On the Dynamics of Operant Conditioning

STEPHEN GROSSBERG

Department of Mathematics,
Massachusetts Institute of Technology, Cambridge, Mass. 02139, *U.S.A.*

Simple psychological postulates are presented which are used to derive possible anatomical and physiological substrates of operant conditioning. These substrates are compatible with much psychological data about operants. A main theme is that aspects of operant and respondent conditioning share a single learning process. Among the phenomena which arise are the following: UCS-activated arousal; formation of conditioned, or secondary, reinforcers; a non-specific arousal system distinct from sensory and motor representations whose activation is required for sensory processing; polyvalent cells responsive to the sum of CS and UCS inputs and anodal d.c. potential shifts; neural loci responsive to the combined effect of sensory events and drive deprivation; "go"-like or "now print"-like mechanisms which, for example, influence incentive-motivational increases in general activity; a mechanism for learning repetitively to press a bar which electrically stimulates suitable arousal loci in the absence of drive reduction; uniformly distributed potentials, driven by the CS, in the "cerebral cortex" of a trained network; the distinction between short-term and long-term memory, and the possibility of eliminating transfer from short-term to long-term memory in the absence of suitable arousal; networks that can learn and perform arbitrarily complex sequences of acts or sensory memories, without continuous control by sensory feedback, whose rate of performance can be regulated by the level of internal arousal; networks with idetic memory; network analogs of "therapeutic resistance" and "repression"; the possibility of conditioning the sensory feedback created by a motor act to the neural controls of this act, with consequences for sensory-motor adaptation and child development. This paper introduces explicit minimal anatomies and physiological rules that formally give rise to analogous phenomena. These networks consider only aspects of positive conditioning. They are derived from simple psychological facts.

1. Introduction

This paper begins a series which will study problems of operant conditioning from a theoretical point of view. The experimental literature on this subject is vast and growing rapidly. Here are presented simple psychological

assumptions—most bordering on the psychologically trivial—that can drive deeply and widely into this literature. These assumptions also suggest underlying physiological and anatomical substrates of the psychological facts.

The results form part of a rigorous theory of discrimination, learning, memory and recall known as the theory of *embedding fields* (Grossberg, 1969*a–f*, 1970*a,b*, 1971). This psychological theory has a physiological, anatomical, and biochemical interpretation. Until the present research was undertaken, most learning in embedding fields was of respondent type. It was found, however, that even respondent conditioning could not be carried out effectively in a complex environment unless suitable precautions were taken, especially in constructing the anatomy of embedding field networks. As these precautions were implemented, it was gradually realized that the prerequisites for effective respondent conditioning are remarkably similar to the prerequisites for operant conditioning. The results, which seem mathematically inevitable, are also compatible with much data and heuristic theorizing by other authors. This paper presents simple facts about the respondent conditioning paradigm from which explicit networks can be derived. References are cited to show that various stages of network processing have clear-cut analogs in experimental data.

An example of an embedding field will be defined below for completeness. This example was itself derived from simple facts about respondent conditioning (Grossberg, 1969*a*). It has also been extensively analysed mathematically (e.g. Grossberg, 1969*d*). Further examples will be referred to in the text. Only the heuristic meaning of following equations will be needed herein:

$$\dot{x}_i(t) = -\alpha_i x_i(t) + \sum_{m=1}^{n} [x_m(t-\tau_{mi}) - \Gamma_{mi}]^+ \beta_{mi} z_{mi}(t) + I_i(t) \qquad (1)$$

and

$$\dot{z}_{jk}(t) = -\gamma_{jk} z_{jk}(t) + \delta_{jk}[x_j(t-\tau_{jk}) - \Gamma_{jk}]^+ x_k(t), \qquad (2)$$

where $[w]^+ = \max(w, 0)$ for any real number w, and $i, j, k = 1, 2, \ldots, n$. These equations have the following interpretation. Let n cell bodies (or cell body clusters) v_i be given with average potential $x_i(t)$, $i = 1, 2, \ldots, n$. If $\beta_{mi} > 0$, then an excitatory axon e_{mi} leads from v_m to v_i. Denote the synaptic knob of e_{mi} by N_{mi}. $z_{mi}(t)$ will be called the excitatory chemical transmitter activity in N_{mi}. Alternative interpretations of $z_{mi}(t)$ are possible, but they are strongly constrained by (1) and (2).

The spiking frequency which is created in e_{mi} in the time interval $[t, t+dt]$ is proportional to $[x_m(t) - \Gamma_{mi}]^+ \beta_{mi}$. The time lag for a signal to flow from v_m to N_{mi} is τ_{mi}, and the spiking threshold of e_{mi} is Γ_{mi}. When the signal from v_m reaches N_{mi} at time t, it causes release of excitatory transmitter

into the synaptic cleft facing v_i at a rate proportional to

$$[x_m(t-\tau_{mi})-\Gamma_{mi}]^+\beta_{mi}z_{mi}(t),$$

whence the rate of change of x_i increases proportionately. All excitatory signals combine additively at v_i, yielding the second term on the right-hand side of (1). The potential $x_i(t)$ also decays exponentially at the rate α_i, and is perturbed by known inputs $I_i(t)$ that are controlled by an experimentalist or independent cells.

The transmitter production process is regulated by cross-correlation of presynaptic spiking frequencies and postsynaptic potentials; for example, $z_{jk}(t)$ in (2) cross-correlates the presynaptic signal $[x_j(t-\tau_{jk})-\Gamma_{jk}]^+\beta_{jk}$ with the postsynaptic potential $x_k(t)$, yielding the term $\delta_{jk}[x_j(t-\tau_{jk})-\Gamma_{jk}]^+x_k(t)$.

Speaking psychologically, $x_i(t)$ is the *stimulus trace* of v_i, and $z_{jk}(t)$ is the *memory trace* (or *associational strength*) of the association $v_j \rightarrow v_k$. The equations can be generalized substantially without destroying basic properties of learning (Grossberg, 1969d). This added generality is not physically inessential, however, as it allows for complex patterns of spiking frequencies (relative and absolute refractory periods, bursts), different time lags and thresholds for signals to pass from v_i to N_{ij} than through N_{ij} to v_j, a variety of decay laws for transmitter that influence the extinction of memory in dramatic ways, etc.

2. Synchronization by UCS-activated Arousal in Respondent Conditioning

It has recently been observed (Grossberg, 1969e, 1970a) that an arousal mechanism, activated by the UCS, and mingling with the CS at prescribed cells, is often needed to guarantee efficient respondent conditioning. By carrying this observation to its limit, we will find that formal prerequisites of even simple respondent conditioning tasks look remarkably like prerequisites for operant conditioning. Three simple assumptions will drive us to the concept of a UCS-activated arousal: (i) the trivial fact that CS and UCS can be separated by different time lags on different learning trials; (ii) the trivial fact that, under sufficiently simple learning conditions, the more we practice a task, the better we learn it ("practice makes perfect"); (iii) the assumption that memory is encoded in synaptic knobs (or at specific postsynaptic sites adjacent to these knobs) by a mechanism that cross-correlates presynaptic spiking frequency and postsynaptic potential (or processes in parallel with these). The following paragraphs show how conditions (i) to (iii) impose the concept of arousal.

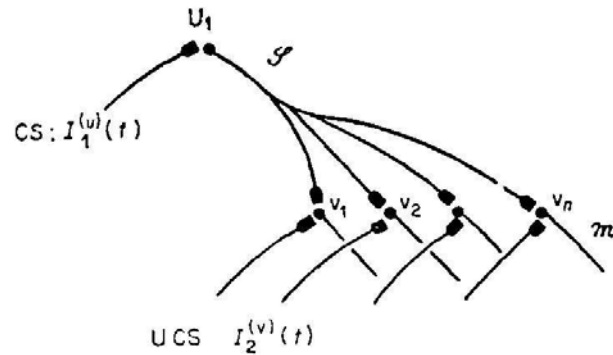Consider the idealized network of Fig. 1 for specificity.

Fig. 1.

Let the CS create the input $I_1^{(u)}(t)$ at the cell U_1, and let the UCS create the inputs $I_i^{(v)}(t)$ at the cells V_i, $i = 1, 2, \ldots, n$, respectively. The collection of cells V_i, $i = 1, 2, \ldots, n$, will collectively be called $\mathscr{M}$ for "motor representation." U_1 will occasionally be denoted by $\mathscr{S}$, for "sensory representation". For simplicity, let U_1 send axons to all cells in $\mathscr{M}$, let all synaptic knobs of these axons receive the same spiking frequencies from U_1, and let all cells in $\mathscr{M}$ have the same local parameters.

Suppose that we pair the CS and the UCS inputs sufficiently often. What is the most general UCS that a future presentation of the CS alone can perfectly reproduce in the $\mathscr{M}$ outputs?

The answer is a spatial pattern; namely, a UCS for which each $I_i^{(v)}(t)$ has fixed relative intensity during learning trials. Such a UCS can be written in the form $I_i^{(v)}(t) = \theta_i J(t)$, where θ_i is the relative intensity of the input to V_i. By letting $\theta_i \geqslant 0$ and $\sum_{k=1}^{n} \theta_k = 1$, $J(t)$ becomes the total pattern intensity at time t (Grossberg, 1969e, section 5).

Suppose that a spatial pattern UCS with weights $\theta = (\theta_1, \theta_2, \ldots, \theta_n)$ follows the CS by a time lag of T_j on the jth learning trial. In principle, the T_j's can be any non-negative numbers, since the environment can present the CS and the UCS at independently chosen times. Because of this, practice on successive trials can disrupt rather than facilitate learning unless further precautions are taken. To illustrate this possibility, let a distinct pattern $\tilde{\theta}$ follow θ on each trial. In particular, if $\mathscr{M}$ directly controls the activation of a large number of muscle groups (e.g. $n \gg 1$), then more than one pattern can surely reach $\mathscr{M}$ from time to time.

