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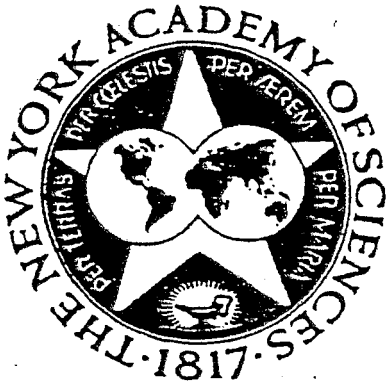

Some Psychophysiological and Pharmacological Correlates of a Developmental, Cognitive and Motivational Theory[a]

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PART I

1. INTRODUCTION: SELF-ORGANIZING INTERNAL REPRESENTATIONS

Studies of event-related potentials (ERPs) can probe a level of neural organization that has behavioral meaning. ERP experiments thereby encourage us to formulate precisely the design problems that are solved by the behaving brain and to translate these design statements into a formal language that is powerful enough to explain how behavioral, physiological, and pharmacological processes are related.

I suggest that these design problems have eluded traditional physical and mathematical thinking because they address a fundamentally new physical situation. These problems concern the design of self-organizing systems, or systems that can generate new internal representations in response to changing environmental rules. This article sketches a psychophysiological theory of how new internal representations are generated. The theory suggests how some ERP-creating mechanisms help to control the self-organization process and how to test these assertions empirically.

In particular, I will suggest that a P300 can be elicited whenever short term memory (STM) is reset by a massive antagonistic rebound within the catecholamine arousal system (Grossberg, 1972b; 1976b; 1978a; 1980a). This suggestion illustrates a sense in which P300s with different anatomical generators can be functionally similar. It also shows why task relevance is important in eliciting P300s, since STM cannot be reset unless it is already active. I will also indicate, however, how a neocortical rebound might elicit a hippocampal rebound by rapidly inhibiting reinforcing signals from cortex to hippocampus. Since the cortical rebound resets a cognitive process and the hippocampal rebound resets a motivational process in the theory (Grossberg, 1975), P300s with different anatomical generators can be functionally dissimilar. Due to the importance of interactions between cognitive and motivational processes for the understanding of both types of processes, I will discuss both cognitive and motivational processes herein and will suggest new explanations and predictions in both domains using the same mechanisms, albeit in different anatomical configurations. I will also suggest that functional homologues of many normal and abnormal motivational properties exist in cognitive properties due to the control of both classes of properties by common mechanisms, notably mechanisms mediated by cholinergic-catecholaminergic interactions. Using these homologues, known motivational phenomena can be used to suggest designs for new types of cognitive experiments and *vice versa*.

The theory also suggests how a mismatch detector, which regulates mismatch negativity in the theory, can sometimes elicit a P300 by triggering a burst of nonspecific arousal to the catecholamine system. An unexpected event can thereby elicit a formal

Antagonistic rebounds can be caused in the absence of a mismatch-contingent arousal burst, as in the hippocampal example above or in a variety of other situations. I suggest that such rebounds occur during perceptual switches between alternative interpretations of an ambiguous figure (Grossberg, 1978a, 1980a) and that each switch elicits a P300 as STM is reset, despite the absence of prior mismatch negativity. The P300s, in turn, elicit negative components, other than mismatch negativity, that play the role of processing negativity in the theory. This processing negativity is assumed to occur as each newly activated STM representation matches the input data compatible with its perceptual interpretation. In this situation, positive activity can elicit negative activity, rather than conversely.

I will also suggest situations, notably overshadowing experiments, in which a monotonic decrease of P300 can predict either a contingent negative variation (CNV) increase or decrease, depending on whether the eliciting cue is predictively relevant or irrelevant in the situation. Also, situations can be contemplated in which processing negativity may either be insensitive or sensitive to stimulus probability, depending on whether the negativity corresponds to the completion of the internal representation of a given item or matching of a predicted item in a probabilistically controlled sequence of items. In this latter connection, the theory suggests how the same anatomical region can emit P300s of different latencies and sizes due to the way in which different sequences of items activate cell populations with different reset parameters. P300 properties in the theory can, moreover, change as long-term memory (LTM) encoding of sequence properties masks individual item codes as a result of sequence repetition (Grossberg, 1978a).

All these interpretations associate formal physiological mechanisms of the theory with a measurement on the scalp. The theory might, of course, be physiologically correct without its scalp interpretation being correct. The same problem is routinely faced in the ERP experimental literature whenever scalp measurements are used to infer brain processes, since we do not yet have a complete theory relating the two levels of description.

ERP interpretations such as those above emerge in the theory as manifestations of how short-term memory and long-term memory mechanisms influence the processing of expected and unexpected events during the self-organization process. To adequately probe these processing interactions, ERP measures might profitably be appended to a greater variety of experimental paradigms wherein STM and LTM properties are highly structured and controllable on the behavioral level. In this regard, I note that STM can be reset by mechanisms other than antagonistic rebound, notably by renormalization of a limited capacity STM system during attentional shifts (Grossberg, 1973, 1975). This multiplicity of possible STM transactions in a neural network requires a finer processing description than is afforded by consensual language or computer analogies and a richer experimental repertoire to probe this description than has traditionally been used in the ERP literature.

Because the theory relates pharmacological systems to STM and LTM properties, it also suggests some pharmacological manipulations that should cause significant ERP measurements. ERPs used as a psychophysiological interface between behavioral and pharmacological paradigms can become a powerful tool for testing psychological, physiological, and pharmacological theories.

2. THE STABILITY-PLASTICITY DILEMMA IN ADULT PERCEPTION

The design problem on which I will base my article is called the stability-plasticity dilemma. This design problem is easy to state because it is so basic. I will state it in several ways so that everyone can resonate with at least one way.

How can a system's adaptive mechanisms be stable enough to resist environmental fluctuations that do not alter its behavioral success, but plastic enough to change rapidly in response to environmental demands that do alter its behavioral success? How are stability without rigidity and adaptability without chaos achieved?

For example, visuomotor maps for reaching towards seen objects seem to be stable in adults until inverting prisms are used to disrupt them, as in the work of Richard Held and his colleagues (Held, 1961, 1967; Held and Hein, 1963). Then a rapid adaptation can occur. Depth percepts seem to be stable in adults until a telestereoscope is used to alter the expected relationships between cues for the kinetic depth effect and cues for retinal disparity, as in the work of Hans Wallach and his colleagues (Wallach and Karsh, 1963a,b; Wallach *et al.*, 1963) Again a rapid adaptation occurs.

If adaptive memory elements can change quickly in response to behaviorally important environmental events, then what prevents them from changing quickly in response to *all* signals that they process, whether meaningful or not—in particular to fluctuations that do not reflect system success or failure? This issue shows that many neural potentials and signals are invisible to the adaptation process and, from a functional viewpoint, are noise rather than signal. To define behavioral relevance, we need to choose a level of discourse that focuses on the interactions within the system as a whole, rather than on local computations at each cell, which cannot distinguish functional signal from noise. It is because this choice is made in my theory that formal analogues of ERPs like N200, P300, and CNV rapidly arise therein.

3. CRITICAL PERIOD TERMINATION AND CHOLINERGIC-CATECHOLAMINERGIC INTERACTIONS

The stability-plasticity issue arises in the infant, no less than in the adult, when we consider how certain environmentally sensitive perceptual critical periods are terminated. Pettigrew and Kasamatsu's recent experiments on kittens probe this issue on a physiological and pharmacological level. Kasamatsu and Pettigrew (1976) show that 6-hydroxydopamine poisoning of the catecholaminergic arousal system to the visual cortex during the visual critical period can prevent normal plasticity from occurring. Pettigrew and Kasamatsu (1978) also show that selective addition of noradrenaline after the critical period has terminated can restore plasticity. This latter experiment raises the question, why should adding a little more noradrenaline restore plasticity when the noradrenaline in the cortical arousal system was perfectly functional all along?

My explanation of this puzzle is based on a model of cholinergic-catecholaminergic interactions that appeared before the Pettigrew and Kasamatsu work was carried out (Grossberg, 1972b, 1976a, 1976b). This model also has implications, to be discussed below, for understanding normal and abnormal cholinergic-catecholaminergic interactions, as in Parkinsonism, hyperphagia, juvenile hyperactivity, drug withdrawal, categorical boundary shifts, intragastric drinking, schedule-induced polydipsia, self-stimulation, kindling, and simple schizophrenia. The Pettigrew paradigm suggests another way to test this model using ERPs, notably P300. The local P300 should be abolished by the 6-hydroxydopamine manipulation. See Part II for these applications.

4. HYPOTHESIS TESTING AND ERROR CORRECTION IN A FLUCTUATING ENVIRONMENT

The stability-plasticity dilemma can also be stated in terms of a system's hypothesis testing or error correction capabilities. How are coding errors corrected, or

adaptations to a changing environment effected, if individual cells cannot even detect that these errors or changes have occurred?

The importance of learned hypotheses in perception and cognition was apparent to Helmholtz over a century ago when he formulated his concept of unconscious inference (Boring, 1950). The inadequacy of traditional physical and mathematical ideas for the purpose of explicating such self-organizing psychological processes has had momentous historical effects since Helmholtz's time. For one, the great interdisciplinary successes of Helmholtz, Maxwell, and Mach in physics and psychology during the last half of the nineteenth century did not inspire interdisciplinary disciples. Instead, the next generation of scientists split physics and psychology into separate scientific streams and psychology entered a century of "turmoil and revolution" (Hilgard and Bower, 1975, p. 2).

During the last decade, experiments on Pavlovian conditioning, notably on the factors that control attentional shifts, have actively probed the unconscious inference issue in a more modern setting. I have in mind the ingenious experiments of, among others, Dickinson *et al.*, (1976), Hall and Pearce (1979), Kamin (1969), Mackintosh *et al.*, (1977), Mackintosh and Reese (1979), Pearce and Hall (1980), Rescorla (1971), and Rescorla and Wagner (1972). Unfortunately, the theories that these workers have suggested to explain their experiments are filled with paradoxes (Grossberg, 1982a). Various popular information processing theories, such as those of Schneider and Schiffrin (1976), also contain serious internal paradoxes because they do not understand the stability-plasticity dilemma (Grossberg, 1978a,b).

I will now summarize some of the Pavlovian experiments because they raise issues about the stability-plasticity dilemma. A thought experiment will consider these issues again from a deeper information processing viewpoint.

5. ATTENTION SHIFTS IN PAVLOVIAN EXPERIMENTS

First, I will summarize some main points about attention shifts by discussing a series of four idealized experiments. Then I will review a striking recent experiment of Mackintosh *et al.* (1977) to illustrate some finer points, as well as the paradoxes into which several scientists have been driven by the pressure of recent data.

FIGURE 1 summarizes the four idealized experiments. Experiment 1 reminds us that an indifferent cue or conditioned stimulus (CS), such as a flashing light, when properly paired with an unconditioned stimulus (US), such as a shock, can be trained to elicit some of the consequences of the US in the form of a conditioned response (CR), such as various indices of fear. Experiment 2 points out that if two equally salient cues, such as a flashing light (CS_1) and a tone (CS_2), appear simultaneously during conditioning trials before the shock (US) occurs, then each of the cues can separately elicit a fearful reaction (CR) on recall trials. The individual cues in a cue complex are thus separately conditionable in some experiments.

Experiments 3 and 4 show that this conclusion is not always true, illustrating the importance of this paradigm for attentional studies. Experiment 3 is a hybrid constructed by performing experiment 1 before experiment 2. In other words, the subject is trained to fear the light before a long series of compound light-tone training trials is undertaken. When the tone is presented on recall trials, it does not elicit a fear reaction, in contrast to experiment 2. Somehow, prior conditioning of the light has "blocked" later conditioning of the tone.

Experiment 4 clarifies the meaning of experiment 3 by altering the US on the compound trials. For example, suppose that US_1, which follows the light, is a

prescribed shock level and that US_2, which follows the tone on compound light-tone trials, is a different shock level. If US_2 is sufficiently different from US_1, then the tone does elicit a conditioned reaction on recall trials. Moreover, if $US_2 > US_1$, then the tone elicits fear, whereas, if $US_2 < US_1$, then the tone elicits relief.

The meaning of these experiments can be summarized in five statements (Grossberg, 1975, 1980a).

1. Many learning subjects are minimal adaptive predictors who only learn about relevant cues. If a subject uses a set of cues, perhaps idiosyncratic to each subject, to generate behavior that elicits expected consequences, then all other cues will be treated as irrelevant. This is why CS_2 in experiment 3 does not condition well; CS_1 already perfectly predicts the US. By contrast, CS_2 in experiment 4 predicts a change in the expected consequence; namely, US_2 rather than US_1.

2. Unexpected consequences somehow redefine the set of relevant cues to which we will pay attention. In experiment 3, US in response to CS_1 is expected, whereas, in experiment 4, US_2 in response to CS_1 is not. That is why CS_2 conditions well in experiment 4 but not in experiment 3.

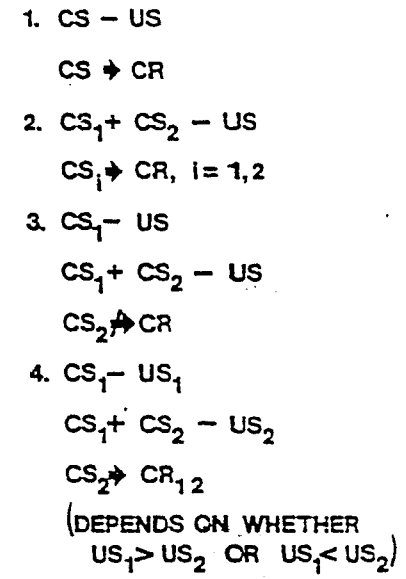


FIGURE 1. Four experiments to illustrate overshadowing. See text.

3. Unexpected consequences often occur after the cues to which we attend have terminated. Somehow these consequences work "backwards in time" on the internal representations of attended and unattended cues to abet the influence of erroneously unattended cues. The conditioned stimuli must therefore be stored in some fashion so that they can be influenced by later unexpected consequences.

4. By its very nature, an unexpected event occurs at a moment when the subject does not know which of the myriad unattended cues should have been attended. Whatever the effects of the unexpected consequence on cue storage, they must affect all the stored representations, since the subject cannot distinguish those which are correct from those which are incorrect. In other words, the novel event influences cue storage by a nonspecific mechanism that somehow differentially influences, or resets, attended cues and unattended cues.

5. Novelty *per se* must be distinguished from what is learned about the experimental situation. Otherwise expressed, STM reset must be distinguished from what is encoded in LTM. For example, both the events $US_2 > US_1$ and $US_2 < US_1$ in

experiment 4 are novel. However, CS_2 can become a source of fear if $US_2 > US_1$ and a source of relief if $US_2 < US_1$.

6. TRANSIENT EFFECTS: CAUSALITY, STM RESET, AND P300

The remarkable experiment of Mackintosh *et al.* (1977) shows that the conditioning situation is still more subtle by focusing on transient conditioning changes. FIGURE 2 summarizes a part of this experiment.

In part 1 of the experiment, all rats experience four trials on which a light (CS) is followed by a shock (US). In part 2 of the experiment, two groups of rats receive an additional single compound light-tone trial. In one group (group I) the light-tone compound is followed by a single shock. In the other group (group II), the light-tone compound is following by two successive shocks that are presented ten seconds apart. A recall trial using the tone alone shows essentially identical fear conditioning to the tone in both groups. In other words, the second shock seems not to have affected tone conditioning.

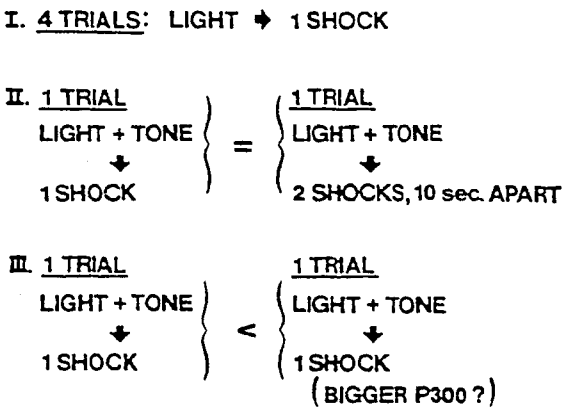


FIGURE 2. Three stages of the 1977 Mackintosh *et al.* experiment. See text.

The remarkable feature of the experiment becomes apparent when two other groups of rats are tested in part 3 of the experiment. One of these groups (group III) receives the same training as group I plus a single compound light-tone trial followed by a single shock before a recall trial using a tone CS is performed. The other group (group IV) receives the same training as group II plus a single compound light-tone trial followed by a single shock before a recall trial using a tone CS is performed. In other words, part 3 of the experiment simply adds an identical learning manipulation onto the group I and II learning paradigms, which by themselves elicited the same reaction to the tone. Remarkably, group IV exhibits better fear conditioning to the tone than group III.

This is a fascinating experimental finding. How can a test after identical second compound trials have different effects if a test after different first compound trials had identical effects? This experiment seems to violate causality on the behaviorally observable level and forces us to conceptualize the behaviorally unobservable mechanisms that underlie attentional shifts.

My explanation of this experiment suggests that the tone on the second compound trial in group IV is more surprising than the tone on the second compound trial in group III and therefore elicits a larger P300 (Grossberg, 1982a).

Mackintosh suggested an explanation of the experiment that led him into a

paradox. I shall quote Pearce and Hall's (1980) summary of Mackintosh's position before indicating that their explanation also leads to a paradox. I mention these paradoxes because ERPs can be used to study more directly the attentional processes whose opaqueness on the behavioral level has led to these paradoxes.

Pearce and Hall (1980, p. 537) state: "According to Mackintosh, the tone conditions normally on the first compound trial in all groups but loses associability when a single shock occurs on this trial, since it predicts this outcome less well than the light." It seems to me that this explanation is either circular or fails to explain the data. Mackintosh says that the tone loses associability because it predicts the single shock less well than the light. However, on the first compound trial, the tone is an even worse predictor of shock than it is on the second trial. Why does the tone condition normally on the first trial if on this trial it is the worst possible predictor of shock, having never before been correlated with shock? The transient conditioning to the tone when it first appears shows that its very unexpectedness can abet its initial conditioning. I suggest below how it does so by resetting STM in a way that elicits a P300.

Pearce and Hall (1980) realize Mackintosh's error. Unfortunately, they build their concepts using the Rescorla-Wagner (1972) learning equation, which does not distinguish STM from LTM effects. Their conclusion (p. 538) is therefore equally paradoxical: "stimuli that fully predict their consequences will be denied access to the processor . . . a stimulus is likely to be processed to the extent that it is not an accurate predictor of its consequences." One implication of this position is that a US that is an excellent predictor of food will be ignored. Thus, Mackintosh emphasizes the expected at the expense of the unexpected, whereas Pearce and Hall make the opposite mistake. What is needed is a theory that shows how STM and LTM mechanisms work together to balance between the processing of expected and unexpected events.

Distinguished ERP scholars are not immune to the error of lumping STM and LTM properties together. Donchin makes the following interesting hypothesis in the EPIC VI panel reports, which must, I believe, be sharpened to distinguish STM and LTM effects before it is freed from paradoxical implications: "P300 amplitude will predict the degree to which the eliciting items will be remembered." I suggest, by contrast, that, when the eliciting item is irrelevant, P300 can predict the degree to which the eliciting item will be extinguished (Section 60). If one reads "remembered as irrelevant" instead of "extinguished," Donchin's assertion can perhaps be salvaged, but this interpretation twists language too much to make the assertion informative. At bottom, this difficulty arises because a large P300 can reflect an STM reset that does not predict what the representations newly stored in STM will encode in LTM. The experimental paradigm as a whole will determine the data that are available for LTM encoding.

7. A THOUGHT EXPERIMENT ABOUT SELF-ORGANIZATION OF SP CODES

The following thought experiment indicates that certain constellations of formal properties should appear together in the data and that certain sequences of cognitive operations should occur that can be tested by searching for a corresponding temporal ordering of these constellations.

Several constellations of properties arise in the thought experiment because it concludes that several design principles work together to correct errors and test hypotheses about codes that satisfy the stability-plasticity (SP) criterion. The thought experiment requires only some general properties from these constellations for its

completion. The mechanisms that subserve these general properties can be specialized in many ways to accomplish specific tasks without destroying the general properties. In this sense, the thought experiment suggests the minimal general purpose machinery needed to generate SP codes. To adapt this general machinery to special task requirements also requires hard work, but work that is greatly abetted by using the general processing mechanisms as a conceptual framework.

The thought experiment has appeared either implicitly or explicitly in several other articles. (Grossberg, 1976b, 1978a, 1980a, 1981a). I repeat it here for two main reasons. First, several of the formal mechanisms that it suggests reflect recent data about ERPs, or describe relationships of ERPs to other experimental measures that have not previously been discussed. Second, several important points which were only briefly discussed in previous expositions are more fully explained here to clarify relationships between cognitive and motivational processes.

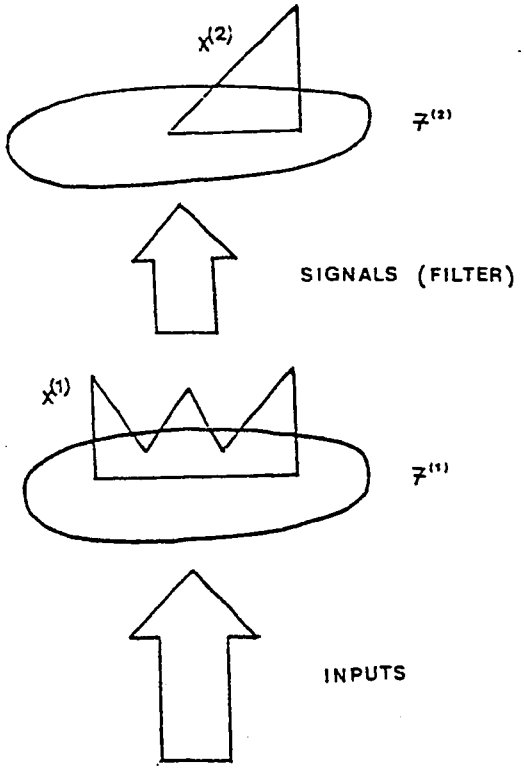


FIGURE 3. The activity pattern $x^{(1)}$ across $\mathcal{F}^{(1)}$ is filtered to elicit a pattern $x^{(2)}$ across $\mathcal{F}^{(2)}$.

8. THE PROBLEM OF STIMULUS EQUIVALENCE

The thought experiment is a story that begins in the middle, rather than the beginning, to avoid an infinite regress. We suppose that certain events have already been "coded," and ask how the system can correct an error that occurs when other events erroneously elicit the same "code." Telling any story requires the choice of an appropriate level of discourse. I consider the processing of *patterns* that are *continuously* fluctuating across space and time within *cellular* tissues. If any one of these italicized words were eliminated, I would have found it impossible, or at best unintuitive, to even state what the design problems are, let alone to solve them. In particular, considering feature detectors instead of patterns, binary rather than continuous data, or linear systems rather than cells would cause a serious reduction of mechanistic insight, and has indeed caused serious internal problems in theories that

embrace alternative languages as fundamental. I analyze some of these problems in Grossberg (1978a,b,c, 1980a,b,c, 1981b, 1982a,b).

Having chosen a level of discourse and a point of departure, we can begin our story. In FIGURE 3, two successive stages of processing are depicted. The reader can fix ideas by thinking of the first stage, $\mathcal{F}^{(1)}$, as a specific thalamic nucleus and the second stage, $\mathcal{F}^{(2)}$, as cortical, or of $\mathcal{F}^{(1)}$ and $\mathcal{F}^{(2)}$ as two successive cortical stages of processing. Suppose that, due to prior developmental trials, the pattern of activity, or potential, $x^{(1)}$, across the cells of $\mathcal{F}^{(1)}$ elicits signals to $\mathcal{F}^{(2)}$ that generate a characteristic pattern, or internal representation, $x^{(2)}$, of $x^{(1)}$ across $\mathcal{F}^{(2)}$. Think of $x^{(2)}$ as representing $x^{(1)}$ across $\mathcal{F}^{(2)}$.

For definiteness, suppose that the $\mathcal{F}^{(1)} \longrightarrow \mathcal{F}^{(2)}$ signals are elicited in the simplest possible way; in fact, a way that corresponds to what we know from elementary physiology. Let each activity at a cell (population) of $\mathcal{F}^{(1)}$ be capable of generating an excitatory signal to all the cells in $\mathcal{F}^{(2)}$ to which it is connected, and let each cell in $\mathcal{F}^{(2)}$ add all the signals that it receives in this way. This simple rule implies that the signals from $\mathcal{F}^{(1)}$ to $\mathcal{F}^{(2)}$ act like a linear filter of the pattern, $x^{(1)}$, across $\mathcal{F}^{(1)}$. Moreover, if the $\mathcal{F}^{(1)} \longrightarrow \mathcal{F}^{(2)}$ pathways significantly diverge from each cell at $\mathcal{F}^{(1)}$ and converge at each cell of $\mathcal{F}^{(2)}$, then infinitely many patterns, $x^{(1)}$, can be represented by the same pattern, $x^{(2)}$. This simple fact raises the following fundamental problem.

Problem of Stimulus Equivalence. How does the system decide which patterns across $\mathcal{F}^{(1)}$ should be allowed to have equivalent effects, notably the same observable behavior, due to equivalent processing at higher network levels?

9. CATEGORICAL PERCEPTION, BAYSIAN STATISTICS, AND TEMPORALLY UNSTABLE FEEDFORWARD CODING

This section considers the simplest version of the stimulus equivalence problem to clarify some issues. This section can be skipped on a first reading.

First we need some notation. Let the activity of the ith cell (population) $v_i^{(1)}$ of $\mathcal{F}^{(1)}$ be denoted by $x_i^{(1)}$. Let the interaction strength of the pathway (axons) from $v_i^{(1)}$ in $\mathcal{F}^{(1)}$ to $v_j^{(2)}$ in $\mathcal{F}^{(2)}$ be denoted by z_{ij}. The simplest rule for generating a signal from $v_i^{(1)}$ to $v_j^{(2)}$ is to let the signal strength be $x_i^{(1)} z_{ij}$. Then the *total* input from $\mathcal{F}^{(1)}$ to $v_j^{(2)}$ is

$$T_j = \sum_{i=1}^{n} x_i^{(1)} z_{ij}. \tag{1}$$

Suppose, for definiteness, that $\mathcal{F}^{(2)}$ chooses the maximal input which it receives for STM storage and suppresses all other activities across $\mathcal{F}^{(2)}$; that is,

$$x_j^{(2)} = \begin{cases} 1 & \text{if } T_j > \max\limits_{k \neq j} T_k \\ 0 & \text{if } T_j < \max\limits_{k \neq j} T_K \end{cases}. \tag{2}$$

It is then easily shown that a convex set P_j of patterns $x^{(1)}$ across $\mathcal{F}^{(1)}$ will activate the same population $v_j^{(2)}$ of $\mathcal{F}^{(2)}$. In other words, a filter followed by a choice implies categorical perception of patterns. The generality of this result argues against categorical perception as a phenomenon peculiar to speech (Studdert-Kennedy, 1980; Studdert-Kennedy *et al.*, 1970). The category that codes a pattern across $\mathcal{F}^{(1)}$ switches when the pattern is deformed so much that it crosses the boundary of some set P_j.

Equation 2 has various pleasant properties. It is a Baysian rule for minimizing risk

in the presence of uncertain data (Duda and Hart, 1973). Two successive filtering stages followed by a choice can encode significant global invariants of spatially distributed data (Grossberg, 1978a, Section 19), notably recognitions that are independent of the pattern's position in space (Fukushima, 1980). Given these pleasant properties in even the simplest examples, why do we need to go any further than a classification of the properties of a few feedforward STM storage rules like (2)?

The answer emerges only when we consider the stability-plasticity dilemma. Then we must study how the interaction strength vectors $z_j = (z_{1j}, z_{2j}, \ldots z_{nj})$ change due to learning as a sequence of input patterns perturbs the network. Otherwise expressed, we must study the learning of new categorical boundaries. Various workers (Anderson *et al.*, 1977; Fukushima, 1980; Malsburg, 1973; Pérez *et al.*, 1975) have studied this problem on computers, using small numbers of input patterns, small numbers of cells in $\mathcal{F}^{(2)}$, and some version of the associative learning law which I introduced in Grossberg (1964, 1967, 1968). What the computer studies do not reveal is that this learning rule is temporally unstable if the class of input patterns is significantly larger than the number of cells in $\mathcal{F}^{(2)}$ (Grossberg, 1976a). Given such inputs, the coded meaning of a given pattern $x^{(1)}$ can continually change at $\mathcal{F}^{(2)}$; its pattern class P_j can change through time. This property makes it impossible to build up a temporally stable hierarchy of codes without further structure; the coding pyramid would be built on perceptual quicksand.

After this theorem was proved, I could see that the feedback mechanism needed to stabilize a code developing in response to an arbitrary input environment was just like the feedback mechanism that I had earlier derived to explain overshadowing data in adult Pavlovian experiments (Grossberg, 1975). The need for the same type of feedback mechanism in infant development and cognitive coding as well as in attentional examples encouraged me to strip away unnecessary details to derive the mechanism in general information processing terms. The thought experiment is the result.

I noted above that categorical boundaries can shift due to changes in the LTM traces, z_{ij}. This is not the only way to alter categorical boundaries. Section 37 will indicate how the boundaries can shift due to an interaction between habituation of transmitter gates and STM competition. Categorical shift mechanisms are often summarized under the single rubic of "adaptation." This terminology is insufficient because the shift due to habituation is functionally distinct from the shift due to new LTM encoding.

