, and the predicted consequence realized, then the gate will remain open and the following pattern at K will go through to action. I do not see any particular disadvantage to this except that the ability of the system to evaluate potential actions is suspended for a short interval after a successful action.]

Nothing has been said about the nature of the action evaluator V*. One possibility is to use the output of the net N-II of 6/4.3 to control directly the motor gate MG. (Note that the output of the N-II of 6/4.2 will not work since the input to this N-II does not have access to the pseudo-stimulus output of net N-P.) The gate MG must have characteristics opposite to those of the gates of 6/4.2.1 in that signals from N-II which have a positive evaluation should open MG (while in 6/4 such signals closed gates G_2 and G_3.) If this is done, then V* is N-II plus the connection from N-II to MG. For this V*, the evaluations on potential actions will be consistent with the goal-structure of the secondary reinforcement system. The system is admittedly incomplete in that there probably must be other ways of opening gate MG.

*6/7.5 The appearance of a pseudostimulus at D should have a special effect on the following reactions of the

system. Suppose that s' is a pseudostimulus of s and appears at D just before a stimulus s-, an incomplete fraction of s, is presented to M-I. Now s' will (by its definition as a pseudostimulus of s) send pulses to many of the junctions that would normally be excited by s, and s- will do the same. Hence the response to s- can be expected to resemble the ordinary response to s to a greater extent than would be expected if s' were not present. Another way of putting it would be to say that in the presence of s' the system will tend to react to parts of s as though all of s were present, or the system appears predisposed to react to "weak" presentations of s. It could be argued that the system might be described as being in a state of "expectation" of s.

It would be futile to analyze this aspect of the system without a preliminary analysis of sensory mechanisms, a subject which has been excluded from this work, for reasons mentioned in chapter I.

6/8 Remarks

A complete discussion of the biological evidence for the operation of the systems discussed in this chapter would carry this work beyond reasonable limits of size.

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* For the convenience of non-biologists, the physiological references were confined to this textbook.