The condition to be avoided is the following: that the CS *alone* can cause U_1 to fire, and thereby to send signals to its synaptic knobs. Were this possible, we could choose T_j on successive trials so that the synaptic knobs of U_1 are active when θ is at $\mathscr{M}$ on some trials and when $\tilde{\theta}$ is at $\mathscr{M}$ on other trials. The knobs will therefore learn a noisy mixture of the two patterns θ and $\tilde{\theta}$, rather than θ (Grossberg, 1970b).

To avoid this difficulty, we require that U_1 will fire only if (i) the CS is being presented, and (ii) the UCS will arrive at $\mathcal{M}$ a fixed time after U_1 begins to fire. We also require that (iii) the duration of U_1 firing will be brief. Consider condition (ii). The time lag between onset of U_1 firing and arrival of UCS-created inputs at $\mathcal{M}$ is fixed so that U_1 will practice θ on successive trials. The UCS arrives at $\mathcal{M}$ only after U_1 fires so that U_1's synaptic knobs can be active when θ arrives at $\mathcal{M}$. Condition (iii) is imposed so that only θ is practiced, and not also later patterns such as $\tilde{\theta}$. Some mechanisms for guaranteeing condition (iii) have been discussed, using, for example, non-recurrent inhibitory interneurons (Grossberg, 1969e, section 12; 1970a, section 3).

A simple mechanism that satisfies (i) and (ii) is readily suggested in two steps; it is not, however, the only mechanism. A disinhibition mechanism is for some purposes more useful, but harder to understand at the outset.

Step (i)

Let both the CS and the UCS create inputs at U_1 via axons to U_1. Let these inputs combine additively at U_1. The UCS now sends inputs both to $\mathcal{S}$ and to $\mathcal{M}$. Let the UCS $\rightarrow$ $\mathcal{S}$ input arrive a fixed time earlier than the UCS $\rightarrow$ $\mathcal{M}$ inputs. This fixed delay can easily be built into the axonal geometry.

Step (ii)

Choose the U_1 firing threshold Γ_1, once and for all, so large that the potential $x_1(t)$ of U_1 can be driven above Γ_1 to initiate axonal firing only if CS and UCS inputs arrive at U_1 almost simultaneously and with sufficient intensity.

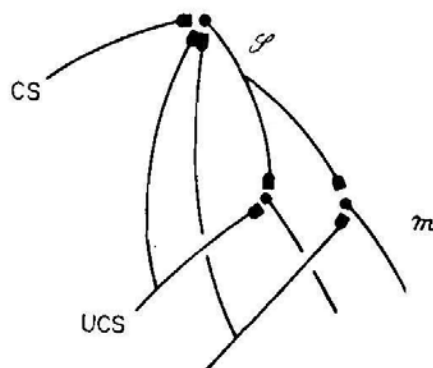The simplest anatomy compatible with this idea is given in Fig. 2.

FIG. 2.

The input UCS → $\mathcal{S}$ is an *arousal* input because it reaches U_1 before the corresponding UCS → $\mathcal{M}$ input arrives: it prepares the sampling cell U_1 for events that are about to follow. The input UCS → $\mathcal{S}$ is also a *non-specific* input since it must be delivered to every CS-activated cell, such as U_1, which can ever sample $\mathcal{M}$. See Fig. 3.
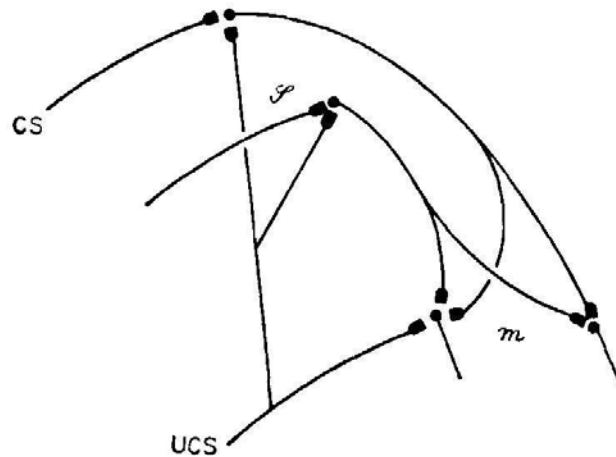

FIG. 3.

The anatomy of this non-specific system in fact is much more complicated than Fig. 3 suggests. Later paragraphs begin to show this.

In summary, the existence of "spatially coded" regions, such as the synaptic knobs, capable of practicing specific tasks on successive trials to guarantee perfect learning, suggests the existence of a non-specific arousal input controlled by the UCS, and mingling with the CS at sampling cells. Each sampling cell might itself be activated only by a narrowly delimited class of CS inputs, and therefore is part of a specific system that filters environmental events (Grossberg, 1970a).

3. Experimental and Theoretical Correlates of UCS-activated Arousal

The reader who wishes to study the conceptual argument without interruption can omit this section. Below are listed some results that are relevant to the above conclusions. The list is incomplete, but might help the reader bridge the gap between our discussion and the large literature to which it relates. It will be useful below to keep in mind the simple postulates that underly our conclusions, and which possibly can form a unifying framework and teleological rationale for many other results; namely, that practice makes perfect, that CS and UCS can be separated on different trials by different time lags, and that cross-correlation of pre- and post-synaptic activity occurs at specific cellular sites.

(A) NON-SPECIFIC AROUSAL

The non-specific arousal system synchronizes firing of signals from $\mathscr{S}$ and arrival of UCS-created signals at $\mathscr{M}$. Without it, the cells in $\mathscr{S}$ cannot fire. Moruzzi & Magoun (1949) have reported the existence of a non-specific projection system from the brain stem to the cerebral cortex whose arousal makes organized cortical activity possible. The system $\mathscr{S}$ becomes a rudimentary analog of the cortex in this analog.

(B) CUE AND AROUSAL FUNCTIONS

To reconcile the demands of data, Hebb (1955) has suggested that a sensory event has two quite different effects: its *cue function* and its *arousal* or *vigilance function*. The cue function guides behavior, the arousal function energizes it. Hebb also suggests that learning without arousal is not possible. The two categories of cue function (e.g. $CS \rightarrow \mathscr{S}$) and arousal function (e.g. $UCS \rightarrow \mathscr{S}$) have clear analogs herein. Moreover, no learning occurs without arousal, since then $\mathscr{S}$ cannot fire and no sampling of $\mathscr{M}$ occurs. Note however, that the cue function and the arousal function thus far reside in *different* sensory events. This will no longer be the case after the next section. We will also find that the cue function and arousal function are not independent. An external sensory input can have a cue function that has arousal functional properties at internal drive representations, and an internal drive input can have a cue function that has arousal functional properties at external sensory representations.

(C) POLYVALENT CELLS

$\mathscr{S}$ contains polyvalent cells, or cells influenced by several modalities. For example, suppose that CS = (a tone) and UCS = (visual presentation of food). Then both auditory and visual inputs are needed to fire the cell U_1. U_1's response is, moreover, dependent on the algebraic sum of CS and UCS inputs, and its pattern of firing influences the plastic network changes that occur during conditioning. John (1966, 1967) has extensively discussed cortical cells of this general type.

(D) D.C. POTENTIAL SHIFTS

Such workers as Rusinov (1953) and Morrell (1961) have shown that anodal d.c. potential shifts augment classical electrocortical conditioning, whereas cathodal d.c. shifts tend to have an opposite effect. These results suggest that anodal d.c. shifts have effects on cortical firing that are analogous to facilitatory arousal.

(E) INVERTED U IN PERFORMANCE

A precise control of arousal level is required in our networks. *In vivo*, the so-called "inverted U in performance" describes an analogous optimal intermediate level of arousal that sufficiently energizes performance without decomposing it (Hebb, 1955; Kornetsky & Eliasson, 1969; Phillips & Bradley, 1970). Two recent theoretical papers (Grossberg & Pepe, 1971*a,b*) prove the existence of such a region in an analogous model of serial learning, and discuss the influence of varying the arousal level, or spiking threshold size, on skewing of the bowed curve, primacy vs. recency, and associational span. These results can be interpreted to mean that over-arousal contributes to poor attention due to massive response interference. Related clinical data are also cited.

(F) MEMORY READOUT

In a general discussion based on his experiments, John (1966, p. 183) has noted that

> "there must be a readout from memory, and somehow the readout must be in the same coin as the readin to memory. . . . Some place the code for what is stored and the code for what is happening at present must come into a common informational domain for the transactions which constitute remembering to take place."

Such properties are evident in our systems, and have been studied mathematically under rather general conditions of readout and readin; namely, if any number of space–time patterns of essentially arbitrary complexity are being learned, remembered and performed (Grossberg, 1969*d,e*, 1970*b*).

4. Conditioned (or Secondary) Reinforcers: Is Arousal also Conditioned during Respondent Conditioning of Specific Tasks?

Consider the following two basic questions.

(i) If U_1 can fire during learning trials only when inputs from both CS and UCS are received, how does U_1 fire during recall trials when only the CS is presented?

(ii) The UCS in a given learning experiment might have been only a CS in a previous learning experiment. By section 2, every UCS controls an arousal input. How does a sensory event gain control over an arousal input as it passes from its past role as CS to its future role as UCS?