10. UNCONSCIOUS INFERENCES: WHY DO LEARNED FEEDBACK EXPECTANCIES EXIST?

After pattern $x^{(1)}$ has been trained to elicit pattern $x^{(2)}$ across $\mathcal{F}^{(2)}$, suppose that a new pattern erroneously elicits a pattern (equivalent to) $x^{(2)}$ across $\mathcal{F}^{(2)}$. How is this coding error corrected? To discuss this situation, I introduce a clearer notation. Denote $x^{(1)}$ by $x_1^{(1)}$, $x^{(2)}$ by $x_1^{(2)}$, the new pattern by $x_2^{(1)}$, and its representation across $\mathcal{F}^{(2)}$ by $x_2^{(2)}$. I write $x_1^{(2)} = x_2^{(2)}$ to summarize that the representations of $x_1^{(1)}$ and $x_2^{(1)}$ are equivalent at $\mathcal{F}^{(2)}$.

Defining the concept of "error" in all its ramifications is itself a formidable task. I will approach this definition by imposing only the simplest processing requirement: Rapidly shut off $x_1^{(2)}$ if it is in error to prevent further processing, notably observable behavior, from being erroneously elicited by $\mathcal{F}^{(2)}$ output. This processing requirement focuses upon the internal consistency of the code across both stages $\mathcal{F}^{(1)}$ and $\mathcal{F}^{(2)}$,

notably on the proper choice of stimulus equivalence classes at $\mathcal{F}^{(1)}$ for patterns at $\mathcal{F}^{(2)}$.

Reinforcement contingencies bear upon this issue only indirectly, although they will be seen in Section 30 to activate analogous mechanisms, albeit in distinct anatomies. To realize that internal consistency and reinforcement are distinct issues, let us contrast two situations: one in which the patterns ($x_1^{(1)}$ and $x_1^{(2)}$) are rewarded on early trials before being punished on later trials; the other in which the patterns ($x_1^{(1)}$ and $x_1^{(2)}$) are consistently rewarded on early trials, after which the patterns ($x_2^{(1)}$ and $x_1^{(2)}$) are consistently punished. In the former case, the world switches from reward to punishment of $x_1^{(1)}$. In the latter case, $x_1^{(1)}$ is consistently rewarded and $x_2^{(1)}$ is consistently punished. Changing the $\mathcal{F}^{(2)}$ representation of $x_1^{(1)}$ when punishment occurs is not adaptive in the first case, since the representation $x_1^{(2)}$ should be associated with punishment. Changing the $\mathcal{F}^{(2)}$ representation of $x_2^{(1)}$ when punishment occurs is adaptive in the second case, reflecting the fact that $x_1^{(1)}$ is consistently rewarded, whereas $x_2^{(1)}$ is consistently punished.

How does the system know this difference, despite the fact that, in both cases, the same pattern $x_1^{(2)}$ across $\mathcal{F}^{(2)}$ and the same temporal ordering of reward and punishment occur? How can the system as a whole detect when to recode $x_2^{(1)}$ at $\mathcal{F}^{(2)}$ by a new pattern $x_2^{(2)}$ ($\neq x_1^{(2)}$) despite its earlier predilection to erroneously choose pattern $x_1^{(2)}$?

With these remarks as background, our first robust conclusion becomes clear. The error cannot be corrected using only $\mathcal{F}^{(2)}$ as a source of data. So far as $\mathcal{F}^{(2)}$ knows, when $x_1^{(2)}$ is active, the correct pattern, $x_1^{(1)}$, is active across $\mathcal{F}^{(1)}$ and no error has occurred. In other words, the knowledge that an error has occurred resides in $\mathcal{F}^{(1)}$; in particular, in the fact that $x_2^{(1)}$, not $x_1^{(1)}$, is active across $\mathcal{F}^{(1)}$ when $x_1^{(2)}$ is active across $\mathcal{F}^{(2)}$.

How does the system know this? On an error trial when $x_1^{(2)}$ occurs in $\mathcal{F}^{(2)}$, $x_1^{(1)}$ is not presented to $\mathcal{F}^{(1)}$. To compute an error, the system must somehow readout $x_1^{(1)}$ across $\mathcal{F}^{(1)}$ to compare $x_1^{(1)}$ with $x_2^{(1)}$. Pattern $x_1^{(2)}$ is the only possible source of this readout in the present framework.

To understand how a readout can occur, we need to reconsider the developmental trials on which $x_1^{(1)}$ was presented to the system. On these trials, the system adjusted its adaptive filter to code $x_1^{(1)}$ by $x_1^{(2)}$. Now we conclude that, as adaptive filtering proceeded, pattern $x_1^{(2)}$ emitted signals from $\mathcal{F}^{(2)}$ to $\mathcal{F}^{(1)}$ whose pathways encoded the pattern $x_1^{(1)}$ in LTM (FIGURE 4). In this way, $x_1^{(2)}$ learns the pattern that it expects to find across $\mathcal{F}^{(1)}$ due to developmental experience. This learned feedback expectancy, or template, plays the role of an unconscious inference in the theory. In the context of the stability-plasticity dilemma, the mysterious notion of expectancy becomes a processing constraint on testing whether an error has occurred.

Tolman's claim that subjects learn expectations or plans rather than habits (Tolman, 1932) also gains a more concrete meaning in this setting. Expectancy learning is, however, only part of a larger story, which clarifies both the genius and the limitations of classical theories that correctly isolated important processing fragments, but often at the price of being unable to wed them to the fragments that were prized by competing theoretical positions.

11. PROCESSING NEGATIVITY AND MATCH DETECTION

FIGURE 5 depicts the rapid sequence of events to which we have been led. In FIGURE 5a, feedforward inputs are encoded by pattern $x_2^{(1)}$ across $\mathcal{F}^{(1)}$. This pattern is filtered by the $\mathcal{F}^{(1)} \rightarrow \mathcal{F}^{(2)}$ pathways and elicits pattern $x_1^{(2)}$ across $\mathcal{F}^{(2)}$ (FIGURE 5b). Pattern

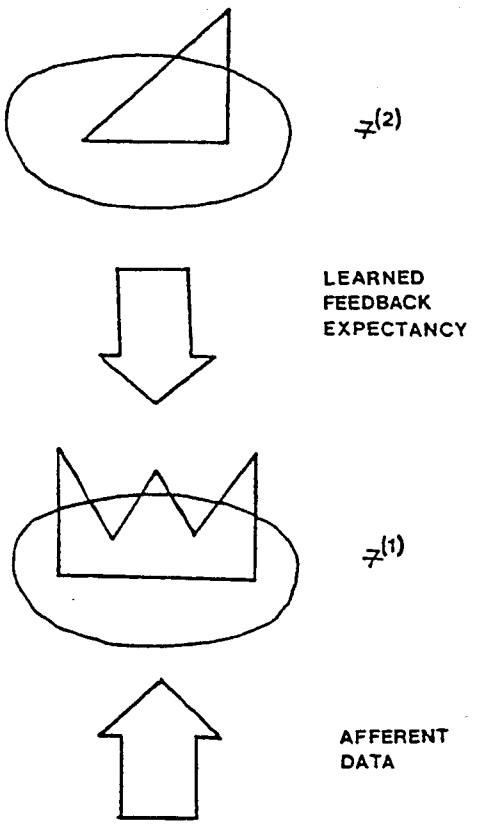


FIGURE 4. The pattern $x_1^{(2)}$ across $\mathcal{F}^{(2)}$ elicits a feedback pattern $x_1^{(1)}$ to $\mathcal{F}^{(1)}$, which is the pattern that is sampled across $\mathcal{F}^{(1)}$ during previous developmental trials. The field $\mathcal{F}^{(1)}$ is an interface where feedforward data and learned feedback expectancies are compared.

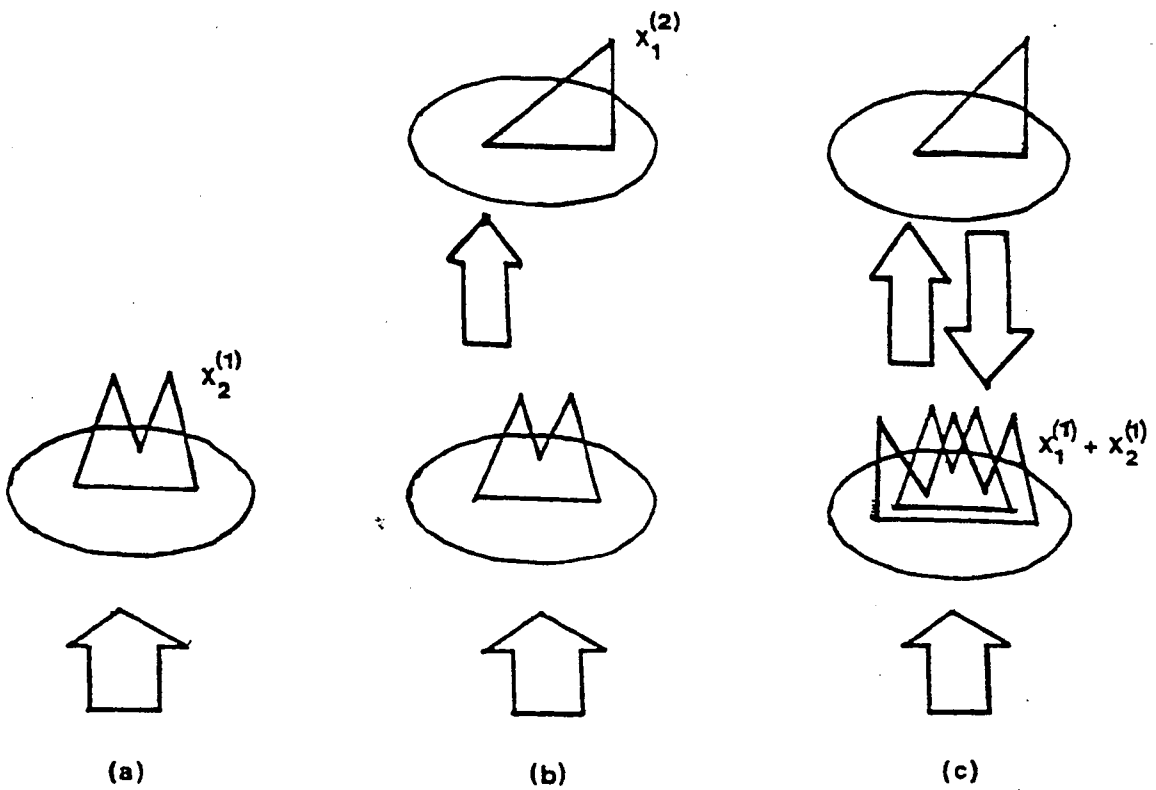


FIGURE 5. Stages (a), (b), and (c) schematize the rapid sequence of events whereby feedforward data is filtered and activates a feedback expectancy that is matched against itself.

$x_1^{(2)}$ thereupon reads out its learned feedback expectancy across $\mathcal{F}^{(1)}$ (FIGURE 5c). To fully understand how this happens, we need to study adaptive filtering (Von der Malsburg, 1973; Grossberg, 1976a; Pérez *et al.*, 1975) and associative pattern learning by parallel sampling sources (Grossberg, 1969a, 1970, 1972c, 1974). For present purposes, we are more interested in an intuitive constellation of properties than in their generative mathematical mechanism.

The expectancy should manifest itself in a constellation of four properties. (1) It is carried by *specific* pathway; that is, a pathway which reads out differentiated patterns. (2) It is carried by a *conditionable* pathway. This will only be apparent when new items are being encoded. I think that such a conditionable pathway would probably carry a cholinergic transmitter (Section 30). (3) It is carried by a *feedback* pathway. (4) It is activated when a *match* occurs between actual and expected data.

I associate this formal event with the processing negativity or match detector or selection negativity component that is discussed by Ritter *et al.* (1983). Testing the conditionability of this component might be possible using a variant of the following experiment. Present the same configuration of n lights to the left visual field on each trial. Use enough lights to make learning the configuration possible but nontrivial. Present n lights to the right visual field but in a different configuration on each trial. Choose a task that requires intermittent attention to each visual field. If processing negativity is independent of stimulus probability, then varying the lights in the right visual field should cause no change in this component. If processing negativity is conditionable, a progressive enhancement of this component should occur as the light configuration to the left visual field is learned.

12. NOVEL EVENTS TRIGGER NONSPECIFIC AROUSAL

Given that $\mathcal{F}^{(2)}$ can read out a learned expectancy to $\mathcal{F}^{(1)}$, both $x_1^{(1)}$ and $x_2^{(1)}$ can be simultaneously activated across $\mathcal{F}^{(1)}$, despite the fact that only $x_2^{(1)}$ is presented by external means.

We can now ask the next design question: How does mismatch of $x_1^{(1)}$ and $x_2^{(1)}$ at $\mathcal{F}^{(1)}$ shut off the incorrect code $x_1^{(2)}$ at $\mathcal{F}^{(2)}$? At this point, we need to realize that, just as the information available at $\mathcal{F}^{(2)}$ is limited, so also the information available at $\mathcal{F}^{(1)}$ is limited, and that the two types of limitations, being complementary, can be overcome when $\mathcal{F}^{(1)}$ and $\mathcal{F}^{(2)}$ work together. At $\mathcal{F}^{(2)}$, we cannot detect that an error has occurred. At $\mathcal{F}^{(1)}$, we can detect that an error has occurred by computing the mismatch between $x_1^{(1)}$ and $x_2^{(1)}$. However, we do not know which cells across $\mathcal{F}^{(2)}$ caused the error. Any pattern whatsoever across $\mathcal{F}^{(2)}$ could have read out the erroneous template $x_1^{(1)}$. Whatever cells at $\mathcal{F}^{(2)}$ are active across $\mathcal{F}^{(2)}$ when mismatch occurs at $\mathcal{F}^{(1)}$ should be inhibited. Since $\mathcal{F}^{(1)}$ cannot know which cells in $\mathcal{F}^{(2)}$ are active, mismatch across $\mathcal{F}^{(1)}$ must elicit a nonspecific event (*viz.*, the same signal) to every cell in $\mathcal{F}^{(2)}$. Somehow, the internal organization of $\mathcal{F}^{(2)}$ will respond to this nonspecific event by selectively inhibiting its active cells. I will call the nonspecific event nonspecific arousal to make contact with classical physiological vocabulary.

I conclude that an unexpected or novel event triggers a burst of nonspecific arousal that is calibrated by a mismatch between a feedback expectancy and feedforward data. This conclusion reminds us that unexpected consequences in the Pavlovian experiments of Section 5 trigger a nonspecific event that resets the balance between overshadowed and attended cues.

13. MISMATCH NEGATIVITY (N_{200})

I will interpret activation of the nonspecific arousal source as a formal analog of the mismatch negativity component of the N_{200} that is discussed by Ritter *et al.* (1983). This type of mismatch negativity satisfies the following constellation of four formal properties that are orthogonal to the formal properties of processing negativity. (1) It is carried by a *nonspecific* pathway; namely, one that reads out the same signal to every recipient cell. By "nonspecific," I do not necessarily imply "intermodal," only "equal." I think that such a pathway would probably carry a catecholaminergic transmitter (Section 26). (2) It is carried by an *unconditionable* pathway. (3) It is carried by a *feedforward* pathway. (4) It is activated when a *mismatch* occurs between actual and expected data.

The possible relationship of mismatch negativity to the orienting reaction will become clearer in Section 16.

14. THE NOISE-SATURATION DILEMMA: NOISE SUPPRESSION AND PATTERN MATCHING IN SHUNTING ON-CENTER OFF-SURROUND NETWORKS

We have concluded that mismatch at $\mathcal{F}^{(1)}$ triggers a burst of nonspecific arousal to $\mathcal{F}^{(2)}$. We must now ask, How does this happen? Our first step is to ask, How does mismatch at $\mathcal{F}^{(1)}$ transform the activity across $\mathcal{F}^{(1)}$? Our next step is to ask, How does the matching transformation at $\mathcal{F}^{(1)}$ trigger arousal to $\mathcal{F}^{(2)}$?

We can now assert that pattern mismatch at $\mathcal{F}^{(1)}$ shuts off activity across $\mathcal{F}^{(1)}$. Suppose not. Since both $x_1^{(1)}$ and $x_2^{(1)}$ elicit $x_1^{(2)}$ across $\mathcal{F}^{(2)}$, leaving these patterns on across $\mathcal{F}^{(1)}$ would lock the system into an uncorrectable error and allow $x_2^{(1)}$ to train its $\mathcal{F}^{(1)} \rightarrow \mathcal{F}^{(2)}$ filter to better elicit the error $x_1^{(2)}$, using the same LTM mechanism that $x_1^{(1)}$ used to train the filter to elicit $x_1^{(2)}$ on its developmental trials. Rapidly shutting off $\mathcal{F}^{(1)}$ prevents this erroneous adaptive filtering from occurring and will provide a basis for triggering nonspecific arousal to $\mathcal{F}^{(1)}$.

One might legitimately worry at this stage that designing a mismatch detector is too sophisticated an evolutionary task. In reply, I will point out that mismatch detection is part of a constellation of properties whose mechanism solves a fundamental design problem. This design problem, which I call the noise-saturation dilemma (Grossberg, 1973, 1978d; 1980a) confronts all cellular tissues and must be solved before continuously fluctuating patterns can be registered at all. Once the problem is frontally attacked, one realizes that a host of properties, such as matching, masking, normative drifts, tuning, spatial frequency detection, filling-in, lightness computation, STM temporal order information, STM resonance, travelling or standing waves, all emerge in a coherent way as special processing capabilities of this general purpose mechanism. See Grossberg (1981a, Sections 10 to 27) for a recent review. All too many recent models, notably in artificial intelligence, have failed to realize this basic fact, which is one reason why every property in these models is based on a separate computational trick. Throughout this article, I will insist on principled solutions to our design problems rather than *ad hoc* tricks.

The noise-saturation dilemma is easy to state because it is so basic. Consider FIGURE 6. In FIGURE 6a, a differentiated pattern of inputs, I_i, is poorly registered in the cell activities, x_i, because the overall input intensity level is too small to override internal cellular noise. In FIGURE 6b, all the inputs are proportionally amplified to escape the cells' noisy range without destroying relative input importance (as when the

reflectances of a picture remain constant despite its illumination at successively higher light intensities). Since cells have finitely many excitable sites, the smallest inputs are now large enough to turn on all the sites of their receptive cells; hence, the larger inputs might not be able to differentially excite their receptive cells, since there might be no more sites to turn on in these cells. The input differences are thereby lost because the activities saturate as all the cell sites are activated. Thus, in a cellular tissue, sensitivity loss can occur at both low and high input intensities. As the inputs fluctuate between these extremes, the possibility of accurately registering input patterns is imperiled.

I have elsewhere proved that mass action competitive networks can automatically retune their sensitivity as inputs fluctuate to register input differences without noise or saturation contaminants. In a neural context, these systems are called shunting on-center off-surround networks. Otherwise expressed, a network whose cells obey membrane equations (not additive equations) and interact *via* an anatomy with a narrow excitatory focus and broadly distributed inhibition can automatically retune its

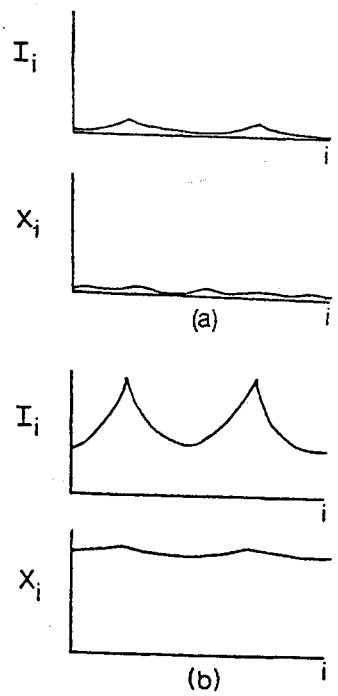


FIGURE 6. The noise-saturation dilemma. (a) At low input intensities, the input pattern $(I_1, I_2, \ldots I_n)$ is poorly registered in the activity pattern $(x_1, x_2, \ldots x_n)$ because of noise. (b) At high input intensities, the input pattern is poorly registered in the activity pattern because of saturation.

sensitivity due to automatic gain control by the inhibitory signals. In this processing context, one proves that all the variegated properties mentioned above hold.

For the present purposes, we need only notice that a noise suppression mechanism implies a pattern matching mechanism. FIGURE 7a depicts a network that responds to a uniform pattern by suppressing it. This is easily accomplished in a mass action competitive network. Intuitively, noise suppression means that zero spatial frequency patterns are inhibited or that inputs that do not discriminate one feature detector from another are inhibited.

Given the property of noise suppression, mismatched input patterns are inhibited (FIGURE 7b) because their peaks and troughs add to create an approximately uniform total input pattern, and is hence inhibited as noise. No sustained processing negativity occurs in this case. By contrast, it can be proved that the sum of matched patterns is amplified (FIGURE 7c) by an interaction between reflectance processing and Weber law modulation. Processing negativity is thereby elicited.

At this point, the reader might rightly wonder: How mismatched must the patterns

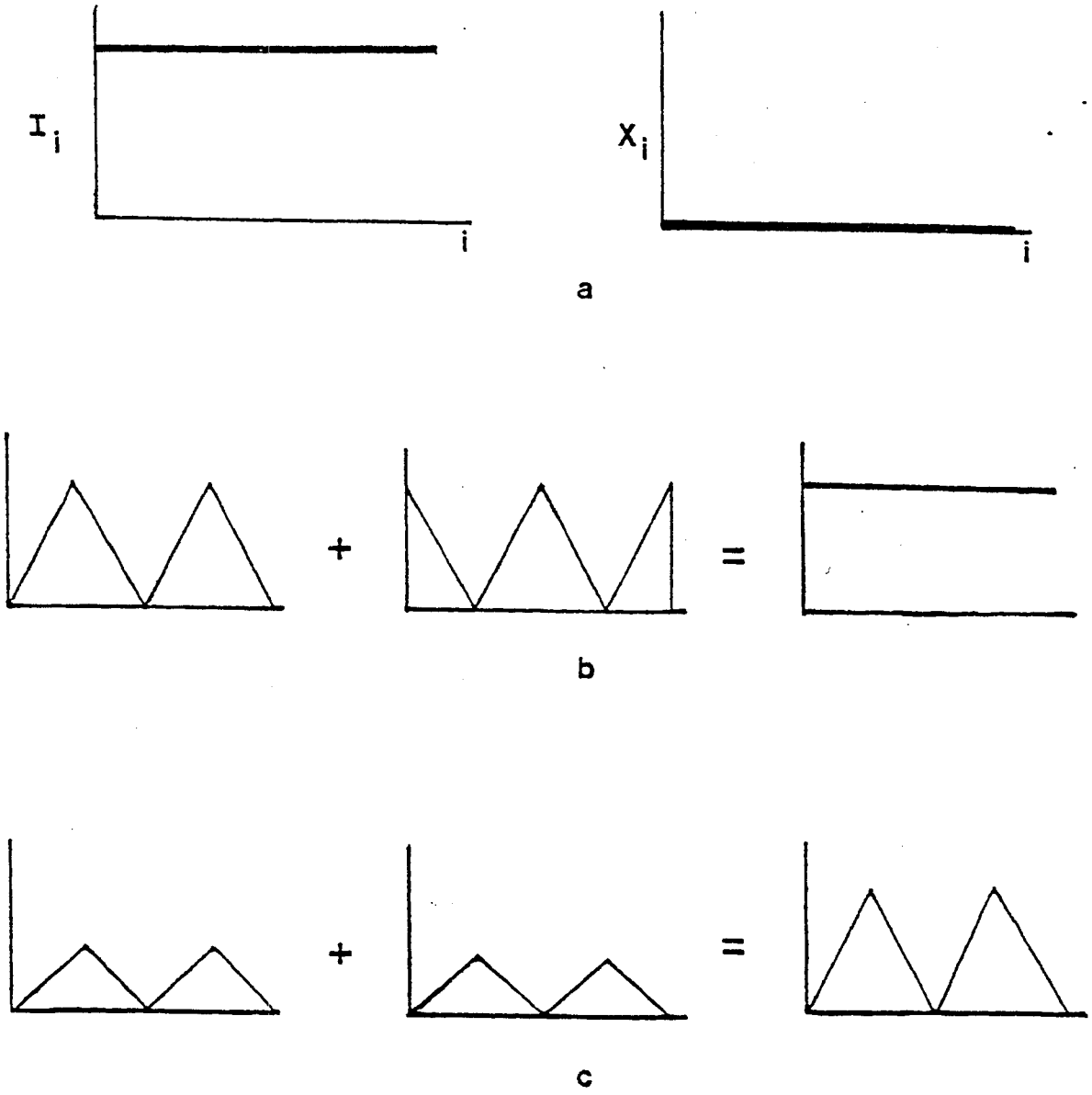


FIGURE 7. In (a), noise suppression converts a uniform (or zero spatial frequency) input pattern into a zero activity pattern. In (b), two mismatched patterns add to generate an approximately uniform total input pattern, which is suppressed by the mechanism of (a). In (c), two matched input patterns add to yield a total input pattern that is more active than either pattern taken separately.

be before they are significantly inhibited? How "uniform" is uniform in these mass action competitive systems? This is not an idle question because the whole point about these systems is that they can be retuned. A signal to this system that nonspecifically modulates the gain, or sensitivity, of the inhibitory interneurons within $\mathcal{F}^{(1)}$ can shift the size of the criterion, or quenching threshold (QT), that determines how uniform the pattern must be before being quenched (Grossberg, 1973, 1981a). Even if reinforcement affected only the QT of $\mathcal{F}^{(1)}$, it could cause recoding of $x_2^{(1)}$ across $\mathcal{F}^{(2)}$. Controlling the QT of $\mathcal{F}^{(1)}$ and $\mathcal{F}^{(2)}$ *via* nonspecific inhibitory gain control is one way to refine categorical boundaries at one extreme, or to shut off $\mathcal{F}^{(1)}$ and $\mathcal{F}^{(2)}$ entirely during intermodality attention shifts and sleep at the other extreme.

15. DISINHIBITION OF MISMATCH NEGATIVITY BY UNEXPECTED EVENTS

Having shut off activity across $\mathcal{F}^{(1)}$ due to pattern mismatch, we now ask how this event can trigger nonspecific arousal to $\mathcal{F}^{(2)}$. What is the source of the activity that energizes the arousal pulse? Is it endogenous (internally driven and tonic) or exogenous (externally driven and phasic)? If it were endogenous, arousal would flood $\mathcal{F}^{(2)}$ whenever $\mathcal{F}^{(1)}$ was inactive, whether due to active pattern mismatch or passive inactivity. This conclusion is unacceptable. Somehow the system must know the difference between inactivity due to active mismatch, which should trigger arousal, and passive inactivity, which should not.

If the arousal is delivered exogenously, or with the input, this problem is averted and leads to a classical physiological conclusion (Hebb, 1955). In FIGURE 8, the input pathway bifurcates before reaching $\mathcal{F}^{(1)}$. One branch, as before, delivers specific information to $\mathcal{F}^{(1)}$ that is progressively evaluated in the hierarchy $\mathcal{F}^{(2)}, \mathcal{F}^{(3)}, \ldots$. The other branch activates the arousal source $\mathcal{A}$. Given that $\mathcal{A}$ is activated whenever an input is processed, why doesn't $\mathcal{A}$ release arousal to $\mathcal{F}^{(2)}$ unless mismatch occurs at $\mathcal{F}^{(1)}$? In light of Section 14, we can rephrase this question as follows: How does activity across $\mathcal{F}^{(1)}$ suppress output from $\mathcal{A}$, and inhibition of activity across $\mathcal{F}^{(1)}$ release output from $\mathcal{A}$? Clearly, the cells across $\mathcal{F}^{(1)}$ send inhibitory pathways to $\mathcal{A}$ that attenuate activity when $\mathcal{F}^{(1)}$ is active. The system can differentiate between active mismatch and passive inactivity because activity across $\mathcal{F}^{(1)}$ inhibits $\mathcal{A}$, whereas mismatch at $\mathcal{F}^{(1)}$ disinhibits $\mathcal{A}$.

We have been led to postulate the following rapid sequence of events (FIGURE 9). The input activates pattern $x_2^{(1)}$ at $\mathcal{F}^{(1)}$ and the arousal source $\mathcal{A}$ (FIGURE 9a). Pattern $x_2^{(1)}$ inhibits $\mathcal{A}$ and is filtered by the specific pathway $\mathcal{F}^{(1)} \longrightarrow \mathcal{F}^{(2)}$, thereby activating $x_1^{(2)}$ across $\mathcal{F}^{(2)}$ (FIGURE 9b). Pattern $x_1^{(2)}$ reads out its learned feedback expectancy $x_1^{(1)}$

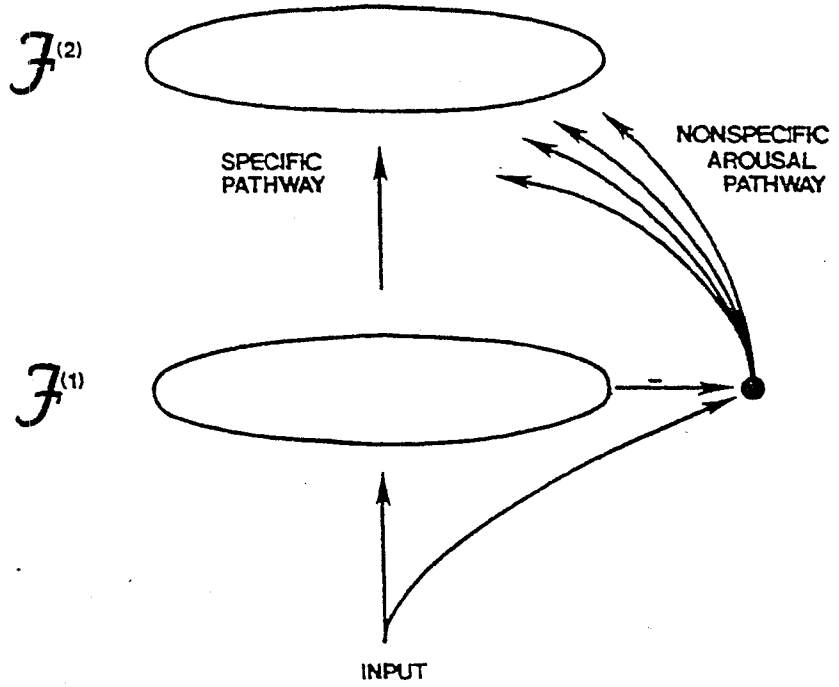


FIGURE 8. The input data bifurcates into a specific pathway that carries the data's cue or feature information and a nonspecific pathway that activates a source of arousal.

across $\mathcal{F}^{(1)}$, whence pattern matching is initiated (FIGURE 9c). Mismatch causes inhibition of $\mathcal{F}^{(1)}$, which disinhibits $\mathcal{A}$ and unleases a burst of nonspecific arousal (mismatch negativity) upon $\mathcal{F}^{(2)}$.