Question (i) is based on the trivial facts that the UCS occurs only during learning trials, and that performance is still possible during recall trials. Question (ii) is based on the much deeper fact that conditioned (or secondary) reinforcers exist (Kelleher, 1966). Remarkably enough, both questions can be given a common answer.

Consider question (i). During recall trials, performance *is* possible. Hence U_1 *does* fire during recall trials. U_1 cannot fire unless it receives the usual CS-created input $I_1^{(u)}(t)$ *and* the arousal input. During recall trials, the UCS is not available to create the arousal input at U_1. Only the CS is available to do so. Hence, as a result of pairing CS and UCS on learning trials, the CS can activate the arousal input on recall trials. The CS also maintains its ability to create the input $I_1^{(u)}(t)$ at U_1. Thus, on recall trials, U_1 receives the input $I_1^{(u)}(t)$ as well as the arousal input, and consequently fires.

Two processes occur in parallel during respondent conditioning: the CS (via U_1) samples the cells $\mathcal{M}$ which control the UCR; the CS also samples the cells which control internal arousal. Once the CS controls both inputs, it satisfies the criteria of section 2 characterizing the UCS. We have hereby given an answer to question (ii) by answering question (i).

Our rationale for the existence of conditioned reinforcers is based on one simple assumption about the learning mechanism and on three trivial facts of life (practice makes perfect, the time lags between CS and UCS onset can differ on different trials, and the UCS does not occur on recall trials). Without conditioned reinforcers, an organism's behavior could not evolve much beyond the most primitive reflexes and primary drive satisfactions. It is therefore highly instructive that an organism without conditioned reinforcers could not even live with the most trivial facts of life.

Now some formal network properties which are implicit in the idea of "conditioned arousal" will be explicated. Not the least of these implications is the fact that there must exist cells, distinct from $\mathcal{S}$ and $\mathcal{M}$, which directly control the arousal to $\mathcal{S}$. These cells will generically be denoted by $\mathcal{A}$, and will include network analogs of reticular formation and limbic system. In fact, the idea of conditioned arousal will bridge the gap between respondent and operant conditioning.

To develop these formal properties, the following hypothetical situation will be referred to for definiteness. Consider an animal $\mathcal{O}$ at two successive stages, E_1 and E_2, of its development. At stage E_1, $\mathcal{O}$ salivates in response to the smell of food but not to visual presentation of food. A respondent conditioning experiment is performed in which CS = visual presentation of food, UCS = smell of food, and UCR = salivation. As a result of this experiment, $\mathcal{O}$ salivates in response to the visual presentation of food. This ability characterizes stage E_2.

Now a second respondent conditioning experiment is performed in which CS = ringing bell, and UCS = visual presentation of food, Ultimately, $\mathcal{O}$ salivates in response to a ringing bell.

In this setting, a direct answer to question (ii) would read as follows: suppose that a CS can acquire UCS properties as a result of practice, just as visual presentation of food does as $\mathcal{O}$ evolves from E_1 to E_2. If we also accept the existence of UCS-mediated arousal, then we can conclude that the CS becomes conditioned to the (covert) arousal input even as it becomes conditioned to the (overt) UCR.

Given conditioned arousal, the cells $\mathcal{A}$ must exist for a simple reason: in order to gain control over arousal inputs, the CS must sample cells which control these inputs. These cells cannot be the cells $\mathcal{M}$, because U_1 must be aroused, and indeed must fire, before $\mathcal{M}$ is activated by the UCS. Hence other cells exist, which we denote generically by $\mathcal{A}$.

We now state some of our previous results in terms of the cells $\mathcal{A}$. First, the UCS (e.g., olfactory input at stage E_1) activates axons leading to $\mathcal{A}$, and $\mathcal{A}$ projects to $\mathcal{S}$. In effect, UCS presentation arouses $\mathcal{S}$ via the "indirect" or "arousal" circuit UCS $\rightarrow \mathcal{A} \rightarrow \mathcal{S}$. (The next section will show that "most" UCS's also activate $\mathcal{S}$ via a "direct" or "specific sensory" circuit UCS $\rightarrow \mathcal{S}$.) See Fig. 4.

The CS (e.g. visual input at stage E_1) does not initially have a strong pathway to $\mathcal{A}$, but we have assumed that the CS can acquire such a pathway as a result of practice. Hence the CS activates a cell site (or cell, or cells) which can activate an axon leading to $\mathcal{A}$. The CS also activates a cell site which can activate an axon leading to $\mathcal{M}$. The connections from these sites to $\mathcal{A}$ and $\mathcal{M}$ will be strengthened by the usual respondent mechanism of cross-correlation at the synaptic knobs. We will now show that these cell sites
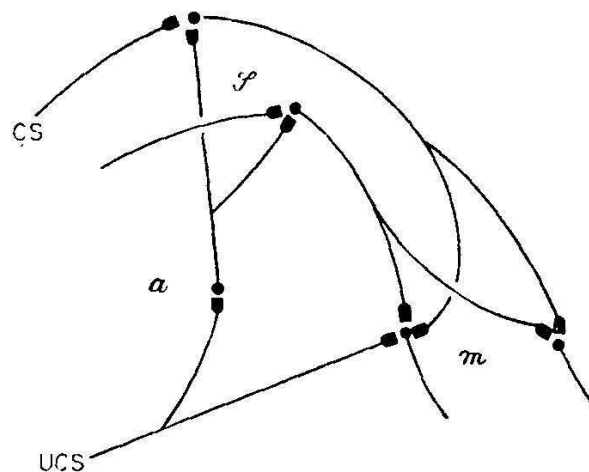

Fig. 4.

cannot be the same. Denote the CS-activated cell site which samples $\mathscr{A}$ by U_{11} and the CS-activated cell site which samples $\mathscr{M}$ by U_{12}. The cells $\mathscr{A}$ project to U_{12} so that the CS and arousal inputs can combine to fire U_{12}, which thereupon samples $\mathscr{M}$. Figure 5 illustrates the four ways in which the CS can deliver inputs to $\mathscr{S}$. Figure 5(a) is impossible because U_1 cannot fire on recall trials unless it is aroused by $\mathscr{A}$. U_1 cannot, however, be aroused by $\mathscr{A}$ unless it first fires to activate $\mathscr{A}$. Figure 5(b) is impossible for similar reasons. Both Fig. 5(c) and (d) avoid this difficulty just so long as U_{11} can fire without first receiving UCS-mediated arousal. Figure 5(d) has an advantage over 5(c) which adumbrates future developments (section 10): if after the CS input terminates one could keep U_{11} active, then all preconditions for learning in response to the CS would be retained by the network. Activity in U_{11} can endure for only a short time, however, unless U_{11} reverberates electrically with another cell, or with itself via a recurrent collateral. If U_{11} reverberates, then signals also travel to $\mathscr{A}$ and U_{12} (by definition of a single cell site). No spurious conditioning will occur during this reverberatory period. By contrast, in Fig. 5(c), both U_{11} and U_{12} must reverberate to maintain the preconditions for learning. In particular, U_{12} samples $\mathscr{M}$, so that learning can occur even in the absence of arousal inputs to U_{12}. In short, Fig. 5(c) cannot exist if the CS-activated sites can reverberate.
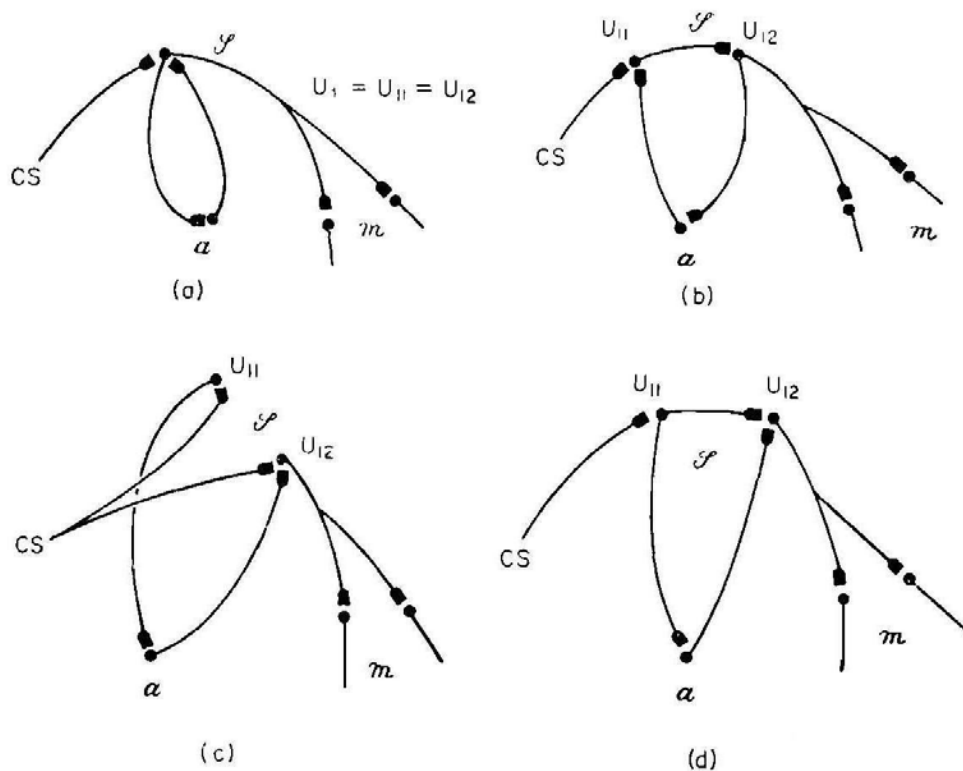

FIG. 5.

5. Higher-order Conditioning: A Principle of Equivalence for the Anatomy of CS and UCS

Given that a CS can acquire UCS properties due to practice, we can conclude that important features of the anatomical representations of CS and UCS are often the same. To see this, let $\mathcal{O}$ be exposed to a sequence of respondent conditioning experiments $E_1, E_2, \ldots$. Denote the CS of E_m by $(CS)_m$ and the UCS of E_m by $(UCS)_m$. Let the CS of E_m be the UCS of E_{m+1}; i.e.

$$(UCS)_{m+1} = (CS)_m, \, m \geq 1.$$

Every such CS can become a future UCS, and every UCS can have been a past CS except possibly $(UCS)_1$. Now assume that axons which process CS and UCS inputs cannot be wholly created or destroyed during the relatively short time needed to pass from any stage E_m to stage E_{m+1}. Thus there exists a common anatomical representation for UCS and CS input processing, except possibly for $(UCS)_1$. By section 4, every $(CS)_m$ has a CS–representation (U_{m1}, U_{m2}) with at least two stages of processing. Thus every $(UCS)_m$, $m > 1$, has the same representation. In other words, all CS and UCS inputs, except possibly $(UCS)_1$, are delivered to $\mathcal{S}$. Figure 6 emphasizes the equivalence of these representations and includes an arrow that marks the direction of successive experiments through time.

Let us call the property of common representation for UCS and CS pathways UCS–CS *path equivalence*. An important consequence of path
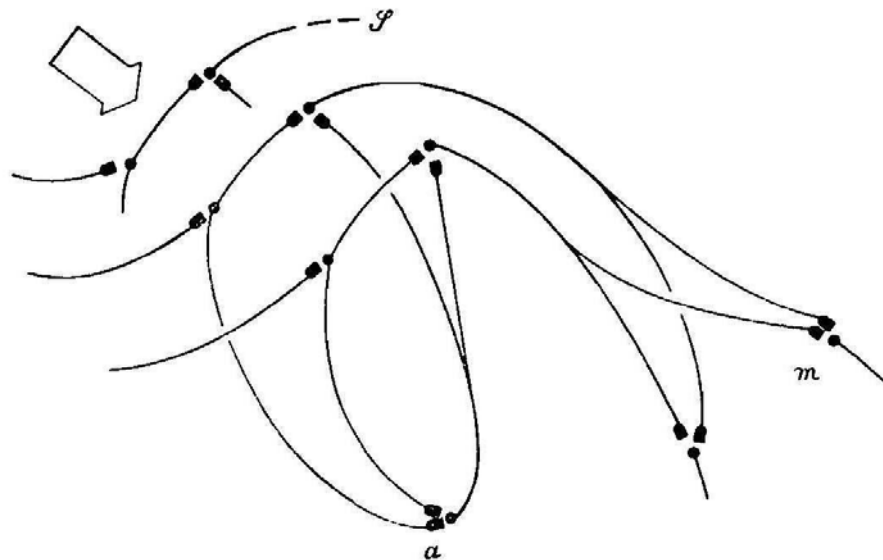

FIG. 6.

equivalence is that both CS and UCS can control both specific and non-specific processes at suitable times in $\mathcal{O}$'s life. Reciprocally, both the CS and UCS representations can receive specific and non-specific inputs at suitable times. The pathway between $\mathcal{S}$ and $\mathcal{A}$ is a two-way street.

6. External Facts vs. Internal Demands

Path equivalence has an important intuitive meaning. Consider the stages E_1 and E_2 of section 4 for definiteness. What intuitive fact has changed when the equation

$$(CS)_1 = \text{visual presentation of food} \tag{3}$$

is replaced by

$$(UCS)_2 = \text{visual presentation of food?} \tag{4}$$

Visual presentation of food has taken on the "significance" of food by being conditioned to arousal. It has acquired an internal "meaning" for $\mathcal{O}$. Arousal prepares $\mathcal{O}$ to be able to learn that the specific external circumstances $(CS)_1$ signals forthcoming satisfaction of the internal demand for food. In particular, the "arousal function" is not always merely an energizer, as Hebb suggested (1955); it can have a cue function also, albeit a cue function concerning the internal state of $\mathcal{O}$ rather than the external state of the world.

Path equivalence shows that "conditioned arousers" sometimes act as "conditioned reinforcers". Since not all arousers are necessarily reinforcers —or at least we have supplied no reason to believe otherwise—we henceforth focus attention on the subset $\mathcal{D}$ of $\mathcal{A}$ whose arousers are reinforcers. We study $\mathcal{D}$ to clarify the concepts of motivation and drive in our network.

Equations (3) and (4) involve the hunger drive. Trivially, hunger is not the only drive, and many drives are partially independent. Thus not all cells in $\mathcal{D}$ represent every drive equally, and $\mathcal{D}$ contains maximal subsets of cells, say $W_1, W_2, \ldots, W_m$, such that W_i corresponds to the ith drive D_i of $\mathcal{O}$. The subsets W_i are not necessarily disjoint—they are the *maximal* subsets related to given drives. The $(UCS)_2$ in (4) therefore activates the subset corresponding to hunger. Henceforth we will call each W_i in $\mathcal{D}$ a *drive representation* (or DR) and each pair (U_{i1}, U_{i2}) in $\mathcal{S}$ a *sensory representation* (or SR).

An SR can, in principle, be conditioned to any of several DR's. Thus each SR sends axons to the several DR's. Similarly, each DR sends axons to several SR's. In just the same *qualitative* sense that outputs from $\mathcal{A}$ (and hence $\mathcal{D}$) to $\mathcal{S}$ are non-specific, the outputs from $\mathcal{S}$ to $\mathcal{D}$ are non-specific.

Nonetheless, there are quantitatively more SR's to which DR's must project than conversely. See Fig. 7.

An important question now arises concerning the reciprocity of connections between $\mathscr{S}$ and $\mathscr{D}$: does each SR which projects to a given DR receive a projection from that DR, and conversely? Reciprocity is important because it enables $\mathcal{O}$ to emit motor behavior in response to the finest sensory discriminations. The following examples illustrate this. If SR projects to DR, but not conversely, then activation of SR cannot release SR–specific motor behavior via DR arousal, even if the SR $\rightarrow$ DR connection has been strengthened by prior conditioning. Suppose two SR's are given, namely $(SR)_1$ and $(SR)_2$, such that only $(SR)_1$ projects to DR, and DR projects only to $(SR)_2$. Let both $(SR)_1$ and $(SR)_2$ always occur together during conditioning trials involving only this given DR. Then on recall trials, if $(SR)_1$ and $(SR)_2$ both occur, then $(SR)_2$ can release motor behavior once it receives $(SR)_1$–activated DR arousal. If either $(SR)_1$ or $(SR)_2$ occur separately on recall trials, then no motor behavior can be released. In other words, changing the total configuration of "stimulus elements" from learning to recall trials can prevent performance by deactivating either the arousal or the motor control. More complicated examples are readily imagined. Henceforth we will always consider the reciprocal case for definiteness.
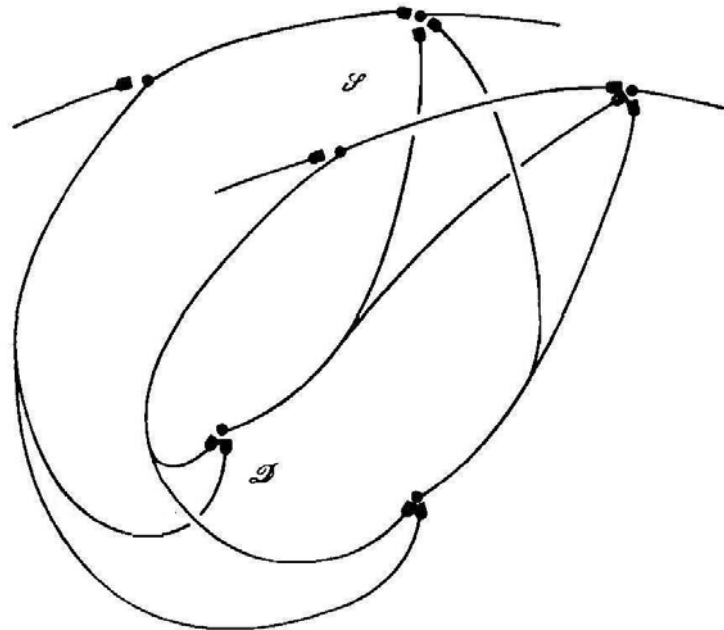

FIG. 7.

7. Internal Facts vs. External Demands:
Existence of Internal, or Homeostatic, Inputs

Thus far, if an SR has previously sampled a DR and a pattern on $\mathcal{M}$, then this pattern can be released by activating the SR. This will occur even if the drive represented by DR is fully satiated whereas another drive is unsatisfied. The difficulty is analogous to permitting an SR to fire in the absence of its sensory event, and can be remedied accordingly. Let each W_i receive, in addition to inputs from $\mathcal{S}$, an internally created input $I_i^{(w)}(t)$ whose size indicates the drive level of D_i. Let the two inputs combine additively at W_i, and choose the spiking threshold of W_i, once and for all, so that W_i fires only if it receives an input from $\mathcal{S}$ *and* an input $I_i^{(w)}(t)$ almost simultaneously and with sufficient intensity. Thus we have imposed steps (i) and (ii) of section 2 on $\mathcal{D}$ as well as on $\mathcal{S}$. See Fig. 8. Now motor behavior based on DR can be released only if its corresponding drive level is positive. Note the trend towards symmetry between the $\mathcal{S} \rightarrow \mathcal{D}$ flow of signals and the $\mathcal{D} \rightarrow \mathcal{S}$ flow.
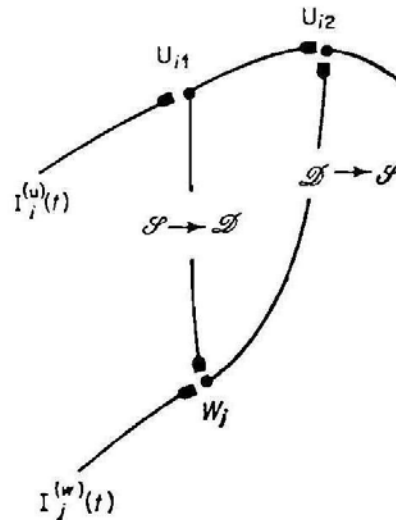

FIG. 8.