This interpretation of the generator of mismatch negativity adds another verifiable

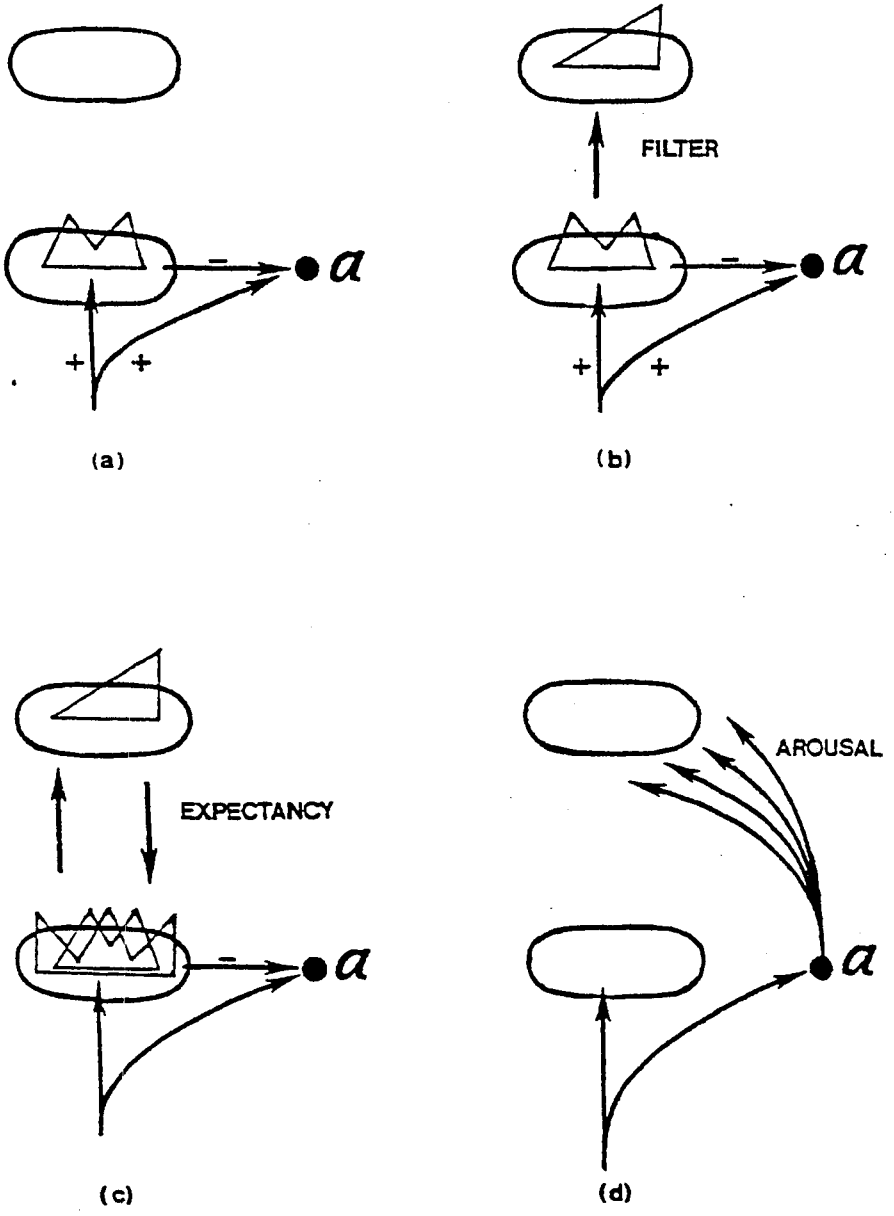


FIGURE 9. In (a), feedforward data elicit activity across $\mathcal{F}^{(1)}$ and an input to the arousal source $\mathcal{A}$, which is rapidly inhibited by $\mathcal{F}^{(1)}$. In (b), the pattern at $\mathcal{F}^{(1)}$ maintains inhibition of $\mathcal{A}$ as it is filtered and activates $\mathcal{F}^{(2)}$. In (c), the feedback expectancy from $\mathcal{F}^{(2)}$ is matched against the pattern at $\mathcal{F}^{(1)}$. In (d), mismatch attenuates activity across $\mathcal{F}^{(1)}$ and thereby disinhibits $\mathcal{A}$, which releases a nonspecific arousal signal to $\mathcal{F}^{(2)}$.

property to its constellation. The source of arousal should be a feedforward pathway that bifurcates off the specific thalamocortical pathway that processes the input. Which feedforward pathway should be transected to selectively abolish mismatch negativity?

16. ATTENTIONAL VS. ORIENTING SUBSYSTEMS: ERROR PERSEVERATION, P300, AND THE HIPPOCAMPUS

The bifurcation of input pathways into specific and nonspecific branches is a special case of a general network design (Grossberg, 1975, 1978a). The specific branch $\mathcal{F}^{(1)}, \mathcal{F}^{(2)}, \ldots$ is part of the network's attentional subsystem for refining the processing of expected events. The nonspecific branch $\mathcal{A}$ is part of the network's orienting subsystem for reorganizing the network in response to unexpected events.

The reader might legitimately protest: Why do you need two distinct subsystems? Why not just let mismatch at $\mathcal{F}^{(1)}$ directly shut off the pattern at $\mathcal{F}^{(2)}$? The answer to this question contains one of the most important processing insights of this article. If this suggestion were implemented, shutting off the pattern at $\mathcal{F}^{(2)}$ would deactivate the feedback expectancy $x_1^{(1)}$ from $\mathcal{F}^{(2)}$ to $\mathcal{F}^{(1)}$, thereby reinstating $x_2^{(1)}$ across $\mathcal{F}^{(1)}$. The cycle of erroneous $x_1^{(2)}$ coding of $x_2^{(1)}$ would begin again, and the network would perseverate in an uncorrectable error. The orienting subsystem overcomes the error perseveration problem that would occur using direct reset of $\mathcal{F}^{(2)}$.

From this perspective, it is of interest that hippocampectomized rats do not orient to a novel stimulus while they are indulging in a consummatory activity, such as running towards a reward. They cannot "shift attention during the presentation of a novel stimulus or in a mismatch situation" (O'Keefe and Nadel, 1978, p. 250).

Another interesting connection can be mentioned at this time at the risk of leaping ahead too fast. I will conclude in Section 21 that the burst of mismatch negativity can reset STM and thereby elicit a P300. This fact raises the question, Given that the arousal burst that elicits a formal P300 is part of the orienting subsystem, what is the relationship between the P300 and the orienting response (OR)? This issue is discussed in the chapter on Orienting and P300 by Donchin *et al.* (this volume). In light of FIGURE 8, the question can be mechanistically translated as: What is the relationship between an arousal burst from $\mathcal{A}$ and the OR? A partial answer is suggested in Grossberg (1978a) using the concept of a QT mentioned in Section 14.

I suggest that activation of $\mathcal{A}$ can trigger an arousal burst that is funneled (directly or indirectly) into several pathways: One pathway resets $\mathcal{F}^{(2)}$, as in FIGURE 9. For definiteness, suppose that another pathway nonspecifically sensitizes those networks at which the terminal motor maps that control spatial orientation are subliminally fluctuating through time. This nonspecific gain control change lowers the QT of these networks. Their terminal motor maps are bootstrapped from a subliminal status to supraliminal reverberation in motor STM. The supraliminal motor patterns can thereupon read out motor commands that drive the spatial orientation process.

The theory suggests that a strong functional link may exist between P300 and at least one aspect of the OR. This link anatomically takes the form of the generator of mismatch negativity and physiologically is expressed as a nonspecific activity that drives sensory STM reset in the attentional subsystem and gain changes such as those which regulate the storage of commands in motor STM in the orienting subsystem.

17. PARALLEL HYPOTHESIS TESTING IN REAL TIME: STM RESET AND RENORMALIZATION

Our next design problem is: How does the increment in nonspecific arousal selectively shut off active cells across $\mathcal{F}^{(2)}$? This mechanism must possess three properties.

(1) Selective inhibition of active cells: Only the active cells read out the expectancy to $\mathcal{F}^{(1)}$ that may mismatch afferent data there. A mismatch implies that an erroneous category at $\mathcal{F}^{(2)}$ is active; hence, it must be suppressed. Inactive cells at $\mathcal{F}^{(2)}$ should not be suppressed both because they did not read out the expectancy and because they must be available for possible coding of $x_2^{(1)}$ during the next time interval. Otherwise the error correction process would grind to a halt.

(2) Enduring inhibition of active cells: The arousal-initiated inhibition of cells across $\mathcal{F}^{(2)}$ must be enduring as well as selective to prevent error perseveration. Otherwise, as soon as $x_1^{(2)}$ is inhibited, the feedback expectancy $x_1^{(1)}$ would be shut off, $x_2^{(1)}$ would be unmasked across $\mathcal{F}^{(1)}$ and would reinstate $x_1^{(2)}$ across $\mathcal{F}^{(2)}$ once again. To prevent error perseveration, the inhibited cells must stay inhibited long enough for $x_2^{(1)}$ to activate a distinct pattern across $\mathcal{F}^{(2)}$ during the next time interval. The inhibition is, therefore, slowly varying compared to the time scale of filtering, feedback expectancy, and mismatch.

Once a selective and enduring inhibition is achieved, the network is almost capable of rapid hypothesis testing. The inhibition "renormalizes" or "conditionalizes" the field $\mathcal{F}^{(2)}$ to respond differently to pattern $x_2^{(1)}$ during the next time interval. If the next pattern elicited by $x_2^{(1)}$ across $\mathcal{F}^{(2)}$ also creates a mismatch at $\mathcal{F}^{(1)}$, then it will be suppressed and $\mathcal{F}^{(2)}$ will be normalized again. In this fashion, a sequence of rapid pattern reverberations between $\mathcal{F}^{(1)}$ and $\mathcal{F}^{(2)}$ can successively conditionalize $\mathcal{F}^{(2)}$ until either a match occurs or a set of uncommitted cells is activated with which $x_2^{(1)}$ can build a learned filter from $\mathcal{F}^{(1)}$ to $\mathcal{F}^{(2)}$ and a learned expectancy from $\mathcal{F}^{(2)}$ to $\mathcal{F}^{(1)}$.

The third property will be discussed separately, since it is one of the surprising, but basic, mathematical consequences of a mechanism that I have already mentioned.

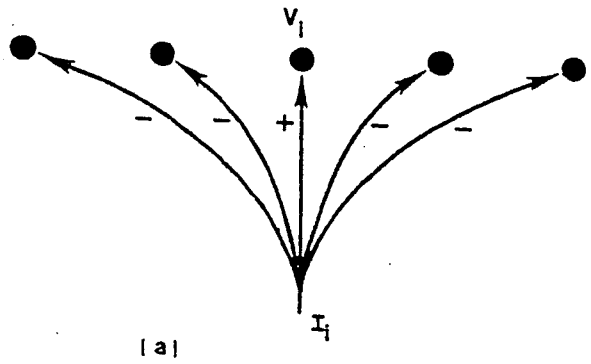
18. CONTRAST ENHANCEMENT AND STM NORMALIZATION IN COMPETITIVE FEEDBACK NETWORKS

The third property is needed to prevent the system from inhibiting too much and thereby terminating the error correction process.

(3) Contrast enhancement and normalization of STM activity: If $x_1^{(2)}$ is the pattern that $x_2^{(1)}$ originally excites and $x_1^{(2)}$ is inhibited by the arousal burst, then how does $x_2^{(1)}$ activate any pattern whatsoever across $\mathcal{F}^{(2)}$ during the next time interval?

Otherwise expressed, if $x_2^{(1)}$ can elicit a pattern in the next time interval, why couldn't it elicit this pattern during the first time interval, when the pattern would be inhibited by the arousal burst. How can we escape from this deadly circular argument? How do we do so in a principled fashion?

We already know that the noise-saturation dilemma confronts all pattern-processing cellular tissues. Hence, we expect $\mathcal{F}^{(2)}$ to be a mass action competitive network. If the competitive interactions were distributed in a feedforward fashion, with input pathways sending off inhibitory input branches from their excitatory on-centers, then the problem just posed could not be surmounted. However, if the competitive interactions are distributed in a feedback fashion, such that the cells themselves send off positive and negative feedback signals to other cells (FIGURE 10), then the required properties occur as mathematical consequences of this design. These facts were proved in Grossberg (1973) and are reviewed in Grossberg (1980a, 1981a). The positive feedback pathways endow $\mathcal{F}^{(2)}$ with a capability for storing patterns in STM. I conclude that a network that can solve the noise-saturation dilemma and that is capable of STM storage can overcome a serious obstacle to cognitive hypothesis testing.



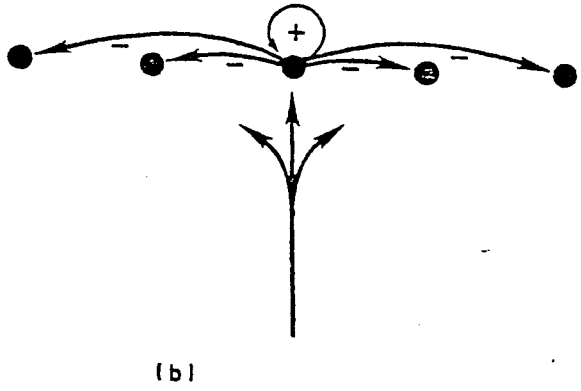


FIGURE 10. (a) A feedforward competitive network. (b) A feedback competitive network.

The two crucial properties of this mechanism that we need are contrast enhancement of input patterns and normalization (conservation, adaptation) of the total suprathreshold STM activity across the network. A network that fails to possess these properties is a bad design from the viewpoint of hypothesis testing.

To see how these properties overcome our difficulty, consider FIGURE 11. FIGURE

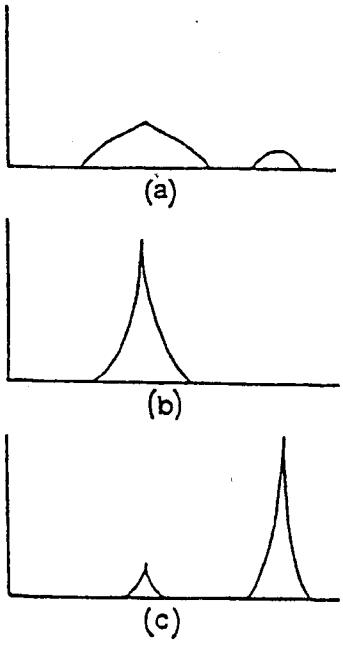


FIGURE 11. The input pattern in (a) elicits activity pattern $x_i^{(2)}$ in (b) by suppressing small inputs and contrast enhancing large inputs. After $x_i^{(2)}$ is suppressed, the small input activities inherit normalized activity from the suppressed populations to elicit the distinct activity pattern in (c).

11a depicts the input pattern that $x_2^{(1)}$ generates at $\mathcal{F}^{(2)}$ due to filtering by the $\mathcal{F}^{(1)} \rightarrow \mathcal{F}^{(2)}$ pathway. This input pattern is rapidly contrast enhanced before generating the activity pattern $x_1^{(2)}$ across $\mathcal{F}^{(2)}$. (The choice of maximal input leading to categorical perception, which was discussed in Section 3, is an extreme case of contrast enhancement.) The contrast enhancement process prevents small inputs from being represented in STM by $x_1^{(2)}$ (FIGURE 11b).

After $x_1^{(2)}$ is inhibited, that part of the input pattern which activated the inhibited cells is no longer effective. Only the cells that receive small inputs are free to be excited. In a feedforward competitive network, these small inputs would elicit small activities that might be insufficiently large to drive subsequent network events. In a feedback competitive network, by contrast, the total suprathreshold STM activity tends to be conserved. Thus, the uninhibited cells inherit the STM activity that used to energize the inhibited cells. The small inputs generate a large pattern in STM (FIGURE 11c) and, moreover, a pattern that is distinct from the inhibited pattern $x_1^{(2)}$.

19. LIMITED CAPACITY STM SYSTEM: AUTOMATIC VS. CONTROLLED PROCESSING

Many remarks can (and have!) been made about this STM mechanism. Its properties are reflected in a vast body of data, but many scientists seem not to realize this because they are as yet unaware of how much is known about competitive systems. Perhaps the most obvious conclusion is that the STM normalization property dynamically explains why STM is a limited capacity system, a fact postulated in essentially all cognitive models that distinguish STM from LTM. It is not usually realized, however, that special versions of this same property can be used to explain aspects of behavioral contrast during discrimination learning (Grossberg, 1975, 1981b), of free recall and reaction time data without assuming that a serial buffer exists (Grossberg, 1978b), of LTM encoding by sequence-sensitive cognitive or motor chunks that are capable of rapidly competing in STM to select the most informative and best predictive chunks in a given temporal context (Grossberg, 1978a), and so on.

These STM concepts also show how to escape the serious internal paradoxes of information processing theories, like that of Schneider and Shiffrin (1976), which arise from associating a serial process with the serial properties of controlled search and a parallel process with the parallel properties of automatic search. The untenability of this assumption is suggested when we consider the learning of any new list of familiar items. Each familiar item is assumed to be processed by a parallel process, while each unfamiliar interitem contingency is processed by a serial process. Does anyone seriously believe that the brain rapidly alternates between parallel and serial processing in this situation? Moreover, how does the brain know how to switch from a hybrid of serial and parallel processing to exclusively parallel processing as the whole list becomes unitized? When we view a picture whose left half contains a familiar face and whose right half contains a collection of unfamiliar features, how does the visual field split itself into a parallel half and a serial half? How does the field get reintegrated as a parallel processor as the unfamiliar features are unitized? The conceptually paradoxical nature of this hypothesis is also reflected in unexplained data. Why is it so that the "time for automatic search is at least as long as that for a very easy controlled search" (Schneider and Shiffrin, 1976)? Do not these data violate our intuitive understanding of the concept "automatic"? No more so than the Schneider and Shiffrin hypothesis, which shows that the accepted intuitive understanding of the concepts "serial" and "parallel" is wanting.

I have elsewhere explained such data without assuming that serial properties imply a serial process, indeed by explicating what kind of parallel process can generate both automatic and controlled properties in different experimental paradigms given different degrees of learning (Grossberg, 1978a, Section 61). I mention this fact here because one might be tempted to say off-hand that the hypothesis testing scheme described herein is a serial process. This is false! Its operations unfold sequentially in time, but its mechanisms are parallel. This distinction cannot be overemphasized. In my papers cited above, such distinctions lead to different predictions of the two types of theory as well as to philosophically less paradoxical concepts.

Excellent theorists are spawning these paradoxes because they are confronting mechanisms that cannot easily be inferred using a black-box approach. As another example, STM normalization is an operation akin to summing all the probabilities of possible events to 1 and multiplicative shunting laws are suggestive of multiplying the probabilities of statistically independent events to compute their joint probability. The hypothesis testing by rapid parallel STM reset and renormalization might then, to a black-box theorist, be analogized to a serial estimation procedure whereby conditional expectations are updated based on prior information. This analogy leads to an incorrect understanding of how the mechanism works and of what it is doing with the hypotheses.

I hope that ERP workers will not passively accept paradoxical information processing theories. Rather, I hope that ERP methods will be used to help test these theories. Such an approach can both enhance the importance of ERP methods and dampen the depressing dogma that any instantiation of an information processing concept is as good as any other.

20. SIGMOID SIGNAL FUNCTIONS AND NOISE SUPPRESSION

I will mention one more seemingly innocuous property of feedback competitive networks before continuing my story. This property will be seen to imply unsuspected inverted U and overshadowing effects in Part II of this article.

The positive feedback signaling in competitive feedback networks can be a mixed blessing if it is not properly designed. Positive feedback can subserve such desirable properties as STM storage, contrast enhancement, and normalization. It can also, if improperly designed, flood the network with noise generated by its own activity. This noise amplification property will have disastrous effects on network processing whenever it occurs, and it can occur in formal network "seizures" and "hallucinations" (Ellias and Grossberg, 1975; Ermentrout and Cowan, 1979; Grossberg, 1973; Kaczmarek and Babloyantz, 1977; Schwartz, 1980).

A proper choice of signal function prevents noise amplification (Grossberg, 1973); namely, a sigmoid, or S-shaped, signal function. The use of a sigmoid signal function implies the existence of a QT in a competitive feedback network. The QT property, in turn, controls a feedback network's ability to be tuned and to achieve contrast enhancement. The sigmoid signal inhibits noise as another manifestation of the QT property. Freeman (1979) provides data and a careful model of sigmoid signaling in the olfactory system.

21. P_{300} AND STM RESET: UPDATING OF THE SCHEMA

Only the third property that is needed for rapid hypothesis testing, namely STM normalization, has thus far been given a mechanistic interpretation. The two properties

of selective and enduring STM reset will next be mechanistically explicated. Before doing this, let us pause to review our main conclusion about error correction in intuitive terms.

Unexpected events can trigger a burst of nonspecific arousal (N_{200}) that resets STM by rapidly inhibiting active representations and allowing less active representations to achieve STM storage by inheriting normalized STM activity. In somewhat less mechanistic terms, the formal N_{200} can cause "updating of the schema" within the network. I use this phrase from the Orienting and P_{300} chapter (Donchin *et al.*, this volume) because the process of STM reset is hypothesized to elicit a P_{300} in my theory.

The Donchin *et al.* suggestion and my own conception will be seen to diverge in several basic ways. For one, I will commit myself to an explicit physiological and pharmacological mechanism subserving the P_{300}. I will also show that this mechanism suggests several operations other than mismatch detection that can trigger a formal P_{300}, and that go beyond alternative theories. Finally, I will show that there are other ways to reset STM (*viz.*, "update the schema") than to trigger a P_{300}. The P_{300} mechanism that I will describe was first used in both a motivational and cognitive context in Grossberg (1972b).

22. GATED DIPOLES: ANTAGONISTIC REBOUND DUE TO SLOW TRANSMITTER GATING IN COMPETING CHANNELS

The next design problem is quite subtle: How can a nonspecific event, such as arousal, have specific consequences of any kind, let alone generate an exquisitely graded, enduring, and selective inhibition of active cells? How can a one-dimensional command selectively reorganize an information processing scheme of very high dimension? My solution will make an essential use of chemical transmitters and competition. Arousal, transmitters, and competition are all ubiquitous in the nervous system. The network *interactions* between these ingredients will be shown to literally create information processing properties. These interactions are invisible to microelectrodes and chemical assays, and are only dimly reflected in most behavioral experiments. Mathematics is the tool that most easily probes this interactive level at the present time.

The design problem can be restated in a suggestive way: What cells selectively shut off the active cells in $\mathcal{F}^{(2)}$ and keep them off while $x_2^{(1)}$ is being recoded by a renormalized STM pattern across $\mathcal{F}^{(2)}$? This reinterpretation faces the fact that a *deus ex machina* cannot be invoked to carry out the reset operation. There are only cells and more cells to do so. We therefore conclude that, associated with each of the cells (or cell populations) that need to be turned off, there exist other cells, specifically related to them, whose activation can maintain selective inhibition. Let us call the cells that are turned on by $\mathcal{F}^{(1)} \rightarrow \mathcal{F}^{(2)}$ signals on-cells, and the cells that selectively inhibit them off-cells.

Having come this far, we can now recognize that these (on-cell, off-cell) pairs, or dipoles, need to exist in the nervous system for an even more primitive reason. What I will show is that the mechanism that achieves the more primitive demand automatically has the property of dipole reset. Because there exists more than one way to cause a dipole reset, there is also more than one way to cause a P_{300} in my theory.

The more primitive property answers the question: How does offset of an event act as a cue for a learned response? For example, suppose that I wish to press a lever in response to the offset of a light. If light offset simply turned off the cells that code for

light being on, then there would exist no cells whose activity could selectively elicit the lever press response after the light was turned off. Offset of the light cannot only turn off the cells that are turned on by the light. Light offset must also selectively turn on cells that will transiently be active after the light is shut off. The activity of these off-cells (the cells that are turned on by light offset) can then activate the motor commands leading to the lever press. Let us call the transient activation of the off-cell by cue offset antagonistic rebound (FIGURE 12).

In a reinforcement context, I claim that such an antagonistic rebound is the basis for a relief reaction (Denny, 1971) upon offset of a sustained fear-eliciting cue (Estes and Skinner, 1941). In a perceptual context, I claim that such an antagonistic rebound is the basis for a negative aftereffect upon offset of a sustained image (Brown, 1965, p. 483; Helmholtz, 1866, 1962).

I will now describe a minimal model capable of eliciting a sustained on-response to onset of a cue and a transient antagonistic rebound to offset of the cue. The intuitive postulates that led to the model's original derivation are given in Grossberg (1972b). An alternative derivation is given in Appendix 1. Appendix 1 also derives a variety of

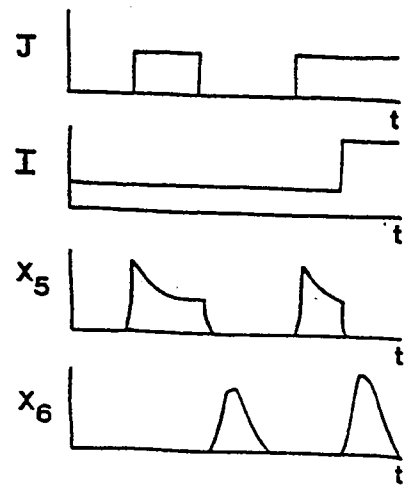


FIGURE 12. An on-response, x_5, occurs to rapid onset of a specific on input, J. Either rapid offset of J or onset of nonspecific arousal I causes an off-reaction x_6, or antagonistic rebound. The notation is explained in Section 22 and FIGURE 13.

gated dipole properties that are helpful in understanding aspects of normal and abnormal motivated behavior and ERPs.

Consider FIGURE 13. In FIGURE 13a, a nonspecific arousal input, I, is delivered equally to both the on-channel and the off-channel, whereas a test input, J, (*e.g.*, light or shock) is delivered only to the on-cell channel. These inputs activate the potentials x_1 and x_2, which create signals S_1 and S_2 in the on-channel and off-channel, respectively. Since $I + J > I$, $S_1 > S_2$. What happens next is crucial. Appendix 1 proves the following assertions rigorously.

The square synapses in FIGURE 13 contain chemical transmitters z_1 and z_2. Each transmitter slowly accumulates to a target level. The slow accumulation rate is essential to the model's properties. The target level is achieved by a constant transmitter production rate that is reduced by feedback inhibition proportional to the transmitter concentration. When a signal S_1 reaches the synaptic knobs containing z_1, transmitter is released at a rate proportional to $T_1 = S_1 z_1$. The multiplicative effect of z_1 on S_1 to yield T_1 is called transmitter gating of the signal S_1. The gating law just says that S_1 and z_1 interact *via* mass action to elicit T_1. In particular, if either $S_1 = 0$ or $z_1 = 0$, then $T_1 = 0$.

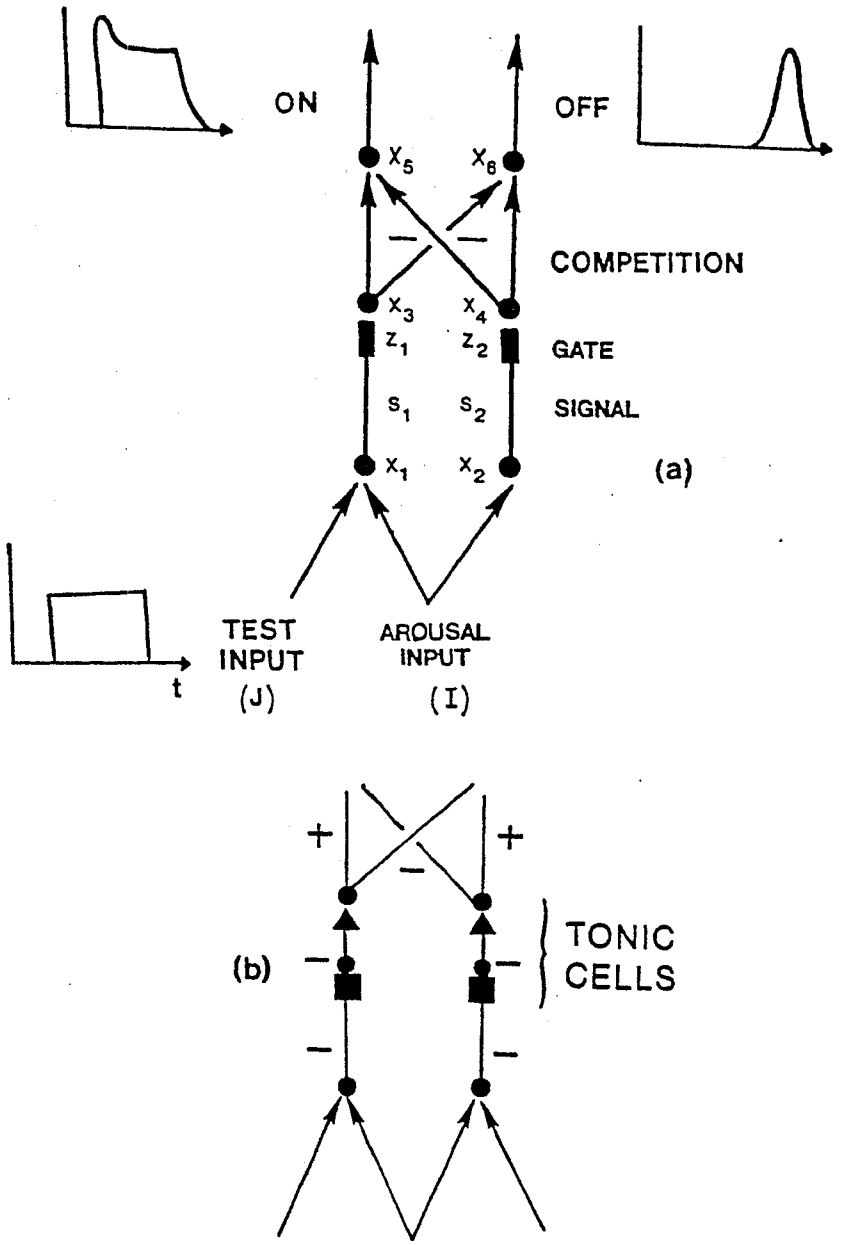


FIGURE 13. Two examples of gated dipoles. In (a), the phasic input, J, and the arousal input, I, add in the on-channel. The arousal input also perturbs the off-channel. Each input is gated by a slowly varying excitatory transmitter (square synapses). Then the channels compete before eliciting a net on-response or off-response. In (b), the slowly varying transmitters are inhibitory and the net effect of two successive inhibitory transmitters (*e.g.*, DA and GABA) is a net disinhibitory effect.

One proves that, if $S_1 > S_2$, then $T_1 > T_2$. That is, transmitter is released at a faster rate by larger signals. Consequently, potential x_3 exceeds potential x_4. These potentials then emit competing signals. Potential x_5 wins the competition over x_6 and emits output signals that are the on-reaction of the network.

So far everything seems quite elementary. Only now do we exploit the slow accumulation rate and the transmitter gating law to show how a transient antagonistic rebound is generated by rapid offset of J.

Because both transmitter stores had accumulated almost equal amounts of transmitter before J turned on, the faster transmitter depletion rate in the on-channel

than the off-channel when J is on implies that $z_1 < z_2$, despite the fact that $S_1 z_1 > S_2 z_2$. When J is shut off, both channels receive the equal arousal input I. The potentials x_1 and x_2 rapidly equalize, as do the signals S_1 and S_2. By contrast, the inequality $z_1 < z_2$ persists because transmitter accumulates slowly! Thus, right after J shuts off, $S_1 z_1 < S_2 z_2$, $x_3 < x_4$, and the off-channel wins the signal competition. An antagonistic rebound is thereby initiated.

The rebound is transient because the transmitters gradually respond to the equal signals I by reaching a common level $z_1 = z_2$. Then $S_1 z_1 = S_2 z_2$ and the competition shuts off the rebound.

There exist many variations on the gated dipole theme. FIGURE 13b points out that the slow transmitters can be inhibitory stages of a disinhibitory pathway. I interpret dopamine and noradrenaline to be the slow inhibitory transmitters in motivational and/or cognitive dipoles. The disinhibitory concept rationalizes many effects of drugs such as amphetamine, chlorpromazine, 6-OHDA, and MAO inhibitors on behavior. The Appendix shows that a single cell, rather than an intercellular network, as in FIGURE 13, can act like a gated dipole. Such a dipole exists, I contend, in vertebrate photoreceptors (Carpenter and Grossberg, 1981). A full understanding of the gated dipole concept requires that we be able to distinguish the varied anatomical substrates of gated dipoles from their commonly shared functional properties.

23. TOLERANCE IMPLIES WITHDRAWAL: REBOUND INSOMNIA AND A WAY OUT

The above account depends upon the property that the phasic input, J, gradually depletes z_1 more than z_2 until z_1 equilibrates to the total tonic plus phasic input level, $I + J$, rather than the tonic input level, I. This gradual adaptation is a type of transmitter habituation. It is the type of habituation due to progressive depletion of a slowly varying chemical gate, not the type of habituation that occurs when nonspecific gain control gradually raises the QT of a network and thereby decreases its sensitivity.

Within the gated dipole context, habituation is a prerequisite for antagonistic rebound in response to input offset. If the net production rate could be increased fast enough to offset the increase in depletion due to the gating action, then no habituation and no rebound would occur.

This type of habituation-rebound interaction goes by different names in different experimental contexts. I believe that an important class of examples, whose biochemical substrates seem to be as yet unclear, occurs in situations where the sustained action of a drug causes tolerance to the drug. If the drug acts like a signal whose effect is modulated by a depletable chemical gate, then tolerance can be interpreted as habituation of the gate. More drug S is needed to generate the same net effect $T = Sz$ as z decreases due to habituation. Rapid removal of the drug, or signal, S before the gate z can re-equilibrate to its resting level will cause an antagonistic rebound, or withdrawal reaction. If this hypothesis is correct, one way to prevent withdrawal is to prescribe the therapeutic drug with parallel doses of another drug that speeds up the production rate of the gating chemical to offset its more rapid rate of depletion.

Richard Solomon (personal communication) has collected data on imprinting that exhibit temporal properties analogous to those of drug addiction. In my theory, the formal bridge between these two types of phenomena is the gated dipole concept. Another important phenomenon that should be further analyzed from this perspective is rebound insomnia (Ostwald, 1971), which exhibits both habituation and rebound.

This example is particularly desirable from a theoretical perspective because Carpenter and I have suggested that suitably connected gated dipoles can generate oscillations whose formal properties resemble circadian rhythms (Carpenter and Grossberg, 1983a, 1983b).

24. A NONSPECIFIC AROUSAL BURST TRIGGERS AN ANTAGONISTIC REBOUND: MISMATCH TRIGGERS A P300

The main property that is needed to understand how an N200 can trigger a P300 (FIGURE 12) is not intuitively obvious, although it can be proved using simple algebra (Appendix). We need to understand how a nonspecific event, such as a mismatch-contingent arousal burst, can selectively suppress the active on-cells, yet spare the inactive on-cells, across a field of gated dipoles. The appendix proves that the following remarkable property holds in the gated dipole of FIGURE 13a if the signals S_1 and S_2 are linear functions of their respective inputs.

The off-rebound size in response to a sustained input J and a sudden arousal increment of size ΔI is

$$\text{OFF} = \frac{ABJ(\Delta I - A)}{(A + I + J)(A + I)}, \tag{3}$$

where A and B are positive constants. Note that a positive off-reaction occurs only if $\Delta I > A$. This criterion is independent of J, which means that an arousal increment ΔI that is sufficiently large to rebound any dipole will be large enough to rebound all dipoles in a field. In other words, if the mismatch is "wrong" enough to trigger a large arousal increment, then all erroneously active cells will simultaneously be rebounded. By contrast, the size of the rebound is an increasing function of the on-input J and equals 0 if $J = 0$. Rebound size is, thus, selective, despite the fact that all active dipoles can be rebounded at once.

Some readers might at this point complain: These formal properties are quite delightful to be sure, but you have proved them using a linear signal function. We thought that a sigmoid signal function is needed to avoid noise suppression in a competitive feedback network; these gated dipoles occur at $\mathcal{F}^{(2)}$, which is such a network. Do these properties also obtain when a sigmoid signal function is used?

This is the type of question that only a confrontation between two physical principles can engender. The answer will be given shortly in terms of inverted U effects, analgesic effects, overshadowing effects, and a host of other physical insights that dropped out of the mathematical skies when I asked the same question. Before addressing this matter, let us face some of the implications of the results that we already have before us.

25. AN AROUSAL TEST OF THE GATING TRANSMITTER

The fact that a nonspecific arousal burst can selectively reset a gated dipole suggests one way to test which transmitter(s) are used in the dipole (FIGURE 14). For example, suppose that one has behavioral control of the phasic on-input (J) (*e.g.*, a conditioned reinforcer such as food, or a localized electrode signal). Suppose that one has also localized a putative off-channel (*e.g.*, a satiety channel) and can micropipette various transmitter antagonists into this location. Finally, suppose that an arousal

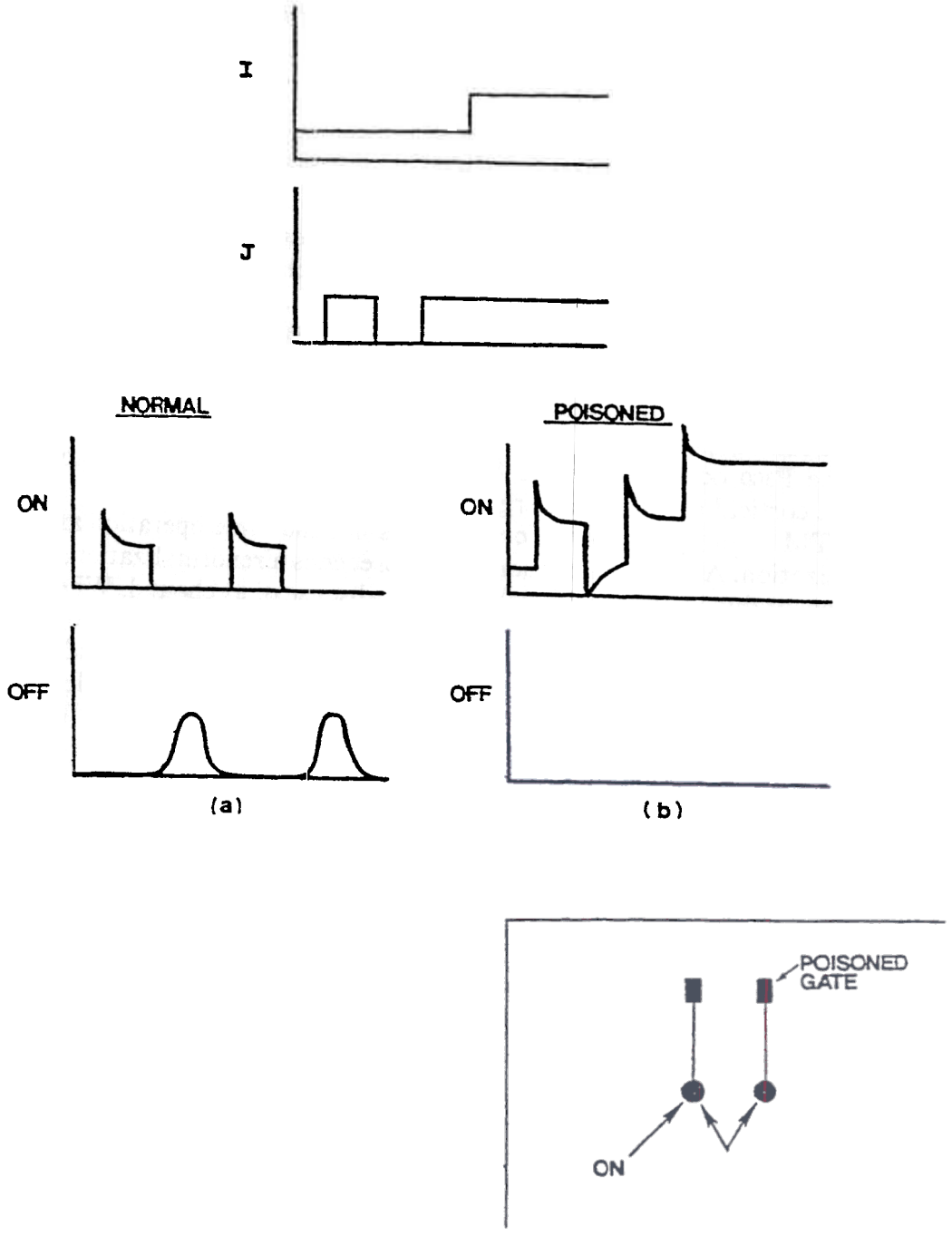


FIGURE 14. Testing whether a network is a gated dipole and what its off-channel transmitter is. The effect of an arousal burst is depicted (a) before and (b) after the off-channel transmitter is poisoned. Note that an off-rebound occurs in response to phasic cue offset in the on-channel of the poisoned dipole. A sustained on-response follows an arousal increment in a poisoned dipole, instead of a transient off-response.

source (I) modulates this system (*e.g.*, electrical stimulation of the reticular formation). FIGURE 14a depicts the responses one should find to a sustained phasic on-input when an arousal burst occurs. FIGURE 14b depicts the responses one should find to the same inputs if one has successfully poisoned the transmitter in the off-channel.

At the present time, this experiment serves more as a thought experiment to sharpen conceptual understanding than an experiment one would run breathlessly to the lab to do. The experiment does suggest, however, that being able to correlate

on-reactions and off-reactions at nearby electrode positions is a serious goal for those who wish to study behavioral effects of transmitter dynamics, say by using a microelectrode with multiple recording sites (Kuperstein and Whittington, 1981).

26. P300, CATECHOLAMINE REBOUND, CNV REBOUND, CRITICAL PERIOD TERMINATION

In all the physical examples where gated dipoles have heretofore been used, the transmitter gate has either been identified as a catecholaminergic transmitter, such as DA or NA (Grossberg, 1972b, 1975, 1976b, 1980a), or as an ion acting like a second messenger, such as Ca^{++} (Carpenter and Grossberg, 1981; Grossberg, 1968). To discuss the cortical P300, I suggest that the transmitter is a catecholamine, probably NA (Pettigrew and Kasamatsu, 1978). This interpretation leads us to several important conclusions.

(1) the P300 occurs whenever STM is reset by a massive antagonistic rebound within the cortical catecholaminergic arousal system.

(2) STM reset by antagonistic rebound is not the same operation as STM reset by renormalization. Although a rebound often precedes a renormalization, a renormalization can occur due to an attention shift in the absense of a rebound. Whereas a rebound is a transient effect due to the action of a slow transmitter gate, a renormalization is a sustained effect of a limited capacity STM field that does not depend on transmitter gating. In this regard, Tecce (1979) has elegantly studied a possible CNV rebound effect, but this effect may really be due to renormalization. A test of time scale is needed to discriminate the two possibilities.

(3) The distinction between rebound and renormalization shows that Donchin's phrase "updating of the schema" is insufficient to capture the role of P300. The fact that an antagonistic rebound can be due either to offset of a phasic cue or to onset of an arousal burst suggests that no single phrase about P300 can capture its processing implications.

The distinction between phasic cue offset and arousal burst onset is, I believe, important for understanding how a cortical P300 might precede a hippocampal P300 if a surprising event rebounds cortical STM while positive conditioned reinforcers are being attended. I suggest that the cortical rebound will register a cortical P300. Inhibiting the conditioned reinforcer withdraws phasic reinforcing input from the hippocampus, whose dipole structure responds with a hippocampal P300. Part II discusses these issues in greater detail.

(4) The above mechanisms indicate how environmentally sensitive critical periods might be terminated and dynamically maintained by the action of learned feedback expectancies. These expectancies modulate an arousal mechanism that buffers already coded populations by shutting them off so rapidly in response to erroneous STM coding that LTM recoding is impossible. In other words, the mechanism helps to stabilize the LTM code against continual erosion by environmental fluctuations as it drives the search for new codes.

If this conclusion is correct, then all the machinery we have postulated should develop before the end of the critical period that it is controlling. These mechanisms include: (a) gated cortical dipoles (P300), (b) catecholaminergic arousal (N200), (c) lateral inhibition across $\mathcal{F}^{(1)}$ and $\mathcal{F}^{(2)}$, and (d) corticothalamic or corticocortical conditionable feedback pathways (cholinergic?).

Two cautionary comments should be made. First, the timing and even the form of the ERPs in (a) and (b) in the infant can differ from those in the adult, as discussed by

Kurtzberg *et al.* and Otto *et al.* (this volume), without denying their role in critical period termination. Second, I wish to carefully emphasize the words *environmentally sensitive* critical periods. Prewired developmental unfolding is not included in this discussion, although all the mechanisms would work in a prewired setting if endogeneously driven input patterns replaced exogenously driven input patterns, and if the QTs of the system were kept low enough to allow pattern processing to unfold.

27. A P300 TEST OF THE CRITICAL PERIOD BUFFER MECHANISM

A variant of the oddball paradigm can be used to test whether the buffering mechanism I have suggested helps to terminate the visual critical period. A pair of visual stimuli should be constructed from complementary feature configurations, such as a white bar (bars) on a black field and a black bar (bars) on a white field. These stimuli should be presented sequentially in such a way that the complementary features excite the same retinal positions.

Two experimental groups would be needed: a group that receives the two stimuli in a regular alternating sequence and a group that receives each stimulus 50% of the time in a random order. Although both groups receive the stimuli the same number of times, the random group should experience larger P300s and a faster rate of feature encoding.

These advantages would be explained by the antagonistic rebounds that occur when a stimulus different from the one expected occurs. If complementary cortical feature detectors are organized into dipole pairs, the rebound due to the nonoccurrence of the expected stimulus should add on to the direct effect of the actual stimulus, since it is complementary to the expected stimulus, thereby enhancing the cortical reaction to the actual stimulus and driving faster LTM encoding in its adaptive filter.

If this prediction turns out to be true, it will support my contention that functional homologues exist between cortical and motivational rebound mechanisms, since a similar mechanism can be used to explain the partial reinforcement acquisition effect (Grossberg, 1975).

28. ADAPTIVE RESONANCE: A SOLUTION TO THE STABILITY-PLASTICITY DILEMMA

My previous discussions have worried about how to protect already encoded populations from adventitious recoding by erroneous events. Now I will summarize what happens when a correct encoding occurs. A pattern across $\mathcal{F}^{(1)}$ elicits a representation across $\mathcal{F}^{(2)}$ that reads out a template that matches the pattern. As I mentioned in Section 14, a pattern match amplifies the reaction that occurs to the pattern alone. Due to pattern matching, the interfield signals $\mathcal{F}^{(1)} \rightarrow \mathcal{F}^{(2)}$ and $\mathcal{F}^{(2)} \rightarrow \mathcal{F}^{(1)}$ mutually reinforce each other and activities at both levels are amplified and locked into STM. In short, an STM resonance is established. Because the STM activities persist much longer in time during the resonant state than the passive decay rates of individual cells would allow, or than the durations between rapid reset operations in the mismatch situation would allow, the slowly varying LTM traces in the adaptive filter and expectancy pathways now have sufficient time to sample the STM patterns and to store them in LTM. I therefore call this dynamic state an adaptive resonance (Grossberg, 1976b, 1978a, 1980a).

I should emphasize a subtle point about the adaptive resonance idea. Before

resonance occurs, the LTM traces in the filtering and expectancy pathways do a perfectly good job of controlling the readout of activities across $\mathcal{F}^{(2)}$ and $\mathcal{F}^{(1)}$, respectively. However, the LTM traces cannot change in response to these STM activities unless the STM activities resonate long enough for the slowly varying LTM traces to sample them. The LTM traces are adaptively blind to the STM activities that they direct until resonance occurs. My solution to the stability-plasticity dilemma freely admits that the LTM traces are potential victims of all the local signals that they process, but saves the LTM traces from chaotic recoding by ensuring that only resonant signals can drive significant LTM changes. The resonant signals, in turn, can only occur when the system as a whole determines that the local signals are worthy of LTM encoding.

The resonant state provides a context-sensitive interpretation of the data that explicates in neural terms the idea that the network is paying attention to the data. The fact that LTM encoding is driven by STM resonance provides a mechanistic explanation of the psychological fact that a relationship exists between paying attention to an event and coding it in LTM (Craik and Lockhart, 1972; Craik and Tulving, 1975).

Freeman (1975, 1980) has provided the most beautiful data I know of that measure a perceptually driven resonance on the physiological level. It might be instructive to perform ERP experiments in parallel with Freeman's physiological experiments to determine which ERPs reflect the resonant state. Presumably, some type of slow negative wave will be measured.

At this point, I can summarize simply and precisely what I mean by the "code" of a network. The code is the set of dynamically stable resonances that the network can support in response to a prescribed input environment. I believe that a major task of cognitive psychology is to classify how prescribed initial choices of the filtering, competition, expectancy, and dipole parameters bias the network to learn different invariants of the data, or categorical boundaries, in response to prescribed sequences of input patterns. This assertion celebrates James Gibson's wonderful experimental intuition, which led him, despite an almost total ignorance of processing substrates, to realize that the perceptual system "resonates to the invariant structure or is attuned to it" (Gibson, 1979, p. 249).

The remaining sections will apply the ideas that are physically or mathematically latent in the previous paragraphs towards the explanation and prediction of interdisciplinary data. Various of these data have not been explained before to the best of my knowledge.

PART II

29. THE DIPOLE FIELD

Part II of this article will use the foundation built up in Part I to derive networks that model cognitive and motivational interactions. An understanding of such interactions is indispensable to the student of abnormal behavior, since a breakdown within either type of network can upset the interactive balance within the system as a whole. Such an understanding is also useful to persons who are interested in normal cognitive or attentional behavior to clarify how changes in motivational parameters (*e.g.*, hunger, thirst, fear, relief) can alter cognitive processing, how cognitive processes can feed back upon themselves *via* pathways that run through motivational mechanisms, and how data and paradigms about motivational phenomena can suggest homologous studies of cognitive phenomena.

A paradigm wherein motivational influences on cognitive processing occur has, for example, recently been developed by Bower and his colleagues (Bower, 1981; Bower *et al.*, 1981). These investigators have studied how subjects' moods during learning can influence their recall when the same or incongruent moods are active during recall. Their results are compatible with my hypothesis (Grossberg, 1972a,b, 1975), reviewed below, that incentive motivational feedback from positive or negative drive representations to cognitive representations is positive, nonspecific, conditionable, and modulates LTM encoding and readout of the cognitive representations. To test whether this mood effect on memory is due to the type of hippocampal-to-cortical feedback that I envisage, a CNV measure might be added to the Bower *et al.* paradigm. If so, learned associations between mood and memory should elicit larger CNVs during recall of mood-congruent lists than during other manipulations.

An example wherein cognitive processing may interact with motivational processing is suggested by the discovery of a hippocampal P300 by Halgren *et al.* (1980). If cortical P300s are ever going to be functionally disentangled from hippocampal P300s, a paradigm is needed wherein the two types of P300s can be differentially manipulated by the experimenter. I suggest the use of a conditioning-attentional paradigm below. To understand my prediction in this paradigm, one needs to study the motivational networks on their own terms, notably how a cortical P300 may elicit a hippocampal P300 and how changes in conditioned reinforcer or drive input strength may alter the size of the hippocampal P300. A hippocampal P300 was predicted in Grossberg (1980a).

Studies of abnormal syndromes, such as juvenile hyperactivity or simple schizophrenia, need to distinguish whether motivational, cognitive, or both types of processes are faulty. For example, I will compare hyperactive behavior with predictions of a formal syndrome that occurs whenever a gated dipole is underaroused. The same formal syndrome occurs whether the gated dipole occurs in a motivational or a cognitive network, but its behavioral manifestations will be different in the two cases. Whatever the interpretation, an underaroused gated dipole exhibits an elevated response threshold, a hypersensitive reaction to a suprathreshold increment in its on-input, and a hyposensitive antagonistic rebound to a phasic decrement that halves its on-input. In a motivational context, these properties might yield hypersensitivity to increments in cues like ice cream or shock, but hyposensitive antagonistic reactions to a partial withdrawal of these cues. In a cognitive context, by contrast, one might expect elevated sensory thresholds and weaker negative aftereffects to decrements in color or orientation cues (Grossberg, 1980a).

The ubiquitous occurrence of gated dipoles also suggests tests of habituation and dishabituation in both motivational and cognitive processors. Due to the slow habituation rate of a gated dipole's transmitters, slow onset of a shock can be less negatively reinforcing than rapid onset to the same shock asymptote. For the same formal reason, slow onset of a sensory cue may lead to weaker initial STM storage of the cue and thus to a smaller P300 when STM is reset. I suggest also that the slow reaction rate of a gating chemical may explain why slow shutting off of a shock may be less positively reinforcing, or why slow removal of an addicting drug can lessen withdrawal symptoms, which I view as a type of antagonistic rebound phenomenon. On the cognitive level, slow removal of a phasic cue may cause a smaller P300 for the same mechanistic reason.

To complement these habituation-related effects of slow chemical gates, I also suggest that certain dishabituation phenomena may occur in motivational and cognitive processes due to gated dipole mediation, that these phenomena should vary in characteristic ways with the arousal level, and that some of these variations may be testable using ERPs. For example, I suggest that, when environmental contingencies unexpectedly change, STM reset in response to the novel event has properties that

endow both previously overshadowed cues and the novel cues with an advantage in STM storage that may be testable using a P300 measure. The enhancement of the STM activity at previously overshadowed representations is interpreted as a dishabituation reaction in my theory. The theory indicates how behavioral manipulations of unexpected events may elicit physiological dishabituation reactions caused by pharmacologically identified gating reactions that are related to psychophysiological P300 measures. If the network's arousal level is lowered into the underaroused range, the same STM reset mechanism can enhance, or dishabituate, STM activities that would ordinarily be reset, or rebounded. This property can both reduce the P300 and cause attentional reset problems. I also suggest that, when this underaroused dishabituation reaction occurs in suitable motivational or motor command networks, it can cause a paradoxical enhancement of a reinforcer's potency or a motor reaction akin to Parkinsonian bracing and should influence the ERPs that subserve these events in a manner homologous to the influences on P300 in cognitive examples. Due to the existence of such pervasive interrelationships between cognitive and motivational

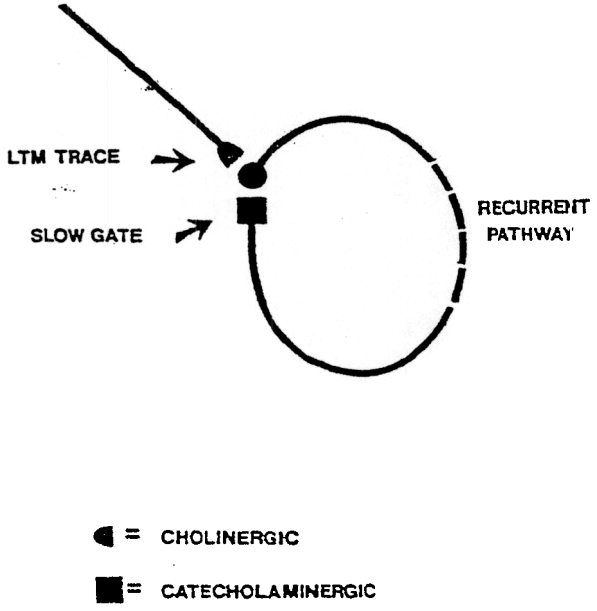


FIGURE 15. A conditionable cue pathway feeds into a gated feedback loop. The LTM trace in the conditionable pathway is assumed to be cholinergic, whereas the gate is assumed to be catecholaminergic.

phenomena, I will move freely between both types of examples below to present the theory in the way that I feel most efficiently presents the formal ideas and their connections. The reader who is primarily interested in just one type of example is invited to skip sections of lesser interest on a first reading.

One of the most important implications of the thought experiments of Part I has not yet been systematically explored. How can we marry the gated dipole structure to the mass action competitive feedback structure? Consider, in particular, those structures wherein a nonspecific arousal burst can rebound activities that are reverberating in STM after the cues that initiated STM storage have terminated. In order for this to happen, the STM feedback loops must contain the transmitter gates, so that STM activity can differentially deplete the active STM loops and thereby prepare them for rebound. FIGURE 15 summarizes this structural arrangement by depicting a conditionable input pathway abutting a gated feedback pathway. I will henceforth assume that the transmitter in a conditionable input pathway is cholinergic and that the transmitter in a gated STM feedback loop is catecholaminergic (Grossberg,

1972b), since a large body of data is compatible with this suggestion. Some of these data will be reviewed below.

There exists another way to derive FIGURE 15, even when no arousal-initiated rebound exists. This alternative derivation holds when the off cells represent features or behavioral categories that are complementary to those represented by the on-cells; *e.g.*, fear *vs.* relief, hunger *vs.* satiety, vertical red bar on green field *vs.* vertical green bar on red field, *etc.* The derivation proceeds in three steps (Grossberg, 1972b).

(1) Sudden offset of a conditioned cue input can cause a rebound, much as offset of a conditioned source of fear can elicit relief (Denny, 1971; Masterson, 1970; McAllister and McAllister, 1970). To accomplish this rebound, the cue input is delivered to the network at a stage before the transmitter gate. Only in this way can the cue deplete the gate so that its offset can drive a rebound.

(2) Onset of a cue input can elicit sampling signals capable of encoding a rebound in LTM, much as a tone that turns on contingent upon shock offset can become a source of conditioned relief (Dunham, 1971; Dunham *et al.*, 1969; Hammond, 1968; Rescorla, 1969; Rescorla and LoLordo, 1965; Weisman and Litner, 1969). Thus, the cue input is delivered to the network at a stage after the transmitter gate, where the rebound can be sampled.