8. Experimental and Theoretical Correlates of Conditioned Arousal

(A) CUE AND AROUSAL FUNCTIONS

Hebb (1955) has emphasized the two-way traffic between cortical (e.g. $\mathcal{S}$) and subcortical (e.g. $\mathcal{D}$) cells in guiding behavior. Note that both $\mathcal{S}$ and $\mathcal{D}$ now have both a cue function and an arousal function; e.g. the input $I_i^{(w)}(t)$ is the "cue" input to W_i and $\mathcal{S}$ supplies the "arousal" input to W_i. Neither $\mathcal{S}$ as an arousal for $\mathcal{D}$ nor $\mathcal{D}$ as an arousal for $\mathcal{S}$ is merely an energizer.

(B) "GO" MECHANISMS

Miller (1963) has described an ingenious alternative to the drive-reduction hypothesis. He discusses "go" mechanisms that "act to intensify ongoing responses to cues," that "are subject to conditioning with contiguity being sufficient," such that "the strength of the CR is determined to a great degree by the strength of the UCR," and "when a chain of cues leads to a UCS for the 'go mechanism', it is most strongly conditioned to those nearer to the UCS", etc. All of these conditions are satisfied by our networks if the "go" mechanism is interpreted as "arousal". Also see Livingston (1967) for a discussion of "now print" mechanisms.

(C) INCENTIVE-MOTIVATIONAL INCREASES

Campbell (1960) and Bindra & Palfai (1967) explain observed incentive-motivational increases in general activity by classical conditioning to the CS of a central motivational state which was originally activated only by the UCS. See Valenstein (1969, pp. 70–71) for a review. This "central motivational state" is presumably analogous to a DR.

(D) DRIVE-REINFORCER INTERACTIONS

Bindra (1968) claims that reinforcing effects occur when a sensory input arising from reinforcing stimulus objects interacts with the corresponding drive state. There must thus be a common neural locus where sensory inputs arising from incentive objects interact with the neural changes due to drive manipulation. Scott & Pfaffman (1967) provide indirect support for this idea in studies of the hypothalamus. Again see Valenstein (1969, p. 72) for a review. In our network, these loci are the DR's which receive internal "homeostatic" inputs and conditioned arousal inputs.

Valenstein, Cox & Kakolewski (1970) note that "hypothalamic stimulation ...seems to create conditions which excite the neural substrate underlying well-established response patterns.... Discharging this sensitized or excited substrate is reinforcing and it can provide the motivation to engage in instrumental behavior...rats which display stimulus-bound eating prefer the combination of food and brain stimulation to brain stimulation alone.... The brain stimulation does not fully activate all the neural circuits underlying reinforcement...." These remarks can be interpreted much as in the previous paragraph.

Kelleher (1966, pp. 179–81) discusses experimental evidence that "stimuli become conditioned reinforcers through respondent conditioning."

John & Morgades (1969) have reported that, in trained animals, differentiated stimuli elicit characteristic responses distributed rather uniformly

through extensive cellular regions, and that there is an increase in late components of potential shifts after training. In our networks, once a CS is conditioned to a DR, the DR can transmit the potential "signature" of the CS–DR interaction to all the cells in $\mathscr{S}$ which it activates. Moreover, the arousal is a "late" input, since it is activated via a pathway that is less direct than the specific sensory pathway.

(E) CONDITIONING AND SELF-STIMULATION

Olds (1958) showed that rats will push a lever at high rates if it activates an electrode placed, for example, at suitable hypothalamic loci. In the present context, the electrical signal replaces the arousal input due to UCS presentation, thereby permitting conditioning both of arousal and of bar pressing to the sensory stimuli elicited by the bar. No drive reduction occurs as a result of bar pressing, since the internal homeostatic input which summates with the electrical signal need not diminish as it would, for example, if it represented hunger drive level and food had been eaten. Thus the bar pressing response can be learned and repetitively performed without drive reduction.

This interpretation is strengthened by studies on the interaction of hunger or androgen level on self-stimulating bar pressing at various anatomical loci. For example, "an apparently rewarding stimulus often increases the hunger drive". The electrical signal presumably has summated with the internal homeostatic input that represents hunger level. The choice of which sensory-motor loop is activated—eating or bar pressing—presumably depends on which sensory stimuli are available, food or lever. Both behaviors can share a common arousal source.

At suitable loci, self-stimulation disappears almost completely after castration. Presumably at these loci, the homeostatic input, maintained by the internal androgen level, has been eliminated. Olds points out that higher current levels were needed to achieve self-stimulation as the androgen level subsided. The effect could be reversed by injections of testosterone propionate in oil.

It was also found that at loci where androgens improved response rates, hunger often had a detrimental effect, and conversely. This data suggests that the two drives reciprocally inhibit each other. See section 11 for a simple mechanism of this type.

(F) CONTINGENT NEGATIVE VARIATION

The CNV (contingent negative variation, or expectancy wave) is a "steady potential" that can be classically conditioned, and which precedes motor

behavior in suitable experiments (Cohen, 1969). Presumably the CNV has much the same effect on the release of motor activity as do directly induced anodal d.c. potential shifts.

9. Operant Conditioning Compared to Respondent Conditioning

Consider the following (simplified description of an) operant conditioning experiment. A tone is presented to an animal $\mathcal{O}$. If and only if $\mathcal{O}$ pushes a lever after the tone is presented—and within prescribed time constraints— $\mathcal{O}$ receives food. On a recall trial the hungry $\mathcal{O}$ pushes the lever in response to tone presentation alone. How does presentation of food strengthen the connection from "tone" to "lever pressing"?

A possible answer is readily available. First assume that when food is presented, internal traces of prior tone presentation—$(SR)_1$—and of lever pressing—$(SR)_2$—are active in $\mathcal{O}$. Also let food presentation create an internal trace $(SR)_3$. The $(SR)_1$ should be reproducible by tone presentation on recall trials. The $(SR)_2$ should be able to release lever pressing if it is reproduced under suitable arousal conditions.

Compare these SR's with the CS- and UCS-representations of respondent conditioning. The $(SR)_1$ is a clear analog of a CS-representation. The $(SR)_2$ cannot be an exact analog of a UCS-representation, because it does not initially activate the DR corresponding to food. The $(SR)_3$ does this. Thus, during learning trials, the $(SR)_1$ samples the $(MR)_2$ activated by $(SR)_2$ *and* the DR activated by $(SR)_3$. During recall trials, $(SR)_1$ connects strongly both to $(MR)_2$ and to DR. If $\mathcal{O}$ is hungry (i.e. the DR also receives a large positive internal input), DR fires back to $(SR)_1$, whereupon $(SR)_1$ can activate $(MR)_2$ and release the motor response which it has sampled on learning trials. Some features of respondent and operant conditioning hereby emerge as part of a unified view of learning. See Fig. 9.
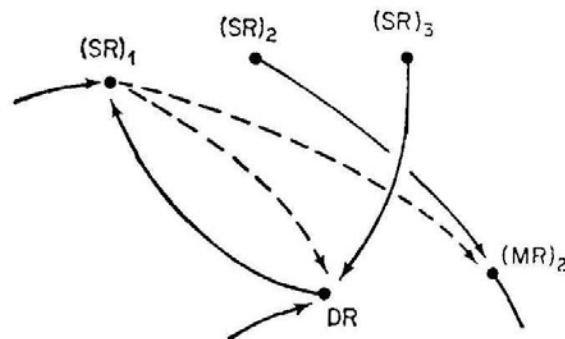

FIG. 9.

This preliminary analysis suggests a long series of questions. The most obvious are: (i) What keeps the tone and lever pressing representations active until food is presented? (ii) What shuts these representations off so that later events can be represented without interference? (iii) Must one classify the lever-pressing representation as an SR rather than as merely an MR? (iv) Given that a lever-pressing representation is active after the lever is pressed, why is not the lever pressed repetitively until the representation is deactivated? (v) Is the DR conditioned to the $(SR)_2$ as well as to the $(SR)_1$? (vi) Given that the lever pressing representation is an SR, and that this SR is not initially conditioned to the DR, how does this SR release motor activity in the first place? Does every such SR control an arousal source, not necessarily a DR, that permits it to release motor activity?

10. Reverberation and Unattended Holding

Consider question (i). At least three types of mechanism can keep $(SR)_1$ and $(SR)_2$ active in our networks: (a) slow passive decay of potentials at fixed cellular sites; (b) temporary chemical encoding in the synaptic knobs of a recurrent network [Fig. 10(a)]; (c) electrical reverberation between recurrent cellular components [Fig. 10(b)]. Mechanism (c) will be seen to have several advantages; non-recurrent chains, or "avalanches," of this mechanism will be considered in section 12.