(3) Properties (1) and (2) are true for all cues that can be conditioned to these categories, since whether a given cue will be conditioned to onset or to offset of any particular category is not known *a priori*. Thus, every cue input is delivered both before and after the transmitter gating stage! The transmitter gate thus occurs in a feedback pathway, as in FIGURE 15.

The existence of two distinct derivations leading to a similar network design is important, since not every recurrent network that can be reset by offset of a cue need possess a mismatch-contingent arousal source, even though an arousal source *per se* is required. These derivations suggest that the anatomical design in FIGURE 15 is basic and that the input mechanisms that control rebound in this common design can be adapted to satisfy specialized processing constraints.

One further constraint can readily be satisfied by this design. The cue inputs arrive before the stage of dipole competition so that at most one of the complementary outputs (on-cell *vs.* off-cell) can be positive at any time. The next section depicts the minimal anatomy that joins together gated dipole feedback pathways and conditionable cue input pathways that terminate before the dipole competition stage.

30. DRIVES, CONDITIONED REINFORCERS, INCENTIVE MOTIVATION, AND CNV

FIGURE 16 depicts such a minimal anatomy and assigns to its pathways a motivational interpretation. In FIGURE 16, the specific inputs to the gated dipole are of two kinds: internal drive inputs and external cue inputs. For definiteness, let the positive drive input increase with hunger and let the negative drive input increase with satiety, either due to gastric distension or slower metabolic factors (Anand and Pillai, 1967; Janowitz *et al.*, 1949; Le Magnen, 1972; Sharma *et al.*, 1961). Let the drive inputs and the nonspecific arousal input be gated by a catecholaminergic transmitter in both the on-channel and the off-channel to calibrate the proper relative sizes of on and off dipole responses.

Let each external cue input send a conditionable pathway to both the on-channel and the off-channel of the dipole, so that each cue can become a conditioned reinforcer

of either positive or negative sign, depending on whether the LTM trace is larger in its on-channel or in its off-channel. To calibrate the relative sizes of these LTM traces in an unbiased fashion, I assume that the transmitter system that subserves LTM encoding in both branches is the same and is cholinergic. These chemical interpretations may eventually have to be changed, but the processing requirements of accurately calibrating relative rebound or conditioned reinforcer strength across competing channels are robust.

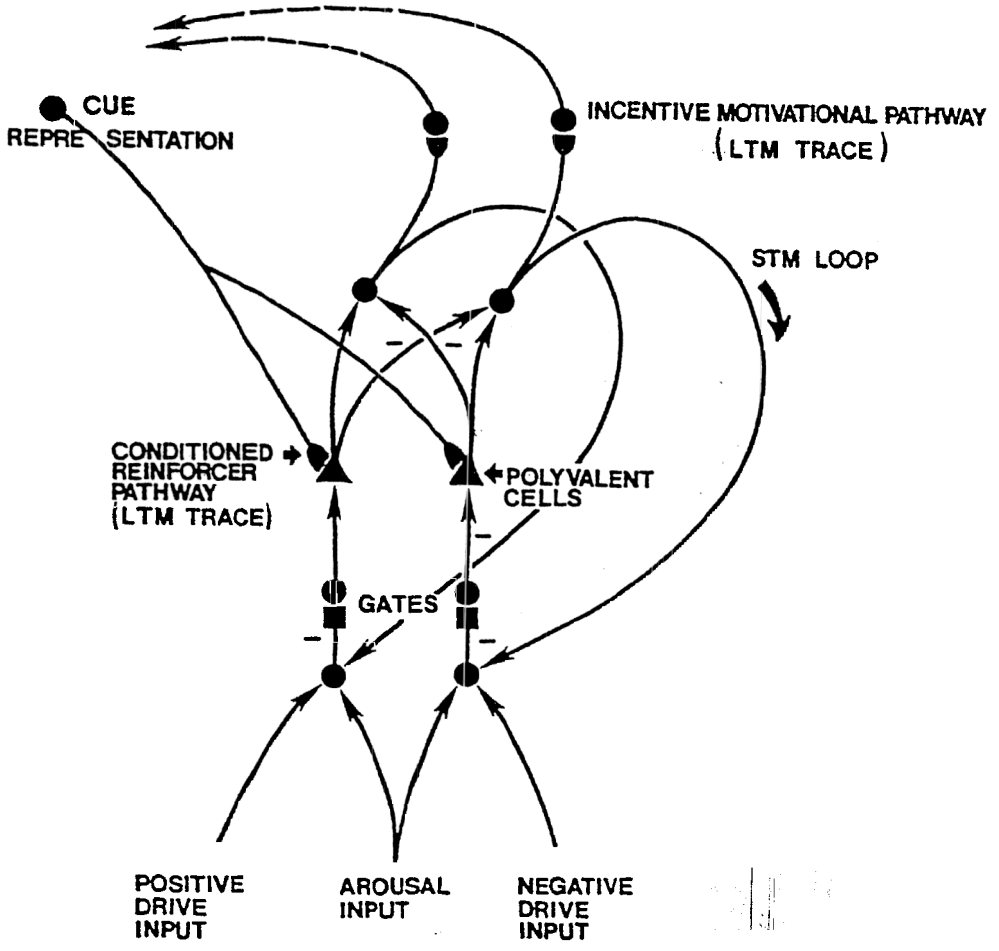


FIGURE 16. Drive inputs and (conditioned) reinforcer inputs are the specific inputs to a gated dipole whose outputs deliver (conditioned) incentive motivational signals back to internal representations of external cues. These outputs also feed back to a stage before the slow gates, so that offset of a reinforcing signal can drive a rebound, whereas onset of a conditioned reinforcer pathway can initiate encoding by its LTM traces of the gated activity pattern. This STM feedback loop can store a motivational decision against small input perturbations (hysteresis), maintain a steady motivational baseline (normalization), and regulate sharp motivational switching (contrast enhancement).

The cells at which external cue, drive, and arousal inputs converge are assumed to be polyvalent: These cells cannot fire vigorously unless both their external cue and their internal drive inputs are sufficiently large. The outputs from these polyvalent cells compete before generating a net dipole output in either the on-cell or the off-cell channel, but not both. These dipole outputs play the role of incentive motivation in the theory.

Drives, reinforcers, and incentives are conceptually distinct in FIGURE 16, by

contrast with several other motivational theories (Bolles, 1967; Estes, 1969; Mackintosh, 1974; Miller, 1963). For example, Mackintosh (1974, p. 233) writes: "Stimuli associated with reinforcers do not motivate instrumental responses" and advocates "discarding the idea of incentive as motivation and replacing it with the idea of incentive as the anticipation of a goal." Part of the difference between our two positions is semantic, since Mackintosh is arguing against the classical view of Hull and Spence that incentive motivation is a type of nonspecific motivation that activates all response tendencies indifferently. The present theory also argues against this position since, within it, incentive motivation is carried by a conditionable pathway that can differentially amplify certain STM representations above others due to prior conditioning. I also suggest that this conditionable incentive pathway subserves a motivational component of the CNV (Cant and Bickford, 1967; Grossberg, 1975; Irwin *et al.*, 1966). However, the present theory recognizes and distinguishes both the motivational and the expectancy properties of cues and views Mackintosh's reaction against Hull and Spence as an instance of throwing out the baby with the bathwater.

The existence of polyvalent cells is compatible with the intuition that incentive motivation need not be released even when drive is high if compatible cues are unavailable. For example, a male animal left alone will busily do the many things characteristic of his species, such as grooming, eating, and exploring. He does not look sexually motivated. However, if a female animal is presented to him, his behavior can change dramatically (Beach, 1956; Bolles, 1967, Chapter 7). Incentive motivation need not be released even when compatible cues are available if drive is low. Seward and Proctor (1960) and Seward *et al.* (1958, 1960) found that, if an animal is not hungry, then no amount of food will be adequate to reinforce its behavior.

31. EXTINCTION, CONDITIONED EMOTIONAL RESPONSES, CONDITIONED AVOIDANCE RESPONSES, AND SECONDARY CONDITIONING

This section reviews how LTM sampling by each cue of both the on-channel and the off-channel contributes to the explanation of some basic motivational processes (Grossberg, 1972a,b) and thereby indirectly supports the claim that both LTM pathways need to be built up from similarly calibrated transmitter mechanisms.

A similar design will be used in Section 57 to explain how incentive motivational feedback regulates attentional processing. The simplest LTM law says that LTM encoding occurs only when a cue pathway and a contiguous polyvalent cell are simultaneously active.

Suppose that, during early learning trials, a cue occurs just before a large unconditional signal, such as a shock, which turns on the on-channel. This unconditional signal elicits the incentive output of the on-channel, which triggers a fear reaction, even before learning occurs. By associating the cue with shock on learning trials, its LTM trace abutting the on-channel grows much larger than its LTM trace abutting the off-channel. If the cue is then presented by itself, the LTM-gated signal to the on-channel is much larger than the LTM-gated signal to the off-channel. The on channel wins the dipole competition, so the cue elicits fear. The cue has thus become a conditioned reinforcer that can elicit a conditioned emotional response (CER) (Estes and Skinner, 1941).

Now suppose that environmental contingencies change after the cue has become a CER. Suppose that the cue no longer reliably predicts future events and that an unexpected event occurs while the cue is on.

Suppose that the unexpected event triggers an antagonistic rebound in the off-channel. Since the cue is on, its LTM trace abutting the off-channel will grow. If this occurs sufficiently often, the off-LTM trace will grow as large as the on-LTM trace. After this happens, presenting the cue will generate comparable LTM-gated signals to both the on-channel and the off-channel. After these signals compete, the net incentive motivational output will be very small. The cue is no longer a CER. It has been rapidly extinguished by unexpected events.

This cue is extinguished because it remains on both before and after the unexpected event. It is an irrelevant cue with respect to the contingency that triggered the unexpected event. By contrast, a cue that turns on right after the unexpected event occurs will sample only the off-reaction of the dipole. Only its LTM trace abutting the off-channel will grow. Later presentation of the cue will elicit a large off-reaction. If the off-reaction corresponds to a relief reaction, then the cue has become a source of conditioned relief by being paired with offset of a source of fear. Although the cue has never been paired with a positive reward, it can thereafter be used as a positive reinforcer or source of consummatory motivation. This mechanism helps us to understand how avoidance behavior can be persistently maintained long after an animal no longer experiences the fear that originally motivated avoidance learning (Grossberg, 1972a,b, 1975; Maier *et al.*, 1969; Seligman and Johnston, 1973; Solomon *et al.*, 1953).

A similar argument shows how secondary conditioning can occur. For example, offset of a positive (or negative) conditioned reinforcer can drive an antagonistic rebound that conditions a cue whose onset is contingent upon the offset event to be a negative (or positive) conditioned reinforcer. This mechanism uses the feedback in the gated dipole in a major way. Offset of the reinforcer can elicit a rebound because it occurs at a stage before the gate, whereas sampling of the rebound can occur because the new cue delivers its signals at a stage after the gate.

32. CHOLINERGIC-CATECHOLAMINERGIC INTERACTIONS IN DRINKING VS. THE BRAIN AS A CHEMICAL BATH

Various data about drinking are clarified by the conceptualization of drives, reinforcers, and incentives and by the labeling of cholinergic and catecholaminergic interactions in FIGURE 16. The main difference between feeding and drinking from the viewpoint of FIGURE 16 is the use of thirst and satiety drive inputs instead of hunger and satiety drive inputs, and the existence of different prewired US inputs and possibly different feature fields of CS inputs to the two types of gated dipoles. The theory suggests that similar formal properties hold in cognitive networks. Since the motivational data base seems to be a more developed source of examples at the present time, it will be used in the next few sections to support some of the theory's implications.

Theories about drinking have often grown from the hope that there exist unitary behavioral correlates of pharmacological manipulations, as if the brain were a homogeneous chemical bath that reacts linearly to its chemical environment. This attitude was encouraged by early results in which injection of norepinephrine elicited feeding, whereas injection of cholinergic drugs elicited drinking at the same hypothalamic site (Fisher and Coury, 1962; Grossman, 1962). These results did not long survive further experimentation.

The reader can interpret all the following data in terms of the cholinergic-catecholaminergic interactions that are hypothesized to occur at gated dipoles. Degeneration of monoamine neurons due to injection of 6-hydroxydopamine (6-OHDA) can cause both adipsia and aphagia (Smith, 1973; Ungerstedt, 1968).

Anticholinergic drugs such as scopolamine can inhibit cholinergic drinking but not angiotensin or isoproterenol drinking, although anticholinergic drugs only partially block natural deprivation thirst at drug levels that totally abolish cholinergic drinking. Haloperidol, a selective dopamine blocker at low dosages, can suppress angiotensin drinking but spare cholinergic drinking. The combined effects of scopolamine and haloperidol can be additive, leading to total suppression of drinking (Fisher, 1973). Such experiments led Fisher to conclude that "It appears highly unlikely that *any* of the major components of thirst utilize or are entirely dependent upon a single transmitter substance" (p. 260). Fisher (1973) found this conclusion "discouraging . . . what is perhaps surprising is the lack of evidence for a single final common path that would be dependent on the availability of a particular transmitter" (p. 260).

My theory was capable of explaining these data when Fisher expressed this opinion (Grossberg, 1972a,b), but possibly because the theory was derived from behavioral postulates concerning an animal's adaptability to a fluctuating environment, its relevance has not yet been noticed by experimentalists on hunger and thirst. This is true, I believe, because a gap exists between known experimental data and the functional properties whereby the theory unifies these data. This gap is partly due to the difficulty of undertaking interactive experiments, but even more so to the lingering view that the brain can be treated like a chemical bath.

This view is illustrated by recent writings of even so distinguished a contributor as Olds (1977), who discusses how monoamine transmitters might influence the rewarding effects of brain stimulation in the following terms:

> . . . if the amines packaged and ready in vesicles inside of nerve terminals were required to make brain stimulation rewarding, then getting them out of the vesicles should have damaged rather than improved this effect. If the amines were already released, and if this was all there was to reward, then why was the animal still stimulating its brain? (p. 60)

Because the brain's functional heterogeneity is not well approximated by a chemical bath analogy, questions concerning whether transmitter is packaged or released at isolated synapses provide little functional insight.

A distinction that is often missed in the drinking literature is between expectancy mechanisms and drive or reinforcement mechanisms. This confusion can possibly be cleared up by adding an ERP manipulation that can more directly measure expectancy changes. An instance of this confusion is summarized in the next section.

33. INTRAGASTRIC VS. NORMAL DRINKING

Kissileff (1973) compares a rat's lever-pressing behavior for water that it can drink with its lever-pressing behavior for water that is intragastrically self-injected. On the first day that an animal is switched from drinking to intragastric self-injection, it self-injects far more fluid than it would normally drink, and its first bout of drinking is usually prolonged. After several days of self-injection, lever pressing occurs with reduced frequency. Kissileff attributes the initial overinjection of fluid to an oropharyngeal expectancy of fluid that is not fulfilled. Kissileff compares these data to data of Miller *et al.* (1957), who show that less water is drunk following fifteen minutes of drinking than following fifteen minutes of intragastric injection of the same amount of water. Kissileff suggests that, in the latter case, an oropharyngeal expectancy has not been met.

Without further analysis, these data do not unambiguously implicate an expectancy in the mechanistic sense. I shall indicate how the data might be explained in terms of drive and conditioned reinforcer concepts. I do not deny the possible influence

of expectancies in this situation. In fact, I will suggest that, if the expectancy mechanism were rate-limiting, it might lead to the opposite conclusion, which can be tested using a P300 measure.

First let us realize that a lever-press command and a taste pathway might control different levels of net positive incentive if only because of differences in their times of activation during a meal. An elegant Le Magnen (1972) experiment illustrates this distinction. Le Magnen associated a different taste, A, B, C, or D, with the onset of each meal during learning trials. During test trials, he introduced distinct tastes at quartiles of a single meal and noted that food intake was amplified almost four-fold. An experiment should be done in which the distinct tastes, A, B, C, or D, are associated with the same stage of each meal, but the stage should be varied across animals from meal onset to satiety. One would then test how presenting the distinct tastes during quartiles of a single test meal alters total food intake as a function of how satiated the animal was when the taste was presented during learning trials. Let us suppose, for the sake of argument, that the total food intake decreases as the association is made closer to satiety.

Now consider an experiment in which a lever press precedes a large food or water reward. The lever press occurs while the net appetitive drive is high; it is, therefore, associated with a maximally positive reaction at the drive representations. Taste and other oropharyngeal pathways that remain active throughout the ingestional interval are, by contrast, associated with incentive motivational patterns that become progressively less positive as fast satiety reactions start to occur. Thus a lever press cue might, by virtue of the instrumental contingency, control a more positive incentive reaction than oropharyngeal cues. Consequently, in the absence of oropharyngeal cues, the larger positive incentive reaction controlled by the lever press cues might cause more eating or drinking to occur, even if no expectancies are operative. This is the first alternative to the expectancy explanation that must be ruled out. If the animal is willing to work harder for larger rewards in a given situation, then conditioned reinforcer factors can play an important role. They can arise due to an interaction between a cue's developing conditioned reinforcer properties and the cue's times of occurrence within the time intervals between high drive and satiety.

A second alternative that must be ruled out is that oropharyngeal receptors, such as those sensitive to mouth dryness or temperature, might generate drive inputs when the mouth is too dry, or hot, and that such drive inputs could activate their drive representations until consummatory behavior rapidly reduces the drive inputs by moistening or cooling the mouth. This oropharyngeal mechanism is related to an expectancy mechanism, but is not itself an expectancy mechanism. It is related to an expectancy mechanism because when the drive inputs are matched by appropriate conditioned reinforcer inputs at the drive representations, then the system can go into a resonance that triggers consummatory behavior. Can a characteristic negative wave be associated with this resonance? The main fact to be explained is not, however, that the resonance occurs, but why it lasts longer if water is not drunk. This can be explained by the drive properties of the system, not its expectancy properties: The oropharyngeal drive is reduced when the water is drunk. Where the expectancy properties of the system are dominant over its drive properties, the opposite result might well occur, since disconfirming the expectancy of water might extinguish the lever press, rather than prolong it. P300 experiments to more directly test this assertion would greatly clarify the meaning of these data. Such an expectancy effect might well be in these data, but it often acts on a slower time scale than a single bout of drinking.

An example of another slowly developing oropharyngeal conditioned reinforcer change seems to be prandial drinking, which is significantly controlled by oropharyngeal factors related to eating dry food. Prandial drinking develops gradually in rats

who are recovering from lateral hypothalamic lesions, in desalivate rats, and in rat pups (Kissileff, 1969, 1971; Teitelbaum and Epstein, 1962).

Another way of pointing out the problem in assigning expectancy properties a rate-limiting role in these data is the following. Bellows (1939) has demonstrated the persistence of drinking in dogs after esophagostomy. As Kissileff (1973) himself notes, these data demonstrate "facilitation by oropharyngeal stimuli associated with drinking in water lack and the necessity of postingestive stimuli for relatively sustained inhibition of drinking" (p. 173). If oropharyngeal stimuli facilitate drinking, then why do animals drink less when oropharyngeal cues are activated than when drinking is intragastric? These data again implicate either differential conditioned reinforcer properties of lever-press *versus* oropharyngeal cues or the drive-reducing effects of water on oropharyngeal receptors, rather than expectancy signals *per se*.

Various other properties of eating or drinking need to be mechanistically distinguished. For example, the eating rate can be transiently accelerated at the onset of an eating bout (Booth *et al.*, 1976) and is a joint function of food palatability and prior food deprivation. Is the rate reduction due to intracellular habituation of transmitters in the active incentive motivational feedback loops or to the action of fast satiety reactions? By explicating motivational concepts clearly enough, experiments can be designed to test such differences.

34. SCHEDULED-INDUCED POLYDIPSIA, FRUSTRATION, AND EXPECTANCY MATCHING

A motivational phenomenon wherein expectancies do seem to play a dominant role is schedule-induced polydipsia (Falk, 1961a,b), which has not previously been mechanistically explained. This phenomenon vividly demonstrates how far an animal's drinking behavior can deviate from homeostatic control. Very hungry animals drink much more water than they need if food is intermittently available on a spaced reward schedule and water is freely available (Hudson and Singer, 1979; Kissileff, 1973; Rossellini, 1979; Wallace and Singer, 1976). The degree of polydipsia can be decreased by decreasing an animal's hunger, increasing the amount of food reinforcement, or increasing the palatability of food. Kissileff (1973) summarizes evidence, such as that above, which suggests that frustration plays a role in regulating schedule-induced polydipsia, notably frustration reduction when water is drunk to reduce the frustrating effects of the nonoccurrence of expected food (Grossberg, 1975). He goes on to say about the frustrative hypothesis that "it classifies a mystery as an enigma" (p. 167). In FIGURE 16, the sudden decrease of a positive reinforcer can elicit a frustrative rebound to drive basic processes of secondary conditioning and extinction. To the extent that frustration is an enigma, the basic processes themselves are enigmas, which is unfortunately true within the traditional motivational literature. Why these frustrative effects have seemed to be enigmatic in the experimental literature is clarified by the following theoretical considerations.

After the organism builds up an expectancy that a cue will occur, the unexpected nonoccurrence of that cue can cause widespread antagonistic rebounds, some of them frustrative rebounds. The offset of an expected cue can, however, elicit an antagonistic rebound by two distinct mechanisms, which develop at different rates through time: offset of the cue as a conditioned reinforcer, the effect of which is to withdraw activity along specific pathways, and offset of the cue as an expected event, the effect of which is to increase activity along nonspecific pathways. My discussion of overshadowing in Section 57 will, in fact, suggest that a rebound driven by the latter mechanism can sometimes elicit a rebound driven by the former mechanism. Both of these events are

presently hypothesized to cause P300s, but P300s with different scalp distributions due to their hypothesized generators in neocortex and hippocampus, respectively. A successful addition of a P300 manipulation to such experiments would provide important information, especially since schedule-induced polydipsia is not the only interim, or adjunctive, behavior that can occur if food is scheduled to be intermittently available to very hungry animals. For example, aggression can occur if a target animal is available (Azrin *et al.*, 1966) even if drinking occurs when water is available (Falk, 1971). The particular interim responses that will occur depend both on the organism being studied and on the environmental possibilities to which the organism is exposed (Staddon and Ayres, 1975). In addition to understanding how certain interim responses can reduce frustration, we must therefore also study how the several motivational sources in a dipole field compete among themselves to determine which one will control overt behavior.

35. SELF-STIMULATION AND KINDLING

The distinctions among drive, reinforcement, and incentive motivation are also basic in explaining effects such as self-stimulation, wherein the stimulating electrode can act like an artificial drive input whose behavioral effects, *via* an incentive motivational output, can be augmented or diminished by the concurrent action of natural drive and conditioned reinforcer inputs (Grossberg, 1971; Olds, 1977). For example, rats work harder to receive a burst of electrical impulses than a single pulse (Olds, 1977). This fact is explicable because the polyvalent cells must integrate their afferents over time before they are sufficiently stimulated to overcome their polyvalent thresholds. Also, a signal that predicts brain reward greatly augments the number of pedal presses that a rat will make for the reward (Cantor, 1971). This fact is explicable if the signal becomes a conditioned reinforcer: Signals (*e.g.*, tones) associated with brain reward can become rewarding (Stein, 1958; Knott and Clayton, 1966; Trowill and Hynek, 1970) in the theory because, when activity in the cholinergic pathway is correlated with active reverberation in the recurrent catecholaminergic pathway, LTM traces at the jointly active synaptic interfaces are enhanced. Consequently, if a signal comes just before brain reward, it can greatly augment the number of pedal presses that a rat will make for that reward, since the strengthened cholinergic pathway activated by the signal abets firing of its polyvalent target cells. From this perspective, the anatomical overlap between self-stimulation sites and reward sites (Hoebel and Teitelbaum, 1962; Margules and Olds, 1962; Caggiula and Hoebel, 1966; Mogensen and Stevenson, 1966) exists because the sensory cues that eventually elicit self-stimulation do so by becoming conditioned reinforcers *via* the cholinergic pathways whose LTM traces are altered by the stimulating effects of the electrode on polyvalent cell firing.

The status of electrode input as an artificial drive input whose signals converge on polyvalent target cells is supported by data showing that, at electrode currents below the rewarding threshold, electrodes in the hypothalamic drinking center will elicit self-stimulation only if water (acting like a reinforcer) is available (Mendelson, 1967; Mogensen and Morgan, 1967). Similar data have been collected with electrodes placed in a feeding center in the presence of food reward (Coons and Cruce, 1968; Poschel, 1968). The ability of electrode inputs to summate with natural drive inputs, as well as the specificity of drive representations, is illustrated by data in which rats were shown to press one lever when hungry and another lever when thirsty (Gallistel and Beagley, 1971), female rats lever pressed at a rate that varied with their estrous cycles (Prescott,

1966), and male rats lever pressed at a rate that varied with experimentally manipulated androgen concentrations (Olds, 1958).

A subtle feature of self-stimulation studies is that a rewarding train of impulses can be made aversive either by increasing the duration of the impulse train or by preventing the animal from controlling the onset times of stimulating pulses (Olds, 1977). The latter effect is understandable if uncontrolled electrode inputs are occurring at a time when an unexpected event triggers an antagonistic rebound that, in this case, activates a negative incentive motivational pathway. I do not argue this too forcefully, however, because arguments about an animal's expectations are routinely confused in the experimental literature with arguments about nonexpectational habituation processes. Adding a P300 measure to these studies can play an important role in overcoming these confusions.

Let me focus instead on the paradox inherent in the fact that elongating rewarding

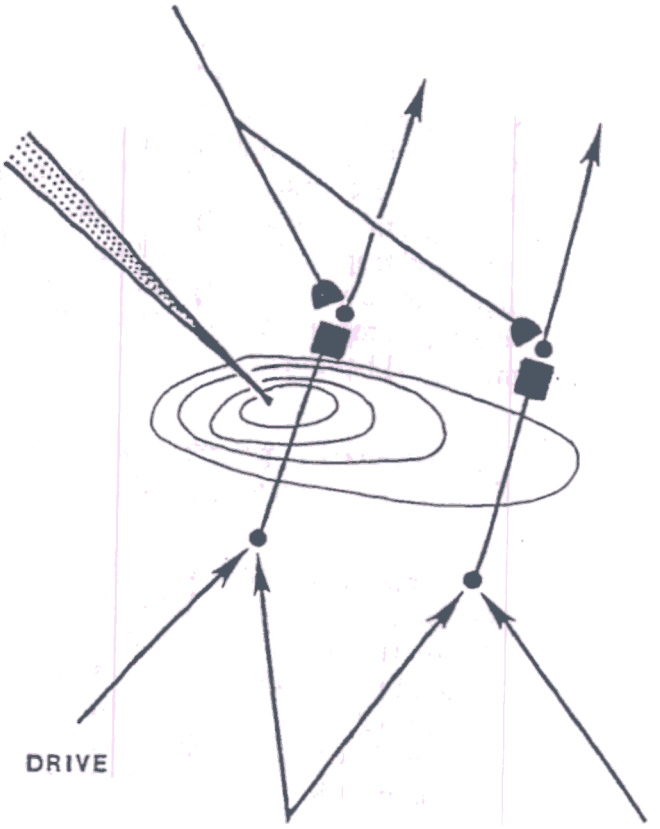


FIGURE 17. An electrode input acts like an artificial drive input to a contiguous drive representation. It thereby abets firing of this representation but can also depress the feedback loop's sensitivity by depleting its transmitter gate.

pulse trains can make them less, rather than more, rewarding. I suggest that two effects going on at once, in opposite directions, at different rates, and within distinct anatomical pathways can explain this effect. Moreover, these two effects are inherent in our discussion of cholinergic-catecholaminergic interactions. Consider FIGURE 17. FIGURE 17 depicts an electrode in a positive incentive pathway. The electrode can also, possibly, produce a smaller input in a nearby negative pathway by electrical currents. The electrode must be turned on long enough to overcome the polyvalent cell threshold in its pathway and to win the competition between pathways for storage in short term memory. Other things being equal, these effects of electrode input will strengthen the active cholinergic synapses that converge on the active feedback pathway. Past a certain electrode input duration and intensity, however, the catecholaminergic transmitter is depleted, or habituates, to such a low level that the net signal due to electrode

input plus drive input plus arousal input, all gated by the depleted transmitter in the positive pathway, is smaller than the net signal due to (possible) electrode input plus drive input plus arousal input, all gated by a less depleted transmitter in the negative pathway. Then the net gated signals, after the competition takes place, favor the negative pathway. The negative effect of long electrode bursts is suggested to be an interactive effect due to depletable transmitter gates in competing pathways.

This explanation suggests why kindling is more efficacious in response to series of brief electrode bursts than in response to a single burst of the same total duration (Goddard, 1967). The single burst maximizes the depletion of the transmitter, whereas the shorter bursts are long enough to open the feedback loop but short enough to prevent the transmitter from being unduly depleted during each burst. In particular, the interburst duration must be sufficiently long to offset intraburst habituation effects. Consequently, each shorter burst can drive the learning process which, in turn, can enhance the reaction on successive learning trials. One further fact is needed to qualitatively understand kindling; namely, that the conditionable pathway is also part of a feedback loop whose activation is easier on successive learning trials for the same reason that conditioned reinforcers abet the firing of polyvalent cells. That conditionable pathways are part of feedback loops is a basic fact about adaptive resonances. But is the feedback pathway that augments the kindling reaction *via* conditioning the same feedback pathway that reduces the kindling reaction *via* transmitter habituation? In general, I suggest that the answer is "no." For example, the gates that habituate during an STM resonance are not the same pathways as the LTM traces that encode new filtering and expectancy patterns. Kindling experiments that can differentiate these predicted opposite effects along conditioning-reset and cholinergic-catecholaminergic dichotomies will greatly enhance our understanding of the kindling phenomenon by relating it more closely to the large literature on psychopharmacological substrates of motivated behavior.

36. CRITICAL PERIOD REVERSAL, P300 SUPPRESSION, AND A TEST OF LTM ENCODING BY CHOLINERGIC PATHWAYS

A major design feature in FIGURE 16 has not yet been exploited. Since a similar design is suggested to hold in cognitive as well as motivational dipole fields, I will use this feature to suggest an explanation of the Pettigrew and Kasamatsu data on critical period termination and reversal. I will also relate this explanation to a P300 prediction. Then I will suggest an experiment to decide between two alternative hypotheses about how cholinergic-catecholaminergic interactions control LTM changes.

In FIGURE 16, the incentive motivational output bifurcates. One branch sends signals to perceptual and cognitive thalamocortical representations. The other branch feeds back to excite its own pathway at a stage before its transmitter gate. After a polyvalent cell fires, it competes with other polyvalent cell channels to test which channels can return positive feedback to themselves. A winning channel can resonate in STM *via* its feedback pathway. Such a resonance can sustain prolonged polyvalent cell firing, which can, in turn, drive LTM changes at active conditioned reinforcer synapses that abut the resonating polyvalent cell.

What would happen to such a circuit if 6-OHDA poisoned the transmitter gate? By destroying the gate, the possibility of STM resonance is also eliminated, and, with it, LTM plasticity. What would happen if catecholamine were poured over a cortical dipole field? Such a manipulation would have two major effects. It would bypass the buffering mechanism that depends on antagonistic rebound to maintain code stability. It does this by upsetting the relative balance of transmitter between on-cell and off-cell

channels. It would also override polyvalent constraints by directly activating the synapses postsynaptic to the transmitter gates. The combined effects of these actions would be to allow various combinations of inputs that could not previously overcome buffering and polyvalent constraints to elicit STM resonance and subsequent LTM encoding.

If a P300 measure is added to a 6-OHDA manipulation in the Pettigrew situation, then the P300 should be attenuated or obliterated, since antagonistic rebound cannot occur in the absence of the gates. If an experiment is chosen that elicits mismatch negativity both before and after the 6-OHDA manipulation, then the hypothesis that the generator of mismatch negativity is fedforward to the P300 generator would be supported (Section 21).

The 6-OHDA experiment focuses on the catecholaminergic contribution to LTM encoding. Part of the subtlety involved in teasing apart cholinergic from monoaminergic influences on learning is clarified by the theoretical conclusion that the target cells where these influences converge are polyvalent cells. For example, conditioned reinforcer, drive, and arousal influences must all be sufficiently active before the target cell can fire vigorously enough to win the competition among drive representations and thereby amplify and maintain its activity *via* positive feedback. Learning can be prevented in a given cholinergic pathway if the catecholaminergic input to that pathway is too weak for the combined effects of its inputs to overcome the competition from other pathways. This learning deficit does not imply that the catecholaminergic transmitter directly regulates the chemistry of laying down an LTM trace *via* positive feedback. It only says that the catecholaminergic transmitter modulates a given pathway's ability to win the competition. The amplified activity due to positive feedback within the winning pathway might be the prerequisite postsynaptic condition for learning at the cholinergic synapse.

Catecholamines can influence LTM in either of two ways, neither of which contradict the hypothesis that the LTM trace is part of a cholinergic system. The weak hypothesis is that catecholamine input is a necessary condition for winning the competition among drive representations, but that the postsynaptic learning signals elicited by a winning pathway are not catecholaminergic; *e.g.*, they might be Ca^{++} or cAMP. The strong hypothesis is that catecholaminergic input directly drives the postsynaptic protein synthesis process, as well as postsynaptic-to-presynaptic learning signals, and that winning the competition merely amplifies this direct catecholaminergic effect. Even the strong hypothesis requires an extra chemical influence (cholinergic?) to select only active synapses to be the ones at which learning will occur.

One way to test this alternative is to insert electrodes in lateral hypothalamic sites at which an animal can learn to self-stimulate. Sites should be chosen (say) at which the animal presses the bar more for stimulation as it becomes thirstier (Olds, 1977). Then deplete catecholamines with 6-OHDA. Finally, seek a level of electrode stimulation that again supports self-stimulation. If sites at which new learning can be achieved in the absence of catecholaminergic involvement can be found, then the weak hypothesis is favored. Not all self-stimulation sites are theoretically plausible candidates for this manipulation. Only sites where iontophoretically applied acetylcholine elicits a rapid enhancement of self-stimulation should be considered. If the electrode is placed along the catecholaminergic feedback loop in FIGURE 16, but at a stage before the catecholaminergic gate, then 6-OHDA will obliterate the effect of electrode input at the polyvalent cells.

These distinctions between direct effects on LTM and modulatory effects due to drive and arousal manipulations enable us to interpret the following data implicating cholinergic influences on memory.

The large suppressant effect of atropine on psychogenic polydipsia (Burks and

Fisher, 1970) compared with its much smaller effect on regulatory drinking (Blass and Chapman, 1971) led Kissileff (1973) to conclude that "acetylcholine may play a greater role in emotionally driven drinking than in drinking driven by thirst" (p. 168). Otherwise expressed, atropine might act by weakening conditioned influences on drinking without weakening drive influences on drinking. Kissileff (1973) also reviews evidence that, when homeostatic mechanisms are eliminated by lateral hypothalamic damage, drinking can recover but under oropharyngeal controls. Routtenberg and Kim (1978) review evidence that neostriatal cholinergic interneurons play an important role in the long term memory consolidation process, including the fact that scopolamine, a cholinergic blocking agent, has a potent effect on human memory (Drachman and Leavitt, 1974). Mabry and Campbell (1978) claim that scopolamine affects adult but not immature rats, even though amphetamine can produce hyperactivity in immature rats. Is the differential effect of scopalamine at later ages due to the fact that learned representations have not been incorporated into the cholinergic system until these ages?

Marsh *et al.* (1971) have reported an impairment in intermediate, but not immediate, memory of Parkinsonian patients' performance on a paired associate task. L-dopa therapy is reported to improve the patients' performance on this task. At the present time, this memory impairment has not been unambiguously explained as a weak or strong involvement of the dopamine system in long term memory formation. An improvement in immediate memory of Parkinsonian patients after L-dopa therapy has also been reported (Barbeau *et al.*, 1971; Cotzias *et al.*, 1969). These data are also compatible with both the weak and strong hypotheses, since, in both cases, a catecholaminergic transmitter gates the feedback pathways that store the neural activity in STM.

37. GESTALT SWITCHING DUE TO AMBIGUOUS FIGURES AND HABITUATION INDUCED SHIFTS OF CATEGORICAL BOUNDARIES

My explanation of the Pettigrew and Kasamatsu data shows how P300 and negative ERP components might be pharmacologically dissociated. Such a dissociation should also be possible without pharmacological intervention. A P300 that is elicited without prior mismatch negativity and that precedes rather than follows processing negativity should occur during perceptual switches between alternative interpretations of an ambiguous figure, such as the Necker cube or monocularly rivalrous patterns (Brown, 1965; Rauschecker *et al.*, 1973). The tendency to alternate can be explained by the fact that persistent reverberation within an active STM representation tends to deplete its transmitter gates, thereby weakening the reverberation and providing a growing advantage to the inhibited, and therefore undepleted, alternative STM representation. When the active STM representation reaches a critical level of depletion, the alternative representation is sufficiently disinhibited to let the competitive feedback dynamics contrast enhance its activity into a state of STM resonance. This STM switch can be driven thalamocortically, without intervention of a mismatch-initiated arousal burst, by the interaction of competitive feedback and STM-induced habituation of transmitter gates. If the perceptual switches are associated with massive off-rebounds, then they should elicit a sequence of P300s. After a switch occurs, a newly activated STM representation can read out a feedback expectancy whose match with the ambiguous data can elicit processing negativity. Papakostopoulos (1976) suggests a similar processing concept when he writes that P300 acts "to arrest planned behavior or to generate the bases for alternation from one behavioral act to another."

It might be difficult to measure these P300s because they occur spontaneously rather than under experimental control. A helpful fact is that the alternation rate in monocular rivalry is up to three times faster with gratings constructed from complementary colors than with black and white gratings. I suggest an explanation of this fact in Grossberg (1980a).

A similar combination of gate habituation and STM competition can help to explain shifts in categorical boundaries that are not due to new LTM encoding (Sawusch and Nusbaum, 1979). Situations in which gestalt switches occur should also elicit categorical boundary shifts, but experimentalists have often been more interested in the switches *per se* than in testing this hypothesis.

38. MOTIVATIONAL SWITCHING AND HYSTERESIS WITHOUT A MYTHICAL CUSP CATASTROPHE

Some fundamental mathematical properties of gated dipoles will now be summarized. Proofs of the inverted U properties are given in the appendix.

The positive feedback loops in the gated dipole of FIGURE 16 turn this network into a feedback competitive network. The slowly varying transmitter gates do not alter the fact that a feedback gated dipole shares many properties with other feedback competitive networks. For example, the dipole now has an STM storage capability, which means that it can defend a motivational decision against sufficiently small momentary fluctuations in cue or arousal inputs. This hysteresis property helps to provide the inertia needed to carry out a sustained motivated act during irrelevant environment perturbations. The STM normalization property refines this capability by maintaining a temporally stable baseline of incentive motivation. The contrast enhancement property helps to control sharp motivational switching between behavioral alternatives when the net balance of inputs succeeds in overcoming the hysteretic inertia of the previous motivational choice.

Frey and Sears (1978) have built sigmoid and hysteresis properties into a cusp catastrophe model of conditioning and attention. Although their model provides one way to visualize sudden switches, the catastrophe variables do not correspond to physical variables and the model provides no physical explanation of why sigmoid and hysteresis properties appear in the data. The gated dipole theory provides a physical explanation that does not correspond to a cusp catastrophe and implies a large body of data and predictions, such as those below, which are invisible to the cusp picture.

The STM hysteresis and normalization properties do not depend on the transmitter gates. Some deeper properties do. A sigmoid signal function is the simplest function that can overcome noise amplification in a feedback competitive network (see Section 20). When sigmoid feedback signals are used in a gated dipole, this network possesses inverted U properties that are reflected in a large body of data about normal and abnormal behavior. I will present these properties in constellations to help experimentalists decide when gated dipoles are generating the data. I will first state the formal properties using the motivational terminology in FIGURE 16 to fix ideas.

The main new fact is this: If the arousal level is chosen either too small or too large, both the on-reactions and the off-rebounds of the dipole are severely reduced. In motivational terms, either underarousal or overarousal can cause emotional depression. The underaroused depressive syndrome is, however, starkly different from the overaroused depressive syndrome both in its etiology and in its constellation of properties (Grossberg, 1972b).

39. FORMAL SYMPTOMS OF UNDERAROUSED DEPRESSION

The following properties of an underaroused gated dipole are proved in the Appendix and will be used to discuss clinical syndromes and drug effects in the next few sections.

(1) High threshold: The threshold phasic input (*e.g.*, intensity of conditioned reinforcer or perceptual event) that can elicit a supraliminal output is abnormally high.

(2) Suprathreshold hyperexcitability: The sensitivity of the dipole to suprathreshold phasic input increments is abnormally high. In other words, a fixed increment in phasic input within the suprathreshold range can elicit an abnormally large on-reaction output.

Thus, the underaroused syndrome is hyperexcitable despite an elevated threshold. The reader may find this result paradoxical, because a low threshold often implies high suprathreshold sensitivity.

(3) Paradoxical on-reaction to unexpected events: The threshold arousal increment that can elicit an antagonistic rebound is abnormally high. Smaller arousal increments elicit an enhanced on-reaction, despite the fact that they would have elicited off-rebounds in a normally aroused dipole.

(4) Paradoxical insensitivity to phasic decrements: A rapid reduction of phasic input from an intensity of J to half intensity, $J/2$, may not elicit a rebound, even though reduction of $J/2$ to 0 does elicit a rebound. This property is a special case of a formula that determines when reduction of input intensity J_1 to intensity K_1 at arousal level I_1 will elicit a larger rebound than reduction of input J_2 to K_2 at arousal level I_2. The interesting feature of reducing J to $J/2$ as compared to reducing $J/2$ to 0 is that both manipulations cause a change of $J/2$ units in phasic input.

In Grossberg (1972b), I used this property to predict how rewarding arbitrary shock offset combinations will be, and to explain the known advantage of $J/2 \rightarrow 0$ over $J \rightarrow J/2$ offset. I also related the size of this advantage to an animal's ability to learn escape from a discrete fear cue and to the advantage of partial reward over continuous reward. All these factors should vary together as the animal's arousal level is parametrically increased. I hope that the current interest in arousal-related diseases at the present time, which is greater than that in 1972, will encourage testing of these indices to sharpen our understanding of the transition from normal to abnormal syndromes.

40. FORMAL SYMPTOMS OF OVERAROUSED DEPRESSION

(1) Low threshold: The threshold phasic input that can elicit a suprathreshold output is abnormally low. However, this does not influence observable behavior because of (2) suprathreshold hypoexcitability: The sensitivity of the dipole to suprathreshold phasic input increments is abnormally low. In other words, a fixed increment in the phasic input within the suprathreshold range can elicit an abnormally small on-reaction output.

Thus, the overaroused depressive syndrome is a low-threshold, suprathreshold hyposensitive syndrome—again, a paradoxical combination.

In a feedback gated dipole, insufficient production of the gating transmitter can reduce the amount of feedback within the dipole and thereby depress its operating level. This type of depression can reduce the total nonspecific input to the dipole, but it

should not be confused with a reduction in the size of the externally applied nonspecific arousal input.

41. THE INVERTED U IS NOT A UNITARY CONCEPT

Inverted U effects are familiar both in psychophysiology (Hebb, 1955) and in discrimination learning (Berlyne, 1969). Lest the reader therefore casually dismiss the importance of the gated dipole syndrome, I should emphasize that there exists more than one type of inverted U. For example, Grossberg and Pepe (1970, 1971) showed that a different inverted U can occur during the processing of serial events, such as a list or a sentence. In a network capable of processing serial events—which is not a gated dipole!—the overaroused syndrome leads to contextual collapse, a reduction of associative span, and fuzzy response categories. We predicted that the approach to overarousal would manifest itself during serial learning by a reversal of the relative learning rate at the beginning *versus* the end of the list and by a shift of the list position of maximal learning difficulty (the bow in the serial position curve) from its skewed position nearer to the end of the list towards a symmetric position at the list's middle. Despite the importance of these predictions for understanding normal serial learning and simple schizophrenia (Maher, 1977) and overarousal disorders, they have not been tested or widely understood during the decade since their appearance.

42. INVERTED U IN P300 AND CNV

Inverted U's have been found in ERPs. Tecce and Cole (1974) reported an inverted U in the CNV, which will be attributed to the dynamics of a gated dipole field in Sections 51–53. The P300 is reduced both in hyperactive children and in schizophrenics, as the reviews (this volume) on aberrant development and psychopathology have shown. I do not, however, share the opinion that a low amplitude of P300 must reflect a deficit common to these diagnostic entities. I will instead argue that hyperactive children are underaroused and simple schizophrenics are overaroused in the gated dipole sense. The two types of P300 should therefore be reduced because they occur at opposite ends of the inverted U.

The next few sections summarize some facts about abnormal syndromes that are clarified by formal properties of the gated dipole inverted U.

43. PARKINSON BRACING, STARTING, AND STOPPING

In Parkinson's disease, dopamine-rich cells of the substantia nigra show marked degeneration (Weiner and Klawans, 1978). This structural correlate of the disease helps to rationalize the symptomatic improvement L-dopa therapy can effect, since L-dopa is a pharmacological "up." I suggest that its use lifts the underaroused gated dipoles that control the affected motor skills to a more normal range of dipole arousal and sensitivity. Animal models of Parkinson's disease support the underarousal hypothesis. Intraventicular application of 6-OHDA severely depletes brain catecholamines and thereby produces symptoms such as catalepsy, akinesia, and Parkinson bracing (Levitt and Teitelbaum, 1975; Schallert *et al.*, 1978a,b, 1979).

I suggest that the following parkinsonian symptoms are manifestations of the underaroused depressive syndrome. The higher threshold for activating a dipole manifests itself in the difficulties parkinsonian patients have in initiating movements. The suprathreshold excitability manifests itself in the difficulties these patients have in terminating movements after they begin; that is, after a large enough phasic input is applied to generate hyperexcitable suprathreshold cyclic behavior in the gated feedback loops of the underaroused motor command oscillators.

The reader might wonder at this point how oscillators have crept into the gated dipole story. G.A. Carpenter and I show in Carpenter and Grossberg (1983) that the activities of feedback dipoles with slowly varying gates can endogenously oscillate, even though feedforward gated dipoles and feedback dipoles with rapidly varying gates cannot oscillate. That story lies beyond the scope of this article, however.

A particularly interesting parkinsonian symptom is bracing; namely, "if suddenly pushed forward or backward while standing, many people with Parkinson's brace rigidly without stepping, or with short shuffling steps which are unable to counteract their fall" (Schallert *et al.*, 1979). Why don't these patients right themselves as normal people do, or just fall over? I associate this property with the paradoxical enhanced on-reaction that occurs in response to unexpected events in the underaroused depressive syndrome. An enhanced on-reaction would strengthen the current motor pattern, rather than rebounding it to an antagonistic pattern that would abet a righting reaction. At a somewhat higher arousal level (or transmitter concentration), small rebounds can occur in the gated dipole. I associate these small rebounds with the short shuffling steps that occur in 6-OHDA treated rats who have recovered a limited degree of spontaneous locomotion. To test this bracing hypothesis, one might try to measure the negative ERP component corresponding to the motor analogue of a mismatch-contingent arousal burst, and to show that a brace, as opposed to passive inaction, correlates with the size of this ERP in response to an unexpected push.

In the articles cited above, Teitelbaum and his colleagues have claimed that, in Parkinson's disease, the subsystem that maintains postural configurations, or static stable equilibria, is working properly, but that the subsystem that regulates dynamic transactions such as walking, orienting, and exploring is deficient. I agree with this claim to the extent that a dynamic deficiency exists because the rebound, or reset, capabilities of gated dipoles are depressed.

44. JUVENILE HYPERACTIVITY

I suggest that juvenile hyperactivity is another instance of the underaroused depressive syndrome. Certain hyperactive children suffer from catecholamine deficiencies (Shaywitz *et al.*, 1977; Shekim *et al.*, 1977). These data clarify why pharmacological "ups" like amphetamine can be helpful in treating these children (Swanson and Kinsbourne, 1976; Weiss and Hechtmann, 1979). Nonetheless, were it not for the theoretical analysis of dipole dynamics, why hyperexcitability should follow from underarousal would still be a mystery.

The dipole model also suggests an experimental question that still seems to be insufficiently studied: Are the behavioral thresholds of hyperactive behaviors higher than normal thresholds for these behaviors? At the EPIC VI meeting, Roy Halliday called my attention to the work of Weber and Sulzbacher (1975), who showed that thresholds during an electroencephalic audiometry test performed on hyperactive children were reduced by medication. If the paradoxical properties (3 and 4) of the underaroused syndrome also hold when threshold elevation is recorded, say in a study of reward and motivation, then a much stronger test would be achieved.

Before considering other underaroused syndromes, it will be useful to briefly mention two important overaroused examples.

45. SCHIZOPHRENIC OVERAROUSAL

Some types of schizophrenia have been ascribed to dopamine hyperactivity of cells in the ventromedial tegmental area, medial to the substantia nigra, that terminate in the limbic forebrain or cortex (Lloyd, 1978), thereby providing the basis for an overaroused syndrome. Dopaminergic agonists, such as L-dopa and amphetamine, can produce a behavioral syndrome, including repetitive activity or perseveration (read: breakdown of the reset system) that has been compared to schizophrenia (Riklan, 1973; Wallach, 1974). Various antipsychotic drugs block dopamine receptors (Kuhar *et al.*, 1978) and, in sufficient quantities, can produce a catalepsy that is reminiscent of Parkinson's disease (Hornykiewicz, 1975). The facts that an underaroused syndrome can be transmuted into an overaroused syndrome using a given drug and that the reverse transformation can be effected by an oppositely acting drug suggest that the two syndromes are extremal points on an inverted U of a common mechanistic substrate.

46. ANALGESIA: ENDORPHINS VS. LOUD NOISE

Overarousal is not always a bad thing. A high net arousal level can cause good as well as bad effects. Where such a high level is due to a nonspecific input, say due to loud noise (Gardner *et al.*, 1961), it can reduce the aversiveness (incentive output) of an unpleasant input, *e.g.*, shock. Such a nonspecific input might be caused by a baseline shift in reticular formation output in response to the noise. An effect akin to overarousal can be caused by a potent specific input to the competing dipole channel, say an increment due to increased production or release of endorphins (Gintzler, 1980; Guillemin, 1978).

Although both manipulations will depress the negative incentive output, they should nonetheless be experimentally distinguishable. The nonspecific input desensitizes the dipole's reaction to the aversive phasic input, but does not change the level of this input. The competing specific input, by contrast, has an effect equivalent to both increasing the arousal level and decreasing the aversive phasic input size.

This equivalence is based on a simple trick that greatly aids the understanding of gated dipoles. Many inputs can perturb a dipole through time. Conditioned reinforcer signals from a large number of internal representations, arousal and drive inputs, and internal feedback signals are all operative. The dipole cannot determine what the input sources are, but only their net effect. I therefore consider the total input size that is felt in the on-channel (L_1) and in the off-channel (L_2) at the stage just before the gate. I call the smaller input the net arousal level I; that is, $I = \min(L_1, L_2)$, and I call the difference between the two inputs the net phasic input J; that is, $J = |L_1 - L_2|$. A nonspecific arousal increment increases I but leaves J alone. A specific competing input increases I and decreases J in such a way that $I + J$ remains approximately constant until the competing input wins (Appendix).

This distinction can be tested by doing studies in which the crossover point from negative to positive net incentive is studied as both the negative and positive inputs are parametrically varied. No such crossover can exist in response to parametric increments in nonspecific arousal.

The next few sections use the inverted U in gated dipole dynamics to suggest how some normal and abnormal motivational cycles work. I present these results not only to explain some paradoxical phenomena for their own sake but also for two other reasons: to sharpen conceptions of how motivational cycles can modulate cognitive processing by altering the incentive motivational signals to sensory and cognitive representations through time and to illustrate how gated dipoles with no endogenous oscillatory properties can be made to persistently oscillate when their outputs generate suitable feedback inputs. The frequency of these rhythms can be made as fast or as slow as we please by changing network parameters. The motivational cycles, operating on a slow time scale, are better studied at the present time, so they will be cited for illustrative purposes. These motivational examples may shed light on faster cognitive rhythms that are functionally homologous. The reader who wishes to focus primarily on cognitive processes can proceed directly to Section 51.

47. THE HYPERPHAGIC SYNDROME AND THE MYTHICAL SET-POINT

I will now explain hypothalamic hyperphagia as an instance of underaroused depression and will argue that the notion of set-point has confused more than clarified the explanation of this syndrome. Since animals with lesions in the ventromedial hypothalamus (VMH) eat voraciously until they become obese, it has been claimed that VMH lesions increase the animals' set-point for body weight (Woods *et al.*, 1974). Since animals with lesions in the lateral hypothalamus (LH) become aphagic, it has been claimed that LH lesions decrease the animals' set-point for body weight (Keesey *et al.*, 1976). Both sets of authors also suggest that, once a weight set-point is determined, the animal eats so as to reduce the error signal that compares its present weight with the weight set-point. Both sets of authors identify an observable behavioral property with an unobservable neural mechanism.

I will argue that, if anything, VMH lesions decrease the animals' setpoint, a viewpoint that is closer in spirit to the Hirsch (1972) model for feeding behavior. I will go further by arguing that the very notion of set-point is inappropriate because it does not illuminate the adaptive design that is disrupted by VMH lesions. Instead, I will indicate how suitable VMH lesions cause the animal to become motivationally underaroused, which is the sense in which the animals' set-point is reduced.

The behavioral properties to be explained include the following ones. VMH lesioned animals (*e.g.*, rats) eat voraciously (hyperphagia) until they become obese. Initially, they seem to have a higher hunger drive (Kent and Peters, 1973; Singh, 1973; Wampler, 1973), which manifests itself in an extended bout of eating followed by discrete meals at a reduced intermeal interval (Balagura, 1972). After the animals become obese, they maintain their higher weight against environmental perturbations (Hoebel and Teitelbaum, 1966; Olds, 1977; Teitelbaum, 1955), a fact which can sorely tempt one to assume that the brain contains a set-point that is increased by VMH lesions.

The behavior of lesioned animals before they become obese can differ notably from their behavior after they become obese. Both dynamic (nonobese) hyperphagics and normal animals eat appreciable amounts of a diet that has been adulterated by kaolin, cellulose, or quinine sulphate (Kennedy, 1953; Stominger, Brobeck and Cort, 1953; Teitelbaum, 1955). Obese hyperphagics reject diets at concentrations of adulterants that are insufficient to disturb feeding by normal animals. By contrast, obese hyperphagics markedly increase their intake of food to which 50% dextrose has been added, whereas normal animals decrease their intake of this diet, since sugar is a more concentrated source of calories than the standard diet (Teitelbaum, 1955).

These data are paradoxical because of the following considerations. Let us start with the classical idea that the overeating that results from VMH lesions is a release phenomenon. The simplest version of this idea suggests that animals eat until they reach a new weight, one at which their hunger motivation is again low. By this argument, the exaggerated sensitivity of the obese animals to negative stimulus qualities of adulterated food might be interpreted as a consequence of low hunger motivation due to prior overeating. This viewpoint suggests that the animal is insufficiently interested in unpleasant food to bother eating it. Such an argument fails to explain why the obese animal overeats in response to positive stimulus qualities of food, as when dextrose is added to its diet. Why doesn't the hypothetical reduction in hunger motivation reduce the animal's interest in all foods? Instead, the obese animal is hypersensitive to both the positive and the negative stimulus qualities of food. Teitelbaum (1955, p. 160) noted these "changes in the reactivity of hyperphagic rats to the stimulus aspects of the diet" and realized that "some change in the internal

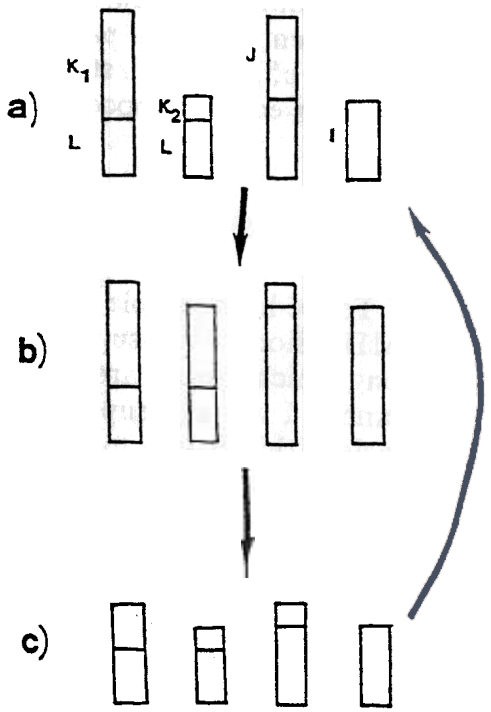


FIGURE 18. A normal gated dipole feeding cycle. (a) A large hunger input, K_1, and small satiety input, K_2, trigger eating by keeping I moderate and J large. (b) Fast growth of the satiety input shuts off eating by increasing I and decreasing J. (c) Digestion decreases I while keeping J small. Then K_1 increases and the cycle begins again.

environment operates in combination with the change in reactivity to the stimulus provided by food." How these changes occur in the animals' sensitivity to external stimuli has not been adequately explained in the past twenty-five years. One reason for this gap is that the phenomena involve "nonhomeostatic" mechanisms that are generally not well understood. A related reason is that not all VMH lesions produce finickiness to stimulus qualities, even if they do produce obesity (Graff and Stellar, 1962; Hoebel, 1976). Thus, to understand the VMH syndrome, one needs a sufficiently precise theory about the interaction between internal and external environmental factors to distinguish how different lesions can differentially induce obesity or finickiness.

My explanation of the hyperphagic syndrome will agree with the classical idea that the hyperphagia is a release phenomenon and will also agree that the obese animal has low hunger motivation, but in a sense that must be carefully defined. I will suggest that certain lesions cause the animal to become motivationally underaroused and that, whenever an animal becomes underaroused in this fashion, it automatically becomes

hypersensitive to the conditioned reinforcer properties of relevant stimuli. Indeed, VMH-lesioned animals can become generally irritable and excessively reactive to all stimuli (Paxinos and Bindra, 1973; Wheatley, 1944).

48. HYPOTHALAMIC STIMULATION AND REBOUND EATING

Before using a gated dipole model to explain hyperphagic data, I will mention some classical data about eating that illustrate how behavioral tests can lead to significant pharmacological inferences if we possess a good conceptual bridge between the two levels. These data support the hypothesis that a gated dipole helps control eating behavior.

The existence of slow transmitter gates between competitive pathways involving lateral hypothalamic and ventromedial hypothalamic sites is suggested by behavioral antagonistic rebound effects. Such effects can occur after hypothalamic stimulation terminates (Grastyan, 1968; Olds *et al.*, 1971; Wyricka and Dobrzecka, 1960). For example, during stimulation of the anterior part of the ventromedial hypothalamus and the adjacent posterior part of the anterior nucleus, hungry animals stop eating, yet, following offset of such stimulation, satiated animals start eating. Is the onset of eating accompanied by a hippocampal P300?