Consider (a). The argument for (a) is simple: let potentials decay so slowly that $(SR)_1$ and $(SR)_2$ are still active when $(SR)_3$ is activated. The counter-arguments are substantial. Passive decay implies that decay rates are fixed through time; thus, each SR decays exponentially after its activating input terminates. In order that $(SR)_1$ and $(SR)_3$ be simultaneously active,

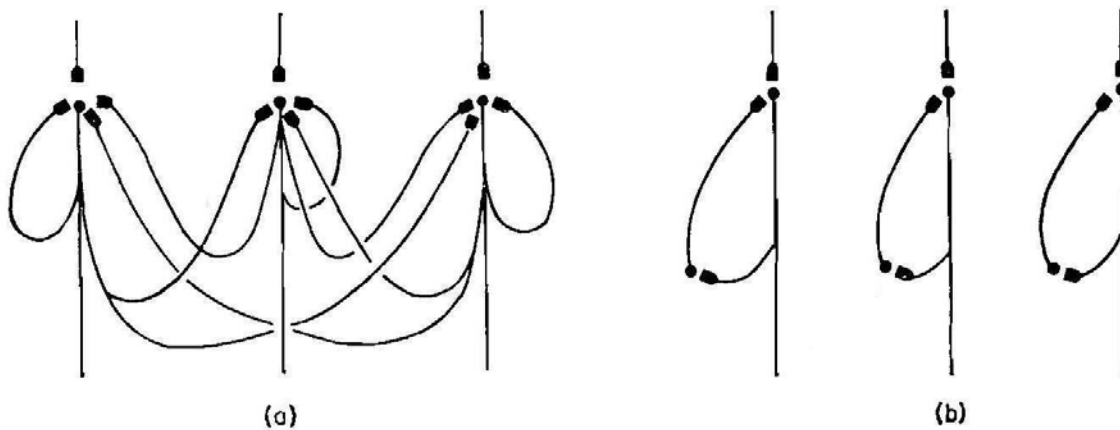(a)   (b)

Fig. 10.

the decay rates of each SR must be so slow that little decay occurs for several seconds. In particular, if a rapid series of sensory events occurs, then large sectors of $\mathscr{S}$ can be simultaneously active, even though only the few last events are behaviorally relevant at present. In short, massive response interference and background noise readily accumulate. Of course, inhibitory inputs could be delivered to $\mathscr{S}$ to eliminate noise at appropriate times. But then the decay rate of inhibitory effects will also be slow, rendering the system unresponsive for times ranging in the seconds. Whereas such a system can in principle work given sufficiently few inputs delivered sufficiently slowly, it lacks flexibility and precision. Moreover, it would require the existence of neural potentials that decay over a period of no less than several seconds.

Consider (b). An example of the network in Fig. 10(a) is given by

$$\dot{x}_i(t) = -\alpha x_i(t) + \beta \sum_{k=1}^{n} \left[x_k(t-\tau_k) - \Gamma_k \right]^+ z_{ki}(t) + I_i(t)$$

and

$$\dot{z}_{jk}(t) = -\gamma_j z_{jk}(t) + \delta_j [x_j(t-\tau_j) - \Gamma_j]^+ x_k(t),$$

where $i, j, k = 1, 2, \ldots, n$. Such a network—and in fact substantial generalizations thereof—can learn and remember any spatial pattern $I_i(t) = \theta_i I(t)$ (Grossberg, 1969d). Thus if a particular pattern playing on the n cells v_i, $i = 1, 2, \ldots, n$, characterizes a given SR, then once this pattern is learned by the weights $z_{jk}(t)$, it can be reverberated electrically through the network for considerably longer than the decay parameter α would alone permit by proper choice of α, β, γ_j, and δ_j. Three difficulties arise here. The first is that, again, it takes a long time to stop the reverberation of a pattern once it begins. Second, the memory traces change even more slowly than the electrical traces. Third, the connections from each cell to all cells might be hard to grow *in vivo*.

Mechanism (c) can avoid these difficulties. Mathematical details concerning the following qualitative properties will appear in another place. First, mechanism (c) can electrically reverberate a given spatial pattern for as long as one pleases. Second, change-over to a new pattern can be made as rapidly as one pleases if the input also non-recurrently inhibits the reverberatory link. Third, using auxiliary connections, the reverberatory cycle can be made either to sample other cell sites continually until arousal releases motor controls, or to briefly sample other cell sites when it is released by arousal. Fourth, the reverberatory cycle can rapidly be suppressed either by arousal or by the firing of the final stage of SR processing. This mechanism therefore provides an answer to question (ii) as well. In summary, electrical reverberation within parallel, but otherwise independent, cell cycles is a

mathematically possible way of maintaining long lived SR's without paying the price of sluggish decay and massive response interference.

The same auxiliary connections can stop electrical reverberation in mechanism (b), but the memory traces take longer to decay. Also, the anatomy in mechanism (b) is probably harder to grow *in vivo*.

A similar reverberatory mechanism can capture some formal properties of the following familiar behavioral phenomenon: an object is consciously picked up and held; then attention focuses on walking and other unrelated matters, while the object is still held for an indefinitely long period; the object can nonetheless be rapidly released at will.

The next two sections sketch how reverberatory cycles and arousal can be used to construct "avalanches" of cells capable of learning and performing complex patterns with considerable flexibility. Section 11 endows $\mathscr{D}$ with some further structure that will be used in section 12.

11. Incompatible Drive States

Different drives can sometimes control incompatible acts. How can simultaneous activation of incompatible drive states be prevented? Grossberg (1970a, section 4) shows that n mutually incompatible behavioral modes can be interconnected to guarantee that at most one is activated at any time. The following discussion mildly generalizes this mechanism.

Let the excitatory input $I_i(t)$ be delivered to v_i and let the inhibitory input $\beta I_i(t)$ be delivered to all v_k, $k \neq i$, by non-recurrent inhibitory interneurons. Also let the potential $x_i(t)$ decay at the rate α. Then

$$\dot{x}_i(t) = -\alpha x_i(t) + I_i(t) - \beta \sum_{k \neq i} I_k(t).$$

Define the ratios $\theta_i = I_i I^{-1}$, where $I = \sum_{k=1}^{n} I_k$. Then

$$\dot{x}_i(t) = -\alpha x_i(t) + J_i(t),$$

where

$$J_i(t) = (1 + \beta)I(t)(\theta_i(t) - \beta/(1 + \beta)).$$

Clearly

$$J_i(t) > 0(< 0) \quad \text{only if} \quad \theta_i(t) > \beta(1 + \beta)^{-1}(< \beta(1 + \beta)^{-1}).$$

In particular, if $\beta \geq 1$, then at most one $J_i(t)$ is positive. Hence at most one $x_i(t)$ can be excited at any time; all other $x_i(t)$ are actively inhibited. Choosing the output threshold of each v_i sufficiently large now guarantees that at most one v_i fires at a time.

Two possible cases of this mechanism are: either (i) the incompatible DR's inhibit each other before they are sampled by SR's; or (ii) SR and DR inputs combine additively, and then the summated potentials inhibit each other.

In case (i), only the dominant DR can be sampled and can fire to release motor activity at any time [see Fig. 11(a)]. In case (ii), *all* DR's can be
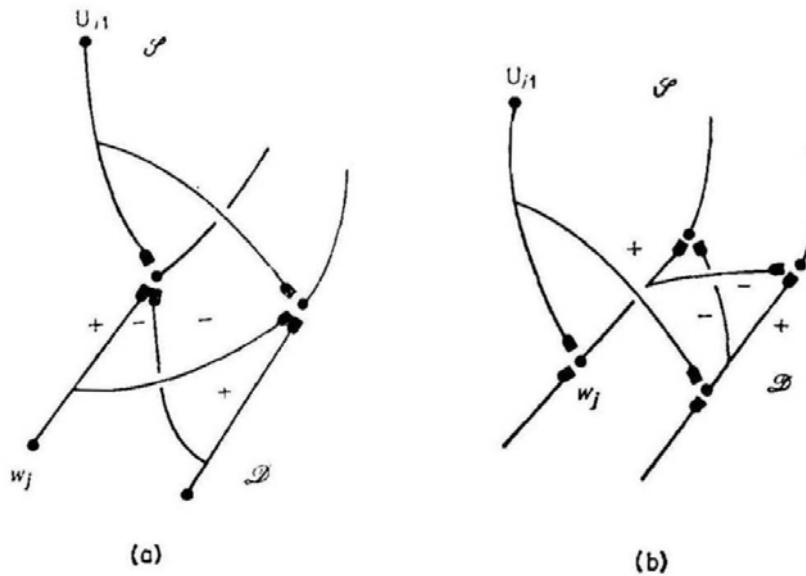

FIG. 11.

sampled, and a non-dominant DR can release motor activity if it is sampled by a sufficiently prepotent SR [see Fig. 11(b)]. In either case, no more than one DR can release motor activity at any time. Case (i) is the anatomy of choice unless the total energy reaching each DR from $\mathscr{S}$ is carefully regulated to mirror environmental demands. Case (ii) also has the unusual property that the SR → DR memory traces need bear little relation to the DR → SR pattern of activity. Section 12 will provide an example of how case (i) can be put to use.