49. A NORMAL VS. HYPERPHAGIC FEEDING CYCLE

To see how a dipole can explain the obesity and finickiness that occur after certain VMH lesions, I will suppose that the lesion partially eliminates the cells or pathways along which the net arousal input and the satiety drive input are delivered to the off channel. I will also suppose that the hunger dipole competes with other motivational dipoles (thirst, sex) to decide which dipole will win the motivational competition. Suppose that, on average, positive incentive motivational output at least T in size is needed to win this competition to induce eating.

FIGURE 18 summarizes the idealized changes through time in hunger and satiety inputs in a normal dipole. In FIGURE 18a, the nonspecific arousal level, L, plus a small satiety input, K_2, equal the net arousal level, I, just before a meal. The net arousal level, I, then falls in the range of normal dipole sensitivity. The net phasic input, J, is large because it is the difference of a large hunger input, K_1, and a small satiety input, K_2. Since I is of normal size, then, as J increases, the incentive motivational output in the on-channel eventually exceeds the level, T, that is needed to win the motivational competition. Eating then begins. As eating proceeds, a fast satiety signal due to gastric distention causes an increase in K_2 before K_1 can significantly change. The input, K_1, will change later as the food is digested. As a result of the change in K_2, I increases while J decreases to keep $I + J$ approximately constant. At this point, one might wonder whether eating will cease as soon as T is no longer exceeded. This is not generally true, because the feedback loops in the motivational dipoles possess hysteresis properties that can keep eating active until J becomes quite small, as in FIGURE 18b. All we need to know now is that K_2 increases quickly before K_1 can decrease, so that I becomes large and J becomes small.

How large and how small? This depends on the value of T that is needed to elicit eating and, thus, on the amount of competition from other motivated behaviors that can be elicited by the behavioral situation. If T is very large (high competition), then eating can cause the dipole to approach an overaroused condition (high I due to high

K_2). When this happens, the dipole becomes insensitive to further increments in K_2 due to the inverted U property. By contrast, if T is smaller, then I will not grow as much, so that the dipole remains more sensitive to novel appetitive or aversive food cues. All in all, I increases and J decreases until eating stops. As digestion proceeds, both K_1 (hunger) and K_2 (satiety) signals decrease (FIGURE 18c). As a result, I decreases and J remains small. The dipole's sensitivity increases, but it cannot win the motivational competition because J remains small. Finally, the hunger input, K_1, begins to grow again until eating is once more elicited. Note that, in this analysis, the total duration of a meal depends both on the I value and the J value that obtains when the meal begins.

FIGURE 19 idealizes the drive input changes that can occur when the K_2 pathway is partially destroyed. The main change is a reduction in the K_2 input and the arousal input that perturb the off-channel. Consequently, the net arousal level, I, is reduced and the dipole is underaroused. The K_1 hunger input that is needed to achieve an on output that exceeds T is thus smaller than the K_1 level needed to exceed T in the normal

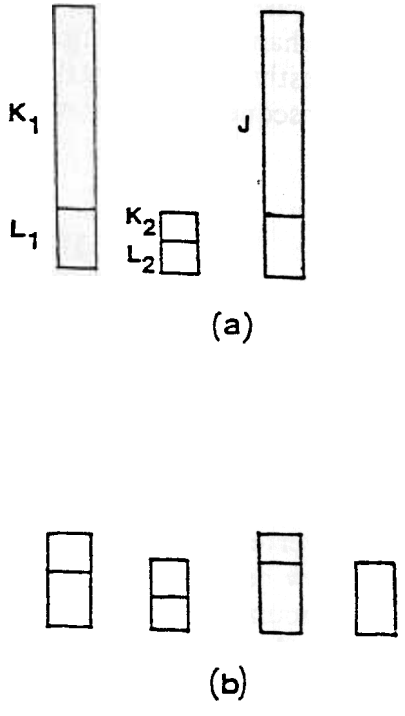


FIGURE 19. A hyperphagic gated dipole feeding cycle. (a) Damage to the off-channel can make K_2 and L small, thereby decreasing I and increasing J. Voracious eating is thereby triggered. (b) Eating is terminated by a reduction in K_1, not in K_2. Thereafter, smaller than normal increments in K_1 can trigger eating since L, hence I, remains small.

dipole (FIGURES 18 and 19a). Two properties are thus achieved. The dipole is hyperactive because it is underaroused and the hunger level that elicits eating can be less than normal, although it will be of normal or greater than normal size right after the operation that causes the lesion. The reduction in the threshold K_1 size needed to elicit eating helps to explain why hyperphagic animals can seem poorly motivated.

Once eating begins, it can be rapidly stopped only if the K_2 input can grow enough to increase I and decrease J until the dipole shuts off. However, if the K_2 pathway is seriously damaged, then the K_2 input cannot grow significantly in response to rapid gastric reactions to food. When this is true, eating can persist until the K_1 input decreases as a result of slower digestive effects (FIGURE 19b). This is not the same mechanism that terminates a meal in the normal dipole. An animal controlled by an underaroused dipole will thus become obese by eating persistently right after its lesion in the off channel.

After the animal becomes obese, the situation changes. The input, K_1, never gets a

chance to grow to large values because of the hypersensitivity of the underaroused dipole to K_1 increments. These smaller K_1 increments trigger eating; eating persists until the K_1 increments are withdrawn. In this way, the animal can defend its new weight against environmental fluctuations even though there exists no "set-point" in the dipole. Note also that the intermeal interval can be reduced because a smaller-than-normal increment in K_1 is needed to initiate the next meal.

How can one achieve a syndrome wherein obesity occurs without finickiness? This is formally easy to do. The similarity of the lesion that produces obesity-without-finickiness to the lesion that produces obesity-with-finickiness helps to explain why this syndrome has caused so much confusion. FIGURE 20b illustrates this formal lesion. It destroys cells and/or pathways in the off channel after the stage at which the signals are gated, by contrast with the previous lesion, which destroyed cells and/or pathways in the off-channel before the stage at which signals are gated.

Because the lesion occurs after the gating stage, all the specific and nonspecific inputs are of normal size. The dipole is not underaroused; hence, it is not hyperactive. However, the competition from the off-channel to the on-channel is eliminated, as are the negative incentive motivational outputs from the off-channel. Even though K_2 increases rapidly as eating occurs, the competitive signal due to K_2 is not felt by the on-channel. Once again, the on-channel is shut off by the slow decrease of K_1 due to digestive factors rather than by the rapid increase of K_2 due to gastric distention. Consequently, the animal eats abnormally large meals and becomes obese.

50. SOME OTHER DRUG-INDUCED INVERTED U'S AND HYPOTHALAMIC-HIPPOCAMPAL INTERACTIONS

The underaroused depressive syndrome helps to explain how dopamine damage can yield insensitivity to weak sensory stimuli but intolerance of intense sensory stimuli (Stricker and Zigmond, 1976).

The fact that D-amphetamine sulfate activates feeding in an anorectic cat at the same dose (2 mg kg^{-1}) that totally inhibits feeding in a normal cat can be viewed as an inverted U effect (Wolgin *et al.*, 1976), as can the fact that amphetamine augments slow behavior and depresses fast behavior (Dews, 1958). Also in normal cats, smaller amounts of norepinephrine can have effects opposite to those of larger amounts (Leibowitz, 1974).

A more speculative inverted U effect concerns the modulatory effect of septal input on the firing rate of hypothalamic cells (Mogensen, 1976). If a hypothalamic neuron is firing slowly, then septal input speeds up its firing rate (ascending end of inverted U), whereas, if the hypothalamic neuron is firing rapidly, then septal input slows down its firing rate (descending end of inverted U). The possible existence of competing pathways in septal-hypothalamic interactions is supported by the observation that, in unanesthetized rats, some cells increase firing rates and other cells decrease firing rates during drinking (Bridge, 1976). Also, the mediodorsal septum tends to excite hypothalamic neurons *via* the fornix, whereas the bed nucleus of the stria terminalis tends to inhibit hypothalamic neurons and wide regions of septum contribute both to the fornix and to the stria terminalis (Mogensen, 1976). Edinger and Siegel (1976) report competitive interactions between the medial and lateral septum and relate this competitive geometry to the effects of afferents from the dorsal and ventral hippocampus.

The hippocampus is interpreted as the final common path of the model's drive and reinforcer interactions in Sections 51–53. I postulate that it emits several types of

output after a winning drive representation is chosen. The medial forebrain bundle (Haymaker *et al.*, 1969; MacLean, 1970) is suggested to be the anatomical analogue of the formal feedback loops that run through the model's circuits (Grossberg, 1972b, 1975).

We are now ready to begin our study of how cognitive and motivational networks reciprocally interact to control the shifting focus of attention through time.

51. ADAPTIVE RESONANCE BETWEEN DIPOLE FIELDS

FIGURE 21 depicts the minimal network that I need to mechanistically explain attentional data. FIGURE 21 describes a feedback module wherein sensory and drive

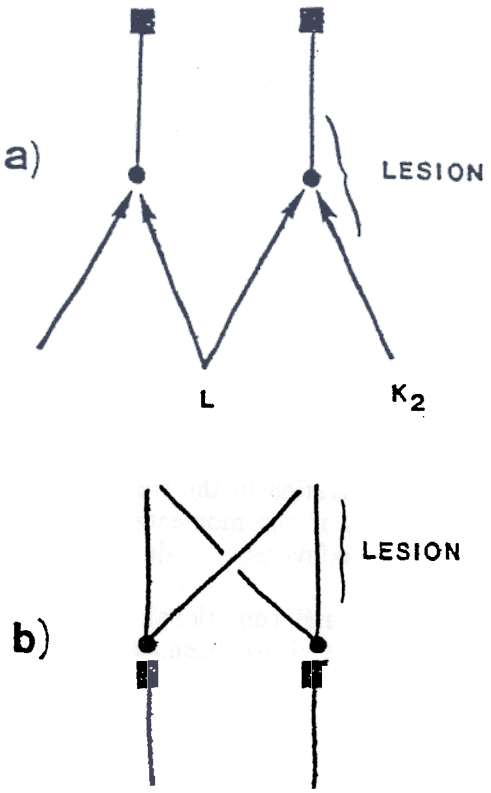


FIGURE 20. Lesions that influence a network's obesity and finickiness. The lesion in (a) occurs before the transmitter gating stage. It thereby lowers the network's net arousal level and causes a full-blown underaroused syndrome. The lesion in (b), by contrast, does not alter the net arousal level. Rather, it merely weakens the inhibitory effects of satiety inputs that occur after the transmitter gating stage.

representations send signals to each other *via* nonspecific excitatory conditionable pathways. These representations are organized into dipole fields. Each dipole field is capable of STM contrast enhancement, normalization, hysteresis, and rebound. The interfield conditionable pathways send branches to both the on-cells and the off-cells of the dipole fields.

The conditionable pathways from sensory-to-drive representations encode the conditioned reinforcer properties of external cues. The conditionable pathways from drive-to-sensory representations encode the incentive motivational properties of internal drives. Adaptive resonance occurs within this network when the reinforcing properties of active external cues sufficiently match the motivational properties of active internal drives to lock STM into a global interpretation of the data.

In the theory I developed in Grossberg (1971, 1972a,b; 1975), the final processing stage in the external cue representations is assumed to be cortical and the final

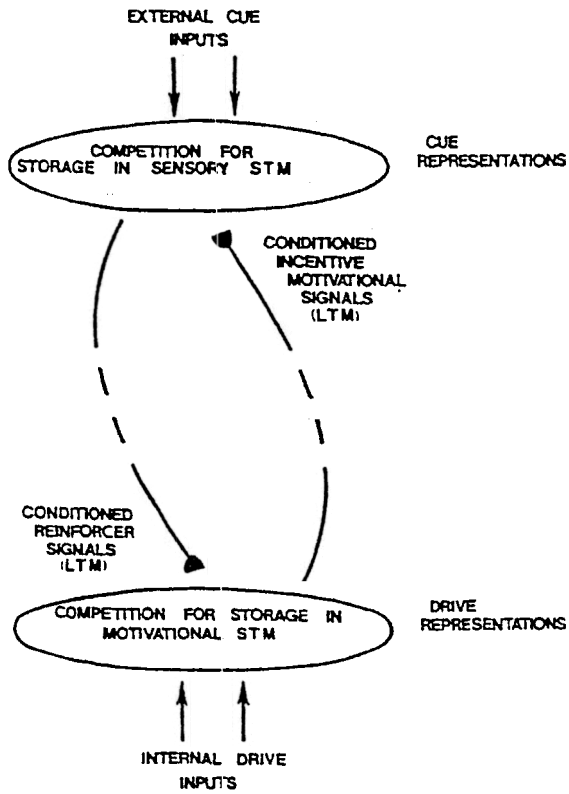


FIGURE 21. Adaptive resonance between dipole fields. When external cues excite the STM traces of their internal representations, these internal representations elicit signals that are distributed nonspecifically across the drive representations. During conditioning, the pattern of reinforcing and drive inputs to the drive representations can alter the LTM traces within certain of these signal pathways, as in FIGURE 16. The corresponding external cues thus acquire conditioned reinforcer properties.

On recall trials, the conditioned reinforcer signals from external cues combine with internal drive inputs at the drive representations to determine which drive representations will fire. Output from the drive representations plays the role of incentive motivation in the theory. Incentive motivation is released from a given drive representation only if the momentary balance of conditioned reinforcer signals plus internal drive inputs competes favorably against these factors within the other drive representations.

The incentive motivational pathways are also nonspecific and conditionable. Each drive representation can become conditioned to the class of cues to which it has been associated in the past. Activating a drive representation creates an STM bias that favors the STM storage of motivationally compatible cues. Those external cue representations which receive the most vigorous combination of incentive motivational signals plus external cue inputs can compete most favorably for the limited capacity (normalized) STM activity and thereby be best attended. FIGURE 23 describes this process in greater detail.

processing stage in the drive representations is assumed to be hippocampal. Gabriel *et al.* (1980) summarized recent data that support a qualitatively similar conclusion. They write "the hippocampal formation is a region critical for encoding or 'modelling' of stimulus-reinforcement contingencies" (p. 189). They note that the hippocampus is reciprocally connected with cingulate cortex and with the anteroventral nucleus of the thalamus and summarize data suggesting that cortical "codes inappropriate to the stimulus item being presented would create mismatch with the hippocampal model, thereby eliciting code-suppression in cortex and thalamus. Thus, no response would occur" (p. 216).

52. A MOTIVATIONAL DIPOLE FIELD: DRIVE-REINFORCER MATCHING AND MOTIVATIONAL COMPETITION

FIGURE 22 depicts an anatomy that possesses the minimal drive representation properties that I will need. In this anatomy, each motivational channel possesses a positive feedback loop that runs through a gated dipole. These positive feedback loops are the on centers of the competitive feedback network that joins together the motivational channels. The competitive feedback network provides a matching interface (Section 14) that runs across the motivational channels. At this particular matching interface, spatial patterns of (conditioned) reinforcer signals are matched with spatial patterns of drive signals. Only a sufficiently good match can trigger sustained polyvalent cell firing. If this network's QT is tuned so high that only a single winning channel can reverberate in STM, then sharp motivational switching will occur. Such a setting of the QT defines the channels as motivationally incompatible. A lower setting of the QT permits compatible combinations of drive representations to be synergistically activated. Possible QT settings depend on the choice of numerical network parameters and can vary across species and individuals without changing the underlying design principle.

53. THETA, CNV, AND MOTOR POTENTIAL CORRELATES OF A HIPPOCAMPAL MODEL

FIGURE 22 summarizes some of the main ideas in the theory of hypothalamic-hippocampal interactions developed in Grossberg (1971; 1972a,b; 1975). The conditioned reinforcer learning at polyvalent cells helps to explain the conditioning at hippocampal pyramidal cells that Berger and Thompson (1978) report in their studies of the rabbit nictitating membrane response. The STM resonance within gated dipole feedback loops that accompanies conditioned reinforcer learning helps rationalize the theta rhythm that accompanies hippocampal conditioning.

The theory postulates that incentive motivational output from polyvalent cells branches into at least two functionally distinct pathways. One branch controls attentional processing by returning conditionable feedback to external cue representations (Section 29). All these feedback pathways are excitatory to abet STM storage of those representations which are postsynaptic to pathways whose LTM traces have grown due to favorable conditioning contingencies. My theory accepts the idea that it is adaptive to carefully attend to fearful cues, if only to better escape from them.

I believe that this positive conditionable incentive motivational pathway is probed by the Bower (1981) experiments on the effects of mood on memory. In particular, Bower does not find that sad-congruent lists are learned any worse than happy-congruent lists. He also finds that incongruent moods can interfere with recall, which can be explained by the competitive interactions between drive representations and cue representations. I believe that this incentive motivational branch is a formal analogue of the pathways *in vivo* that activate a motivational component of the CNV. If this is so, then learning of mood-to-memory associations in Bower's paradigm may yield larger CNVs during mood-congruent recall than in Bower's other recall conditions.

The outputs from the other incentive motivational branch are not all excitatory. These outputs are assumed to preserve their motivational sign, whether positive or negative, and to be used as inputs to a spatial map. It is hypothesized that the spatial distribution of active motivational signs determines the momentary approach or avoidance direction read out from the spatial map. O'Keefe and Nadel (1978) clarify

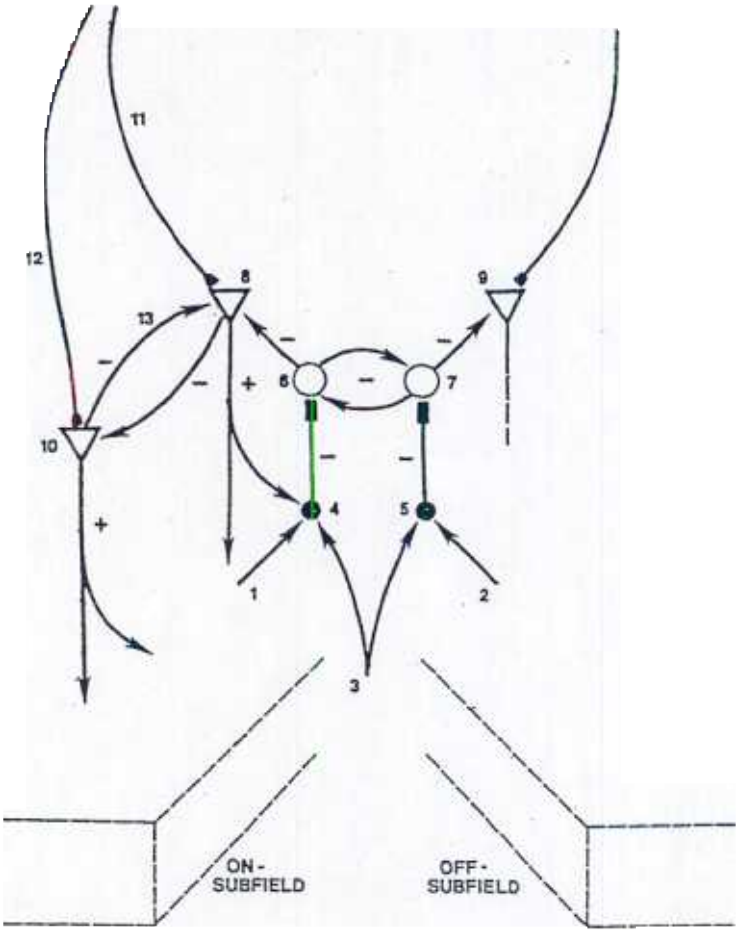


FIGURE 22. A motivational dipole field. Pathways 1 and 2 carry specific, but complementary, drive inputs (*e.g.*, hunger *versus* satiety) to a single dipole. Pathways labeled 3 carry nonspecific arousal to this dipole. Cells 4 and 5 add these inputs and thereupon inhibit the tonically active cells 6 and 7. (Tonic cells have open symbols; phasic cells have closed symbols.) Pathways 4 → 6 and 5 → 7 contain slow transmitter gates (square synapses), assumed to be catecholaminergic. If input 1 exceeds input 2, then the transmitter in pathway 4 → 6 is depleted more than the transmitter in pathway 5 → 7, thereupon calibrating the dipole for a possible antagonistic rebound later on.

The tonic cells 6 and 7 inhibit each other equally until input 1 exceeds input 2. Then cell 6 is inhibited more than cell 7. This imbalance disinhibits tonic cell 8 and further inhibits tonic cell 9. Both cells 8 and 9 are polyvalent, meaning that all their excitatory inputs must be active for these cells to vigorously fire. (Triangles denote polyvalence.) The polyvalent cells are assumed to be pyramidal cells. Because cells 8 and 9 are polyvalent, a larger input to cell 1 than to cell 2 cannot fire these cells. However, such an imbalance can prevent cell 9 from firing.

To see how cell 8 can fire, we consider the polyvalent cells 8 and 10 of two different motivational channels. Cells 8 and 10 compete *via* the inhibitory (interneuronal) pathways 13. Polyvalent cells 8 and 10 also receive inputs from external cue representations *via* conditionable pathways 11 and 12, respectively, whose LTM traces (within the filled hemicircles abutting 8 and 10) encode conditioned reinforcer properties of their respective external cues. These LTM traces are assumed to be cholinergic.

The conditioned reinforcer inputs combine with drive and arousal inputs at their respective polyvalent cells, which begin to fire if their thresholds are exceeded. The polyvalent cells thereupon compete among themselves *via* the "intrinsic" feedback inhibitory pathways 13, as they simultaneously try to excite themselves *via* positive feedback pathways such as 8 → 4 → 6 → 8.

If, for example, cell 8 wins this competition, then the transmitter gate in 4 → 6 is depleted as the suprathreshold reverberation bursting through cell 8 *via* pathway 8 → 4 → 6 → 8 drives LTM changes in pathway 11. The reverberation thus induces conditioned reinforcer changes even as it prepares the network for motivational reset by rapid offset of 11 or a rapid increment in 3.

this aspect of hippocampal functioning. Although the O'Keefe and Nadel concept of an absolute map of an animal's position in space can be criticized on philosophical no less than on scientific grounds, a weaker notion should, I believe, receive further study. This is the concept of a bilaterally organized spatial map in which the asymmetry of excitatory and inhibitory incentive signals with respect to the map's body axis controls the net approach *versus* avoidance direction of motion.

The existence of hippocampal place and misplace cells (O'Keefe and Nadel, 1978) suggests the possibility that a gated dipole structure exists within this hippocampal spatial map; in particular, that the spatial map is a specialized dipole field whose dipoles may be organized symmetrically with respect to the map's body axis. If such an organization exists, then an unexpected event can reset the direction of motion to one complementary to the direction pursued before the unexpected event occurred. If, by contrast, the dipoles just rebound the agonist-antagonistic patterns of active muscle commands, then the unexpected event will cause coordinated motor braking without a change in direction.

The hypothesized existence of both a CNV branch and a spatial mapping branch of the incentive motivational computation helps rationalize why CNV and motor ERPs are often so closely related (Tecce, 1972). A theory capable of sharply distinguishing CNV from motor ERPs would need to incorporate better how bilaterally organized signed spatial maps are organized and how their commands are read out as motor behavior. The final sections of this article will consider how the hypothesized CNV branch of the incentive motivational output helps explain conditioning and attentional data. To show how I think this CNV branch influences attentional processing, I shall first need to describe the processing at cue representations in greater detail.

54. A SENSORY DIPOLE FIELD: THE SYNCHRONIZATION PROBLEM AND DC POTENTIAL SHIFTS

FIGURE 23 depicts the minimal anatomy that I will need to join together external cue representations. This dipole field has more structure than that in FIGURE 22 because it solves a specialized design problem, which I call the synchronization problem of classical conditioning. The synchronization problem recognizes that, without specialized network buffers, Pavlovian associations could rapidly extinguish whenever a CS and US were presented with different interstimulus delays on successive learning trials. The synchronization problem was solved in Grossberg (1971) and provided the impetus for my later work on reinforcement and motivation.

I hope that the reader wants to ask, What does a lack of synchronization between CS and US delays across Pavlovian trials have to do with reinforcement and motivation? The answer is found in the minimal network that solves the synchronization problem. In this network, drives and conditioned reinforcers interact to control incentive motivational feedback that is necessary to fire polyvalent cortical cells. This solution to the synchronization problem clarifies how Pavlovian and instrumental paradigms can engage common network mechanisms, a fact that has inspired various two-factor learning theories (Dunham, 1971) and complicated efforts to dissociate instrumental from Pavlovian effects in biofeedback paradigms.

For my present purposes, I need to emphasize one difference between FIGURES 22 and 23. The anatomy in FIGURE 23 separates the firing of polyvalent cells from the STM reverberation through gated dipoles. Due to this property, a sensory representation can reverberate in STM and, thereby, deliver signals to a polyvalent cell, or cells, without firing those cells. A polyvalent cell in FIGURE 23 can fire only if it

simultaneously receives STM signals from an external cue representation and incentive motivational signals from a drive representation. This property is analogous to John's (1966, 1967) reports that certain polyvalent cortical cells involved in cortical conditioning can fire only in response to a sum of CS and US signals. The property is also analogous to the effects of anodal dc potential shifts on cortical conditioning (Morrell, 1961; Rusinov, 1953). In my theory, the anodal dc shift replaces the requirement of an incentive motivational signal to fire polyvalent cortical output cells.

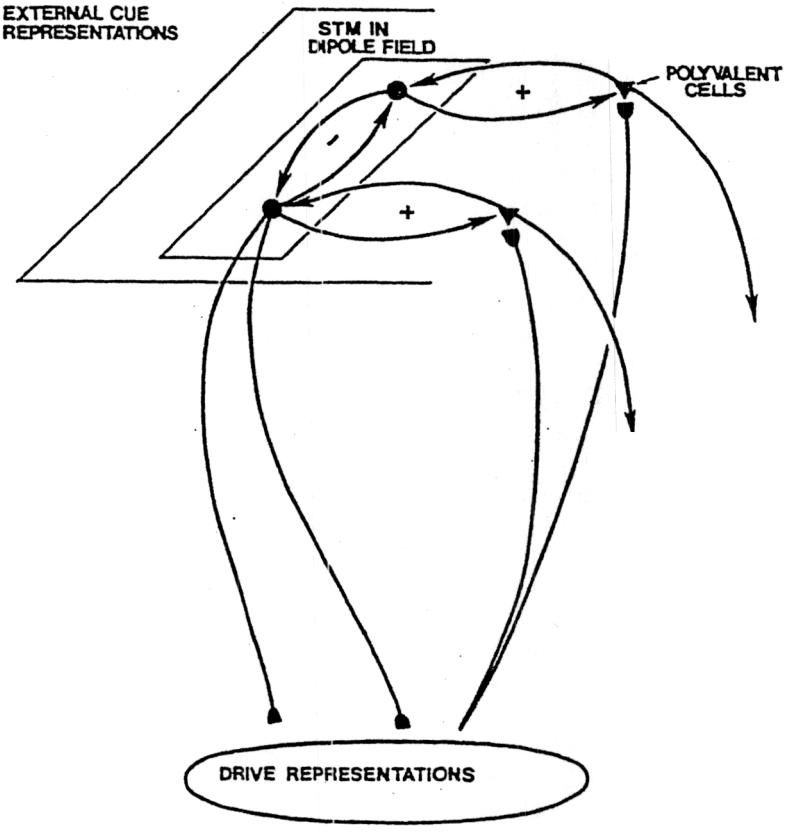


FIGURE 23. Specific STM signals to the polyvalent cells are insufficient to fire these cells. Sufficiently large incentive motivational signals must simultaneously converge upon the polyvalent cells to fire them. When a polyvalent cell fires, it delivers positive feedback signals to the cells that supply it with specific STM signals. This positive feedback selectively augments the STM activities of these cells, which thereupon more strongly inhibit their competitors for normalized total STM activity.

55. SECONDARY CONDITIONING

The functional separation of STM reverberation and polyvalent cell firing implies the following description of how a CS acquires US properties to control observable behavior. Let a CS activate the population v_{11}, which thereupon begins to reverberate in STM. Then v_{11} sends specific signals to the polyvalent cell population v_{12} (among others) and nonspecific signals to the drive representations. Nothing else happens until a US arrives at population v_{21}. This is because v_{12} can fire only if it receives an input from v_{11} and an incentive motivational input from a drive representation, but the signal from v_{11} to the drive representations is initially too small to fire them. When the US

perturbs v_{21}, v_{21} sends signals to the polyvalent cells v_{22} and to the drive representations. These latter signals can fire a certain drive representation, if its drive input is large enough, because the cue firing v_{21} is a US for that drive representation, which I will henceforth denote by D_2. When D_2 fires, it releases nonspecific incentive motivational signals to all the external cue representations. Now five things happen.

First, since v_{11} and D_2 are both active, the LTM traces in the pathway from v_{11} to D_2 are strengthened. When these LTM traces get strong enough, the CS alone will be able to fire D_2. Second, the nonspecific incentive motivational signal from D_2 combines with the US-derived signal from v_{21} at v_{22}, thereby firing polyvalent cell signals from v_{22} that read out a UR pattern. Third, because the incentive motivational signal is nonspecific, it also combines with the CS-derived signal from v_{11} at v_{12}, thereby firing the polyvalent cells v_{12}. Fourth, since D_2 and v_{12} are both active, the LTM traces in the pathway from D_2 and v_{12} are strengthened. Fifth, the polyvalent cells v_{12} fire sampling signals to the cells at which the UR pattern is being read out. These signals encode (a fractional component of) the UR in the LTM traces of this pathway. The encoded pattern will henceforth be read out as a CR pattern. The CS thus acquires US properties by learning to control conditioned reinforcer, incentive motivational, and habit strength LTM traces.

This network provides a simple answer to the synchronization question: How does the US turn on the CS with just the right time lag to sample read out of the UR pattern? The same incentive motivational burst that allows v_{22} to read out the UR also allows v_{12} to emit sampling signals that read in the CR.