12. Avalanches of Reverberating Cycles Modulated by Arousal

Grossberg (1969e, 1970d) constructed a cell which can learn, remember, and perform a space–time pattern of essentially arbitrary complexity; namely, an "outstar avalanche". See Fig. 12. In physiological terms, the cell axon sequentially emits clusters of axon collaterals that sample a given MR, or other groups of cells. Each cluster can encode a spatial pattern, and the space–time pattern is encoded and retrieved as a sequence of spatial
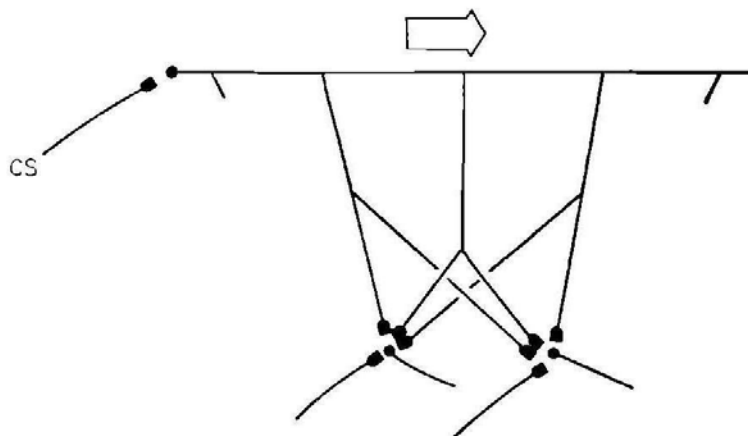
FIG. 12.

patterns. Grossberg (1969*b*) also discusses related anatomies having particular advantages. The outstar avalanche has some disadvantages too. Some can now be corrected. They are: (i) Performance is ritualistic. Once the cell body starts to fire, the entire space–time pattern must be performed before O can do anything else. If a more urgent demand is imposed by the environment (external or internal) during performance, O cannot meet the demand. (ii) Once performance is initiated, successive spatial patterns are sampled at a fixed rate that is independent of O's state of arousal or interest. Using reverberating cycles and arousal, the avalanche anatomy can be modified so as to terminate pattern performance abruptly, switch to a new pattern that the environment demands, and to perform successive spatial patterns at a rate which reflects O's changing arousal level.

Clearly the weaknesses (i) and (ii) are due to the fact that, once the axon is excited, the excitation propagates non-decrementally along the entire axon at a fixed rate. To avoid this, first interpolate cell bodies at each outstar source, as in Fig. 13(a). The firing of these cells must be made sensitive to shifts in the focus of arousal through time. This is done by replacing each cell with an SR. Thus the avalanche is replaced by a non-recurrent chain of SR units (NCSR), as in Fig. 13(b).

The structure of $\mathcal{D}$ will clearly influence NCSR dynamics. For definiteness, let the DR's be mutually incompatible, and endow $\mathcal{D}$ with the anatomy of case (i) in section 11. Let only one DR project to the NCSR.

Consider the *i*th link of the NCSR. The following properties are evident: (i) U_{i2} can fire only if U_{i1} and the DR fire to it. Successive activation of spatial patterns in the NCSR will therefore end as soon as the DR is inhibited by an incompatible drive. (ii) A different NCSR that is compatible with the newly dominant DR and ongoing sensory events can simultaneously be activated. (iii) The rate with which U_{i2} begins to fire after U_{i1} begins to
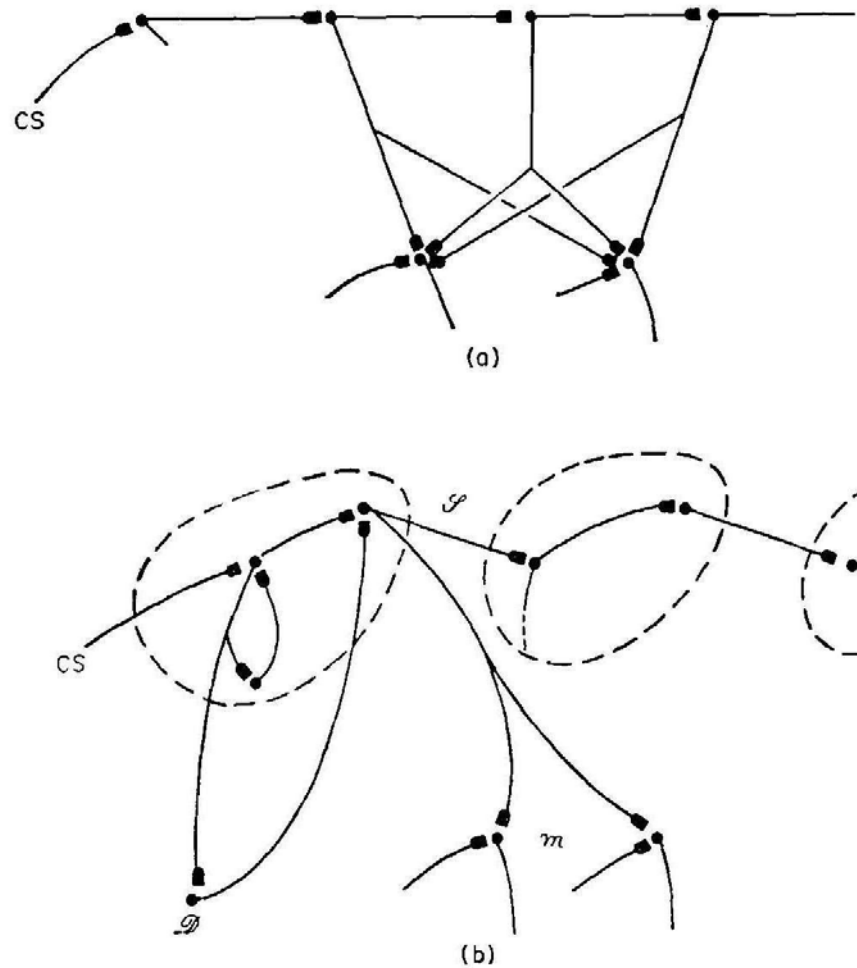
(a)

(b)

FIG. 13.

fire depends on the size of the DR input: reaction time is a monotone decreasing function of DR input size, other things equal (Grossberg, 1969c). Thus performance speed can be continuously modulated as an act is being performed. This modulation does not alter the "information processing" of the network, but only its "energy processing".

Now attach a reverberating cycle to each U_{i1}, as in section 12. Let U_{i1} be activated, but suppose that DR arousal is inhibited before U_{i2} can fire. What happens? The ith reverberatory cycle is activated, and NCSR performance stops. As soon as DR arousal resumes, NCSR performance continues at the point where it left off. $\mathcal{O}$ has "paid attention" to other matters, and then "continued the conversation" where it left off. Of course, in more realistic situations, not all SR's are mutually compatible, just as not all DR's are mutually compatible. Hence the occurrence of an incompatible SR can sometimes eliminate reverberation even as it shifts the arousal focus.

13. Experimental and Theoretical Correlates of NCSR's

(A) OPERANT VS. RESPONDENT

Miller and his colleagues (Di Cara, 1970) have emphasized the unity of respondent and operant conditioning mechanisms by carrying out operant conditioning experiments on the autonomic nervous system.

(B) SHORT-TERM VS. LONG-TERM MEMORY

Hebb's classic book (1949) focussed attention on a possible role for reverberation in keeping alive the memory trace. Later freezing and shock experiments demonstrated, however, that memory can persist even if normal electrical events are disturbed. Mechanism (c) of section 10 avoids Hebb's problem. Electrical reverberation now merely prolongs the duration of an excitatory phase that does not encode new memories.

Mechanism (b) is closer to Hebb's view. This mechanism cannot always reverberate the pattern imposed by inputs if the anatomy is changed; many recurrent anatomies distort the pattern of inputs [see Grossberg (1969f) for an example].

The distinction between short-term and long-term memory has attracted much experimental interest (Chorover & Schiller, 1965; Cohen, 1970; Coons & Miller, 1960; Russell & Nathan, 1946). The short-term reverberatory phase and longer-term chemical storage phase in our networks seem to parallel this distinction. Our reverberatory phase is not, however, a "learned memory" phase, since it prolongs representations rather than preserving a connection between two representations.

Our short-term phase keeps traces alive long enough for reinforcement (or $\mathscr{D}$) to act on them. Disruptions of $\mathscr{D} \to \mathscr{S}$ arousal can prevent the short-term phase from influencing the long-term phase. For example, if $\mathscr{D} \to \mathscr{S}$ arousal does not occur, then no cell site U_{i2} can fire, and no new sampling by U_{i2} of $\mathscr{M}$ (or other cells) can occur.

A possible experimental analog of this disruption is discussed by Milner (1958). Bilateral hippocampal ablation prevents transfer of information from the short-term into the long-term memory store: a distracting event can end all recall of a previous recent event.

Possibly the hippocampal loss disrupts more than $\mathscr{D} \to \mathscr{S}$ arousal. Is the $\mathscr{S} \to \mathscr{D}$ pathway also disrupted? Can a hippocampus-deficient individual learn emotional responses? If the $\mathscr{D}$ cells are also missing, the answer should be "no".

If the disruption occurs in the $\mathscr{D} \to \mathscr{S}$ pathway, how are "old" memories released? Only two choices are presently available: either the $\mathscr{D} \to \mathscr{S}$ pathway shifts from the hippocampal focus to another arousal source as practice

continues, or the direct $U_{i1} \rightarrow U_{i2}$ connection becomes stronger, bolstered by indirect $U_{i1} \rightarrow \mathscr{D} \rightarrow U_{i2}$ arousal, as practice continues. "Short-circuited" connections of this latter type, were they to exist, might properly be called "functionally autonomous."

(C) SERIAL ORDER IN BEHAVIOR

In his classic discussion of serial order in behavior, Lashley (1951) noted that some complex motor skills can be performed so quickly that they cannot be continuously controlled by sensory feedback. Virtuoso piano playing is a good example of such a skill. Avalanches can also activate successive MR's with great speed and without sensory feedback. Their performance can nonetheless be modified or even stopped by altering the arousal level.

An electrode in the temporal lobe of man can vividly activate a sequence of perhaps very old memories, including visual and auditory memories (Penfield, 1958). Discontinuing electrode current while the sequence is being recalled can stop recall. Reapplying current at the same point can reinitiate recall of the same sequence.

Analogous performance can be achieved by an NCSR, if we suppose that the electrode activates both a specific site U_{11} and an arousal source, and let the electrode continue to excite $\mathscr{D}$ even after U_{12} has fired. Then successive links of the avalanche will be activated until current is shut off and arousal thereby ended. These conditions need not, of course, be satisfied in all electrode penetrations, either because the cell site is not analogous to the source of an NCSR or because the proper conditions of arousal are not met.

(D) IDETIC MEMORY

An avalanche of the above type must activate cells which can sample auditory and visual representations, as well as MR's. The existence of cells which can sample the sensory cortices is suggested also by data on idetic memory (B. Julesz, personal communication). This data reports the following remarkable phenomenon. Two pictures are constructed by computer from 10^4 randomly distributed black and white dots. These pictures conceal a figure in depth that can only be seen when the pictures are viewed binocularly (Julesz, 1964). An idetic woman studies the first picture with one eye on day one of the experiment and returns the next day to study the second picture with the other eye. She then identifies the concealed figure! See also Haber (1969).

A simple network can do this formally. All we need is an outstar (or a cluster of outstars that fire in unison) that projects to the correct visual

representation area. If the woman can activate the outstar at will, then she can sample the first picture to an arbitrary degree of accuracy on day one. This is proved by Grossberg (1969*d,e*, 1970*b*). On day two, if she again activates the outstar, the internally produced representation of the first picture will interact with the externally produced representation of the second picture to produce the usual binocular effect of a figure in depth. To sample 10^m pictures playing on 10^n visual cells, one needs no more than $10^m + 10^n$ ($\leqslant 2 \times 10^{\max(m,n)}$) cells using this mechanism, not 10^{m+n} cells, as is occasionally claimed. The most remarkable feature of eidetic memory in our networks is the ability to internally activate the cluster of outstars at will. This is presumably a rare capability. This mechanism also requires that clusters of cells project to broad regions of suitable visual representation areas.

(E) THERAPEUTIC RESISTANCE AND REPRESSION

A familiar psychoanalytic phenomenon is "therapeutic resistance". For various reasons, the patient finds it hard to unlearn emotionally disturbing facts. A possible contributing mechanism occurs in our networks, and is due to two factors: (i) conditioning of SR → DR pathways; and (ii) the destructive effects of over- or under-arousal on the ability to pay attention and learn (Grossberg & Pepe, 1971*a,b*). For example, suppose that particular sensory events occur during a very disturbing emotional experience. Consequently, let an SR be strongly conditioned to a DR, say $(DR)_1$, that massively projects back to $\mathscr{S}$. The question of whether $(DR)_1$ overexcites or overinhibits $\mathscr{S}$ will be temporarily overlooked; both effects will impair learning. Whenever this SR is presented again—say by the psychotherapist— massive overarousal or inhibition of $\mathscr{S}$ will occur, and will prevent new learning about the consequences of SR.

To avoid this interference, two therapeutic steps can be (and have been) attempted: (i) reversible blocking of $(DR)_1$ firing during therapy, say by drugs; and (ii) conditioning of a new pathway SR → $(DR)_2$, while $(DR)_1$ is blocked, to a $(DR)_2$ that is incompatible with $(DR)_1$. Continue conditioning until the new pathway SR → $(DR)_2$ is stronger than the old pathway SR → $(DR)_1$. Then discontinue drugs. On later presentations of SR, the SR $\overset{+}{\to} (DR)_1$ pathway can be inhibited by the SR $\overset{+}{\to} (DR)_2 \overset{-}{\to} (DR)_1$ pathway. Thus the new learning "spatiotemporally masks" the old learning (Grossberg, 1969*b*).

Another familiar psychoanalytic phenomenon is "repression," which prevents unforgotten facts from being retrieved. Any mechanism that inhibits arousal can prevent retrieval without necessarily destroying specific memory traces.

(F) INSTINCTIVE AVALANCHES

Lehrman (1965) has reported that the release of successive acts in the reproductive cycle of ring doves requires an interaction between the external and internal environments of these birds. In a clear sense, the internal control of this cycle has an avalanche structure in which successive stages of the avalanche can be released only by a predetermined mixture of external and internal inputs. Also see Hailman (1969) for a discussion of how "instinctive" behavior is sharpened by learning in young laughing gulls.

14. Control of Motor Outputs

Consider questions (iii) to (vi) of section 9. An answer to question (iii) is suggested by question (iv). Suppose that the lever-pressing representation is an MR. We wish to keep the MR active without creating continual lever-pressing. If reverberation maintains this activity, then at least two cell sites, as in an SR, are needed—one to maintain the reverberation, and a second to release motor activity. As the first site reverberates, it sends signals continuously to the second site. The second site therefore requires a second input to fire it and release motor activity. This second input is presumably an arousal input. Such an MR has the same structure as an SR; hence we assume that the lever-pressing representation is an SR. Once this SR releases the motor act, either reverberation stops or the arousal yielding motor output ceases. Yet the reverberation persists at least until reward occurs in the operant paradigm. Thus in the former case, some feedback to the "motor" SR due to performance of the motor act can be anticipated; e.g., proprioceptive feedback. In either case, the times during which arousal is active must be carefully controlled.

A qualitative answer to question (iv) is now available. Since the lever-pressing representation is an SR, the lever is only pressed when this SR is aroused. This analysis leaves open the question of how SR reverberation is finally terminated. The most naive answer is: it is inhibited by the more recent activation of incompatible SR's, much as in an on–off field.

Question (v) will be answered in the affirmative because *every* SR that can sample a DR can be conditioned to this DR. Given an $\mathcal{O}$ with a large repertoire of SR's, a major problem therefore arises: how can $\mathcal{S}$ be hierarchically organized to prevent irrelevant SR's in a given experiment from gaining control over performance?

Question (vi) will also be answered affirmatively. A general motor arousal, distinct from drive arousal, is needed to release behavior until this behavior is controlled by drives. Indeed, it is hard to believe that much spoken language is released by specific drive arousals.

15. Experimental and Theoretical Correlates of Motor Arousal: Conditioning Sensory Feedback to Motor Controls

(A) PASSIVE VS. ACTIVE ADAPTATION

Given motor arousal, the sensory feedback created by a motor act can be conditioned to the enduring trace of the motor control. By contrast, the same sensory inputs, created by passively moving $\mathcal{O}$ through its environment, will not form new connections. Thus, active motor exploration of a new environment, but not passive exploration without motor activity; will create sensory-motor adaptations to that environment. Held and his colleagues (Held, 1965; Held & Bauer, 1967; Held & Hein, 1967; Steinback, 1969) have studied this phenomena extensively in several species.

(B) BABBLING

Fry (1966) notes that in the child's development of language skills, the babbling stage is followed by the establishment of an auditory feedback loop ($\mathcal{M} \to \mathcal{S} \to \mathcal{M}$). Subsequently, babbling can be elicited by adult vocalizations ($\mathcal{S} \to \mathcal{M}$). In deaf children, by contrast, babbling usually develops at the normal age, but in the absence of a developing auditory feedback loop, eventually fades.

(C) CIRCULAR REACTION

Piaget (1952) has discussed extensively the development of "circular reactions" between vision and motions of the hand and fingers in children. First the glance tries to follow what the hand does ($\mathcal{M} \to \mathcal{S}$). Later on, the hand can be guided by vision ($\mathcal{S} \to \mathcal{M}$). Of course, these arrows dramatically over-simplify a complex sequence of events. Nonetheless, ideas of this kind are continually brought to mind during a reading of Piaget's marvelous observations.

16. Conditioning of Sensory Representations to Drive States

Suppose that a given drive becomes active and particular sensory events must be sought to satisfy the drive. How is this done? For example, how does hunger elicit the statement "I want food"? Such an act requires that a DR can selectively activate SR's, or that DR $\to$ SR pathways can also be conditioned. This we now assume.

This assumption removes another asymmetry between the flow $\mathcal{S} \to \mathcal{D}$ and the flow $\mathcal{D} \to \mathcal{S}$ in our networks. This fact suggests that $\mathcal{S}$ and $\mathcal{D}$ contain homologous cell types. Since we have interpreted $\mathcal{S}$ as neocortex,

it is not implausible to expect that $\mathscr{D}$ contains archicortex, as is found in the hippocampus (Crosby, Humphrey & Lauer, 1962, p. 411).

The existence of $\mathscr{D} \to \mathscr{S}$ conditioning simplifies the construction of NCSR's. In section 11, only one DR projected to the NCSR. Now it is possible, before NCSR learning occurs, for many DR's to project to the SR's which will ultimately comprise the NCSR. After learning, only the one DR to which the NCSR was conditioned can arouse it.

17. Concluding Remarks

Simple assumptions have led to neural networks whose properties are compatible with a broad and nontrivial collection of data. Because these networks are rigorously defined, they also include implicit suggestions for new experimental work. This paper has studied only aspects of "positive" conditioning by these networks. Later papers will study problems of punishment, avoidance, and frustration. They will find some distinctions between respondent and operant conditioning. We will then have at our disposal rigorously defined networks capable of learning, remembering, and performing complex tasks in harmony with fluctuating positive and negative external and internal environmental demands.

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