In Grossberg (1982c), I suggest two interpretations of the cue representation anatomy in FIGURE 23, one in terms of intracortical interactions and the other in terms of thalamocortical interactions. In both interpretations, the pyramidal cells are assumed to be cortical, but the differences between the two interpretations lead to testable predictions.

56. VALENSTEIN EFFECT: NONSPECIFIC DRIVE REPRESENTATIONS OR NONSPECIFIC CONDITIONED REINFORCERS AND CONDITIONED INCENTIVES?

A controversy that bears on the existence of nonspecific conditionable projection systems between cognitive and motivational processors is the Valenstein effect (Valenstein *et al.*, 1969, 1970). The Valenstein effect is often cited as evidence against the existence of anatomically separate motivational systems in the hypothalamus, since the behavior elicited in response to hypothalamic stimulation can gradually change without any alteration in the stimulation parameters. For example, if the food which a rat originally ate in response to stimulation is removed from its cage, then alternative behaviors such as drinking or gnawing can gradually emerge. The specificity controversy has been intelligently debated by marshalling a wide variety of experimental results (Teitelbaum, 1973; Valenstein, 1973), but I believe that the controversy is a misplaced one because the Valenstein effect can occur whether or not hypothalamic motivational sites are anatomically disjoint. The controversy has focused on the wrong issue. Whether or not the motivational sites are disjoint, external cue representations send pathways nonspecifically across several motivational systems. Conditioning of these pathways is one step whereby cues acquire conditioned reinforcer properties. Moreover, feedback pathways from each motivational system reach nonspecifically across many external cue representations. Conditioning of these feedback pathways is

one step whereby cues acquire incentive motivational properties. These nonspecific conditionable interactions of external cue representations with motivational representations can give rise to the Valenstein effect even if the motivational representations are entirely disjoint (Grossberg, 1971, 1972a,b). In other words, the controversy has been elaborated within the homeostatic viewpoint, although the phenomenon can be explained by nonhomeostatic mechanisms.

Both Teitelbaum (1973) and Valenstein (1973) recognize that there must exist mechanisms that decide which motivated behavior will appear at any time. Teitelbaum argues by analogy with von Bekesy's model of cochlear "funneling," and Valenstein calls a similar process "channeling." Unfortunately, these personal languages are inadequate as tools to dissect the functional components in the data.

With the above results as background, we can now analyze some interactions between cognitive and motivational networks during conditioning and attention shifts, as well as some of their predicted ERP substrates.

57. DISCRIMINATION AND OVERSHADOWING DUE TO CONDITIONED INCENTIVE MOTIVATIONAL FEEDBACK: CNV CORRELATES

In his book *Conditioned Reflexes*, Pavlov (1927) gave a brilliant account of how individual cues could be extinguished while the same cues presented as a composite could elicit conditioned responses, and how more intense or more salient cues in a composite could progressively overshadow the other cues in the composite as conditioning proceeded. The four experiments reviewed in FIGURE 1 are a version of these classical experiments. The following overshadowing and discrimination learning experiments will be used to illustrate both how conditioned incentive motivation helps determine which cues will be overshadowed and how CNV and P300 measures might correlate with these conditioned changes.

Newman and Baron (1965) reinforced pigeons who pecked a vertical white line on a green key (the S^+) but not a green key alone (the S^-). They tested cue discrimination by tilting the line at various orientations during recall trials. A generalization gradient of pecking was found, indicating that the vertical line was discriminated. By contrast, no generalization gradient was found if the S^- on learning trials was a red key or if the S^- was a vertical white line on a red key.

Newman and Benefeld (Honig, 1970) also used a vertical white line on a green key as S^+ and a green key as S^-, but tested and found generalization of the line orientation on a black key. They also tested generalization on a black key following training without a green S^- and again found a generalization gradient, in contrast to the case where testing used a green key. They interpreted this effect as "cue utilization during testing rather than cue selection during learning." This interpretation does not explain how the orientation cue could be learned on training trials if it was not discriminated using a green background on test trials, yet how the orientation cue could be discriminated using a black background on test trials if it was not learned on training trials. The reader might already sense that STM normalization will somehow come to the rescue.

My explanation of these data begins by noting that color cues are prepotent over orientation cues in the pigeon, other things being equal. Consequently, when a vertical white line on a green background is first presented, the green representations will partially overshadow the orientation representations. (I will talk about "green" and "orientation" representations as a shorthand for more sophisticated coding notions that we do not need here.) Grossberg (1978a, Sections 39–40; 1983, Sections 33–44)

describes some factors that control how prepotent representations can mask the STM activities of other representations due to competitive feedback interactions.

When the line-on-green cues are first presented, they will enjoy an additional advantage in their STM storage. Their unexpectedness in the context of the experiment's situational cues will strengthen the STM activities of the line-on-green cues as the STM activities of the situational cue representations are rebounded. These rebounds should elicit a P300.

After the line-on-green representations are initially stored in STM, the green cues can increase their relative STM advantage as they acquire conditioned reinforcer and conditioned incentive motivational properties. FIGURE 23 shows that, when an external cue representation can activate its polyvalent cells using conditioned reinforcer-incentive feedback, polyvalent cell signals can, in turn, feed back to the cue representations to further enhance their own STM activity. Due to STM normalization among the cue representations, the differential enhancement of some representations acts to the detriment of other representations.

The orientation representations can also acquire conditioned reinforcer and incentive motivational properties as long as their STM activities exceed the network's QT. Their learning rates will be slower than those of the green representations, since their sampling signals in the conditionable pathways are smaller, due to their smaller STM activities. Hence, their conditioned pathways will remain weaker than those of the green representations. As conditioning continues, the orientation representations might be entirely quenched if the conditioned advantage of the color cues becomes sufficiently great to drive orientational STM activities below the QT by competitive feedback across the cue representations.

The unexpected nonoccurrence of reward in response to the green key causes an antagonistic rebound that differentially excites the off-cells of the previously most active STM representations. The active incentive motivational pathways thereupon sample a large off-response in the green representational dipoles. As this experimental contingency recurs on several trials, the net incentive motivational feedback to the green dipoles becomes progressively smaller due to dipole competition between the conditioned on-cell and off-cell pathways to these dipoles.

Even zero net incentive feedback may not be small enough to extinguish the green representation, however, because of the innate advantage of color over orientation. Negative net incentive feedback may be needed to overcome green's innate competitive advantage. Net negative feedback is needed if net positive conditioned reinforcer-incentive feedback to the orientation representation is not sufficient to offset the innate competitive advantage of the color representation when the latter receives zero net conditioned feedback.

This discussion suggests that, although the CNV is always a negative potential in the large, it may cause off-reactions in the cortical dipoles corresponding to predictively irrelevant but prepotent cues even as it strengthens the on-reactions of cortical dipoles corresponding to predictive but nonprepotent cues. I am not sure how this effect can be measured by ERPs, but it should be greatest when the initial disadvantage of the nonprepotent cue relative to the prepotent cue is as large as possible.

This mechanism easily explains why the white vertical line is discriminated on a black background during test trials even if it is not discriminated on a green background during test trials in an experiment without a green S^- on learning trials. Removing green on test trials eliminates competitive feedback from the color representations to the orientation representations. The STM field is thus renormalized. In the renormalized field, even small conditioned reinforcer-incentive feedback signals can provide the white-vertical-orientation representation with a significant competitive advantage for STM storage.

58. THE PROBLEM OF PERSEVERATING PREPOTENT CUES

The above mechanism shows how conditionable reinforcer-incentive feedback enables representations to overcome innate competitive STM disadvantages. Some further remarks might clarify why the incentive pathway needs to send branches both to the on-cells and to the off-cells of cortical dipoles. The main benefit is that some cues can lose net positive feedback as other cues gain net positive feedback while both sets of cues are conditioned to the same drive representation. This property avoids the following dilemma.

Suppose the rebound that conditions zero net feedback to the green representation occurs among the drive representations rather than among the cue representations. Then rebound activates a negative drive representation and the net conditioned reinforcer feedback (rather than the net incentive feedback) controlled by the green representation becomes small. This mechanism is unstable for the following reason. As soon as the orientation representation takes over in STM, its positive conditioned reinforcer signals activate the positive drive representation. When this drive representation sends conditioned incentive feedback to the cortex, the green representation receives conditioned positive feedback too, since the negative drive representation is momentarily inhibited. Then the green representation can quickly overshadow the orientation representation using its innate competitive advantage. As soon as the green representation is reinstated in STM, its conditioned reinforcer signals lead to read-out of net negative incentive from the competing drive representations. The green representation is consequently shut off, the orientation representation is disinhibited, and the cycle repeats itself.

Any viable alternative to the present network description must also avoid this problem of perseverating prepotent representations. In particular, a more sophisticated coding analysis would replace "green" and "orientation" representations with heterarchical network encodings wherein one representation's prepotence for masking other representations would depend on its heterarchical position as well as peripheral factors (Grossberg, 1978a, 1983).

59. CORTICAL RESET TRIGGERS HIPPOCAMPAL RESET: TWO DISTINCT P300s

At this point, the reader might rightly worry, Haven't you thrown away one good property to salvage another one? Shouldn't there also be a rebound in the drive representation due to the nonoccurrence of expected reward after the green key is presented? After all, this is a frustrating situation in the motivational sense, no less than a situation that needs counterconditioning in the cognitive sense.

I fully agree. First, the rebound among the cortical dipoles is sampled by the LTM traces of the active incentive motivational pathway. Next, offset of the green representations in the cortex shuts off their positive conditioned reinforcer signals to the positive drive representation. Finally, the sudden reduction in conditioned reinforcer input is felt by this drive representation. If the reduction is large enough, STM hysteresis within the positive drive representation is overcome and an antagonistic drive rebound occurs that activates the negative drive representation in its dipole. Any cue that is initially stored in STM at this time can acquire negative conditioned reinforcer properties by sampling this negative drive rebound (Section 31).

The above discussion predicts that the nonoccurrence of expected reward can trigger a cortical P300 by mismatching a learned expectancy. The reset of cortical STM

can thereupon trigger a hippocampal rebound by rapidly withdrawing conditioned reinforcer input. If these P300 predictions hold up, they will clarify that a P300 can be elicited by different operations in different brain regions. They will also refine our understanding of the information processing substrates of overshadowing and discrimination learning by distinguishing rebounds that motivationally extinguish cues due to their cognitive irrelevance, without extinguishing their conditioned reinforcer pathways, from rebounds that directly elicit new conditioned reinforcer learning.

60. P300 SIZE PREDICTS NOTHING ABOUT WHAT IS ENCODED IN LTM

I can now illustrate my contention of Section 6 that a large P300 predicts nothing general about LTM. All it tells us is that an STM reset has occurred. The STM reset event will enable advantageously stored cues to elicit large sampling signals, but what these signals encode in LTM will depend on the entire experimental context.

For example, the unexpected occurrence of a reward in response to the line-on-green cue will elicit a P300. As this P300 shrinks on successive rewarded trials, the line-on-green cue should elicit a growing motivational CNV. By contrast, the unexpected nonoccurrence of a reward in response to green alone will elicit a P300. As this P300 shrinks on successive unrewarded trials, the green cue should elicit a shrinking motivational CNV. A conditioned response is learned in the former case, whereas a conditioned response is extinguished in the latter case. P300 size does not differentiate these opposite outcomes in LTM.

61. THE MACKINTOSH, BYGRAVE AND PICTON EXPERIMENT: A P300 PREDICTION

Since a more complete analysis of the moment-by-moment network processing of the Mackintosh *et al.* (1977) experiment (Section 6) has appeared elsewhere (Grossberg, 1982a), I will only briefly summarize my main hypothesis here and make a P300 prediction. I suggest that the main effect of the second shock on the first compound trial is to make the occurrence of the tone on the second compound trial more unexpected within the context of the experiment's situational cues. This greater unexpectedness elicits a larger P300 and gives the tone greater initial STM activity. The larger STM activity elicits larger sampling signals in the conditioned reinforcer pathways of the tone representation. The larger sampling signal supports faster conditioning of the conditioned reinforcer LTM traces that abut the negative drive representation that is activated by the shock. The tone thus becomes motivationally more fear-inducing due to the cognitive effects of a prior shock.

62. CONCLUDING REMARKS

This article has attempted to show how a properly posed thought experiment about hypothesis testing in a changing environment can suggest new information processing concepts and mechanisms. The physiological realizations of these concepts reveal new cognitive and motivational neural designs that can be tested by a combination of psychological, physiological, pharmacological, and evoked potential techniques.

I have also indicated that surprising mathematical properties of these designs shed

new light on important behavioral syndromes. The explanation of these syndromes does not lie in an ever finer dissection of brain components. Rather, one must study the interactions within neural tissues because these interactions literally create the behavioral properties. In particular, no amount of saying that too little catecholamine correlates with Parkinsonism and juvenile hyperactivity or too much catecholamine correlates with schizophrenia can explain the behavioral properties of underaroused or overaroused depression.

Evoked potential experiments can probe this interactive neural level. They are, therefore, a powerful tool for studying neural designs that purport to clarify the development and stability of cognitive and motivational processes.

APPENDIX
GATED DIPOLES

63. TRANSMITTERS AS GATES

The transmitter model presented here was derived from associative learning postulates in Grossberg (1968, 1969b). The gated dipole model was derived from conditioning postulates in Grossberg (1972b). The transmitter derivation below shows that our transmitter law is the minimal dynamic law for unbiased transmission using a depletable signal (Grossberg, 1980a).

We start by asking the following question: What is the simplest law whereby one nerve cell can send unbiased signals to another nerve cell? The simplest law says that if a signal S passes through a given nerve cell v_1, the signal has a proportional effect

$$T = SB, \tag{4}$$

where $B > 0$, on the next nerve cell v_2. Such a law permits unbiased transmission of signals from one cell to another.

A difficulty occurs, however, if the signal from v_1 to v_2 is due to the release of a chemical $z(t)$ from v_1 that activates v_2. If such a chemical transmitter is persistently released when S is large, what keeps the net signal, T, from getting smaller and smaller as v_1 runs out of transmitter? Some means of replenishing or accumulating the transmitter must exist to counterbalance its depletion due to release from v_1.

Based on this discussion, we can rewrite (4) in the form

$$T = Sz \tag{5}$$

and ask, How can the system keep z replenished so that

$$z(t) \simeq B \tag{6}$$

at all times t? This is a question about the sensitivity of v_2 to signals from v_1, since if z could decrease to very small values, even large signals S would have only a small effect on T.

Equation 5 has the following interpretation. The signal, S, causes the transmitter, z, to be released at a rate $T = Sz$. Whenever two processes, such as S and z, are multiplied, we say that they interact by mass action, or that z gates S. Thus, (5) says

that z gates S to release a net signal T, and (6) says that the cell tries to replenish z to maintain the system's sensitivity to S.

What is the simplest law that joins together both (5) and (6)? It is the following differential equation for the net rate of change, dz/dt, of z:

$$\frac{dz}{dt} = A(B - z) - Sz. \tag{7}$$

Equation 7 describes the following four processes going on simultaneously.

Accumulation or Production and Feedback Inhibition

The term $A(B - z)$ enjoys two possible interpretations, depending on whether it represents a passive accumulation process or an active production process.

In the former interpretation, there exist B sites to which transmitter can be bound, z sites are bound at time t, and $B - z$ sites are unbound. Then the term $A(B - z)$ says simply that transmitter is bound at a rate proportional to the number of unbound sites.

In the latter interpretation, two processes go on simultaneously. The term AB on the right-hand side of (7) says that z is produced at a rate AB. The term $-Az$ says that once z is produced, it inhibits the production rate by an amount proportional to the concentration of z. In biochemistry, such an inhibitory effect is called feedback inhibition by the end product of a reaction. Without feedback inhibition, the constant rate of production, AB, would eventually cause the cell to burst. With feedback inhibition, the net production rate is $A(B - z)$, which causes $z(t)$ to approach the finite amount B, as we desire by (6). The term $A(B - z)$ thus enables the cell to accumulate a target level B of transmitter.

Gating and Release

The term $-Sz$ in (7) says that z is released at a rate Sz, as we desire by (5). As in (5), release of z is due to mass action activation of z by S or to gating of S by z.

The two equations, (5) and (7), describe the simplest dynamic law that corresponds to the constraints (5) and (6). Equations 5 and 7 begin to reconcile the two constraints of unbiased signal transmission and maintenance of sensitivity when the signals are due to release of transmitter.

64. INTRACELLULAR ADAPTATION AND HABITUATION

First let us determine how the net signal, $T = Sz$, reacts to a sudden change in S. We will suppose that $z(t)$ reacts slowly compared to the rate with which S can change. For definiteness, suppose that $S(t) = S_0$ for all times $t \leq t_0$ and that, at time $t = t_0$, $S(t)$ suddenly increases to S_1. By (7), $z(t)$ reacts to the constant value $S(t) = S_0$ by approaching an equilibrium value $z(t_0)$. This equilibrium value is found by setting $dz/dt = 0$ in (7) and solving for

$$z(t_0) = \frac{AB}{A + S_0}. \tag{8}$$

By (8), a larger value of S_0 causes more transmitter to be released. In other words, $z(t_0)$ is a decreasing function of S_0. By contrast, (5) implies that the net signal to v_2 at time t_0 is

$$S_0 z(t_0) = \frac{ABS_0}{A + S_0}. \tag{9}$$

By (9), the rate of transmitter release is an increasing function of S_0. Now let $S(t)$ switch to the value $S_1 > S_0$. Because $z(t)$ is slowly varying, $z(t)$ approximately equals $z(t_0)$ for awhile after $t = t_0$. Thus, the net signal to v_2 during these times is approximately equal to

$$S_1 z(t_0) = \frac{ABS_1}{A + S_0}.$$

Equation 10 has the same form as a Weber law, $J(A + I)^{-1}$. The signal S_1 is evaluated relative to the baseline, S_0, just as J is evaluated relative to I. This Weber law is due to slow intracellular adaptation of the transmitter gate to the input level through time. It is not due to fast intercellular lateral inhibition across space, as reviewed in Grossberg (1980a, Appendix C and D).

As $z(t)$ in (7) begins to respond to the new transmitter level, $S = S_1$, $z(t)$ gradually approaches the new equilibrium point that is determined by $S = S_1$, namely

$$z(\infty) = \frac{AB}{A + S_1}.$$

The net signal consequently decays to the asymptote,

$$S_1 z(\infty) = \frac{ABS_1}{A + S_1}. \tag{12}$$

Thus, after $S(t)$ switches from S_0 to S_1, the net signal Sz jumps from (9) to (10) and then gradually decays to (12). The exact course of this decay is described by the equation

$$S_1 z(t) = \frac{ABS_1}{A + S_0} e^{-(A+S_1)(t-t_0)} + \frac{ABS_1}{A + S_1}(1 - e^{(A+S_1)(t-t_0)})$$

for $t \geq t_0$, which shows that the rate, or gain, $A + S_1$ of the response increases with the signal S_1, just as in the case of shunting lateral inhibition (Grossberg, 1980a). The sudden increment followed by slow decay can be intuitively described as an overshoot followed by habituation to the new sustained signal level, S_1. Both intracellular adaptation and habituation occur whenever a transmitter fluctuates more slowly than the signals that it gates. The size of the overshoot can be found by subtracting (12) from (10). For definiteness, let $S_0 = f(I)$ and $S_1 = f(I + J)$, where $f(w)$ is a function that transmutes the inputs I and $I + J$ that exist before and after the increment J into net signals S_0 and S_1, respectively. Then the overshoot size is approximately

$$S_1 z(t_1) - S_1 z(\infty) = \frac{ABf(I + J)[f(I + J) - f(I)]}{[A + f(I)][A + f(I + J)]}$$

In Section A4 below, I will show that the rebound size in response to specific cue offset is related to (14) in a way that allows us to estimate both $f(w)$ and the arousal level, I.

Much confusion can be avoided by realizing that more than one type of habituation can occur in the nervous system. Intracellular habituation due to a slow transmitter gate is not the only type of habituation. An intercellular variety of habituation can also occur. After a feedback expectancy is learned, a mismatch of the feedback expectancy with feedforward data can trigger an orienting reaction, or a dishabituation of the network's orienting subsystem. Feedback expectancies and slow gates are both needed to regulate perceptual and motivational events, but they are quite distinct. For example, I suggested in Section 37 that bistable perception of ambiguous figures is caused, even when the eye does not move, by a temporally cyclic habituation of transmitter gates, which shifts the pattern of STM activity and thereby cyclically activates alternative feedback expectancies. The expectancy that is active at any time

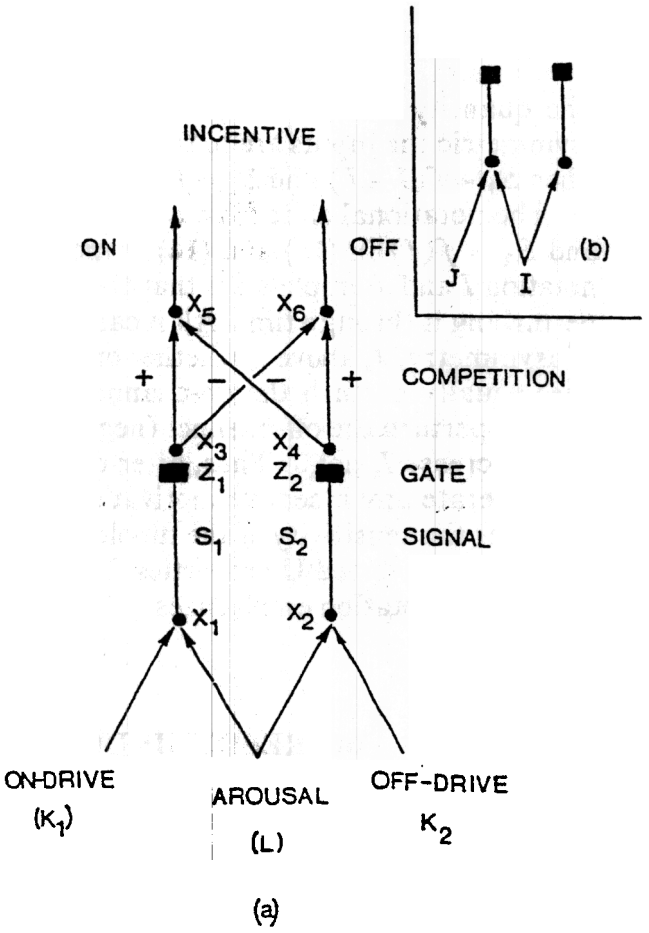


FIGURE 24. (a) Specific inputs (K_1 and K_2) and a nonspecific input (L) have the same effect as (b) a specific input J and a net arousal level I if $K_1 > K_2$.

organizes the ambiguous visual data into an unambiguous global percept (Grossberg, 1978a, 1980a).

65. A GATED DIPOLE

I will now indicate how, if transmitters gate signals before the gated signals compete, antagonistic rebound can be elicited by offset of a specific cue, as in light-on *versus* light-off, or fear *versus* relief. I will also show how unexpected events can cause an antagonistic rebound. They do this by triggering an increase in the level of nonspecific arousal that is gated by all the transmitter pathways.

FIGURE 24 depicts the simplest network in which two channels receive inputs that are gated by slowly varying transmitters before the channels compete to elicit a net

output response. Such a network is called a feedforward gated dipole. Two types of inputs will be introduced. Specific inputs are turned on and off by internal or external cues and nonspecific arousal inputs are on all the time, even though their size can vary through time. Each channel can have its own sum of specific inputs, K_1 or K_2, such as a hunger or satiety drive input, respectively, added to conditioned positive or negative reinforcing signals, and both channels receive the same arousal input, L. The total signals to the two channels are, therefore, $S_1 = f(K_1 + L)$, where the signal function, $f(w)$, is monotone increasing. We will see that the relative sizes of S_1 and S_2 and their rates of change through time relative to the transmitter fluctuation rate determine whether an antagonistic rebound will occur. To emphasize this fact, I define

$$I = \min (K_1 + L, K_2 + L)$$

and

$$J = |K_1 - K_2|$$

The quantity I determines the network's net arousal level and J determines how asymmetric the inputs are to the two channels. Suppose, for definiteness, that $K_1 > K_2$. Then $S_1 = f(I+J)$ and $S_2 = f(I)$.

The notational shift from $S_1 = f(K_1 + L)$ and $S_2 = f(K_2 + L)$ to $S_1 = f(I + J)$ and $S_2 = f(I)$ in (15) and (16) is motivated by more than formal convenience. The notation I and J emphasizes that the dipole doesn't know how many input sources are perturbing it through time. All it can compute is the net arousal level, I, and the degree of asymmetry, J, above I, whether one or a million input sources are active. If a million cues equally perturb the on-channel (positive reinforcers) and another million cues equally perturb the off-channel (negative reinforcers), the net effect of all the cues will be to increase I, not J. Thus, after dipole competition takes place, all these cues need not generate any incentive motivation. On the other hand, by increasing I, these cues can alter the sensitivity of the dipole to other asymmetrically distributed inputs due to the dipole's inverted U properties. This is the kind of simple but subtle distinction that the I and J notation emphasizes.

66. REBOUND DUE TO PHASIC CUE OFFSET

A rebound can be caused if, after the network equilibrates to the input J, the input is suddenly shut off. This effect is analogous to the reaction that occurs when a light is shut off or an aversive cue is shut off. To see how this rebound is generated, suppose that the arousal level is I and that the cue input is J. Let the total signal in the on-channel be $S_1 = f(I + J)$ and that in the off-channel be $S_2 = f(I)$. Let the transmitter in the on-channel, z_1, satisfy the equation

$$\frac{d}{dt} z_1 = A(B - z_1) - S_1 z_1 \tag{17}$$

and the transmitter in the off-channel, z_2, satisfy the equation

$$\frac{d}{dt} z_2 = A(B - z_2) - S_2 z_2.$$

After z_1 and z_2 equilibrate to S_1 and S_2, $(d/dt)z_1 = (d/dt)z_2 = 0$. Thus, by (17) and

(18),

$$z_1 = \frac{AB}{A + S_1}$$

and

$$z_2 = \frac{AB}{A + S_2}.$$

Since $S_1 > S_2$, it follows that $z_1 < z_2$; that is, z_1 is depleted more than z_2. However, the gated signal in the on-channel is $S_1 z_1$ and the gated signal in the off-channel is $S_2 z_2$. Since

$$S_1 z_1 = \frac{ABS_1}{A + S_1} \tag{21}$$

and

$$S_2 z_2 = \frac{ABS_2}{A + S_2}, \tag{22}$$

it follows from the inequality $S_1 > S_2$ that $S_1 z_1 > S_2 z_3$, despite the fact that $z_1 < z_2$. Thus, the on-channel gets a bigger signal than the off-channel. After the two channels compete, the input J produces a sustained on-output whose size is proportional to

$$S_1 z_1 - S_2 z_2 = \frac{A^2 B[f(I + J) - f(I)]}{[A + f(I)]\,[A + f(I + J)]}.$$

Division of (14) by (23) yields an interesting relationship between the size of the overshoot in the on-channel and the size of the steady-state on-output; namely,

$$\frac{\text{on-overshoot}}{\text{steady on-output}} = \frac{f(I + J)}{A},$$

which provides an estimate of $f(w)$ if J is parametrically varied. In particular, if $f(w)$ is a linear signal, $f(w) = w$, then (23) becomes

$$S_1 z_1 - S_2 z_2 = \frac{A^2 BJ}{(A + I)\,(A + I + J)},$$

which is an increasing function of J (more fear given more shock) but a decreasing function of I (linear analgesic effect).

Now shut J off to see how an antagonistic rebound (relief) is generated. The cell potentials rapidly adjust until new signal values, $S_1^* = f(I)$ and $S_2^* = f(I)$, obtain. However, the transmitters z_1 and z_2 change much more slowly, so that (19) and (20) are approximately valid in a time interval that follows J offset. Thus, the gated signals in this time interval approximately equal

$$S_1^* z_1 \simeq \frac{ABf(I)}{A + f(I + J)} \tag{26}$$

and

$$S_2^* z_2 = \frac{ABf(I)}{A + f(I)}. \tag{27}$$

Thus, $S_1^* z_1 < S_2^* z_2$. The off-channel now gets the bigger signal, so an antagonistic rebound occurs, the size of which is approximately

$$S_2^* z_2 - S_1^* z_1 = \frac{ABf(I)[f(I + J) - f(I)]}{[A + f(I)]\,[A + f(I + J)]}$$

Division of (28) by (23) yields an interesting relationship between the maximal off-output and the steady on-output; namely,

$$\frac{\text{off-output}}{\text{on-output}} = \frac{f(I)}{A}, \tag{29}$$

which provides an estimate of $f(w)$ as I is parametrically varied. A comparison of (24) with (29) shows that, as I is parametrically varied, (24) should have the same graph as (29), shifted by J. This comparison provides an estimate of J (that is, of how the behavioral input is transformed into neural units) and also a strong test of the model. Once $f(w)$ is estimated, (23) and (28) can be verified.

If $f(w) = w$ in (28), then

$$S_2^* z_2 = S_1^* z_1 = \frac{ABIJ}{(A + I)\,(A + I + J)}$$

The rebound is then an increasing function of J (offset of larger shock elicits more relief) and an inverted U function of I (an optimal arousal level exists).

The rebound is transient because the equal signals, $S_1 = S_2 = f(I)$, gradually equalize the z_1 and z_2 levels until they both approach $AB(A + f(I))^{-1}$. Then $S_1 z_1 - S_2 z_2$ approaches zero, so the competition between channels shuts off both of their outputs.

67. REBOUND DUE TO AROUSAL ONSET

A surprising property of these dipoles of on-cell and off-cell pairs is their reaction to sudden increments in the arousal level, I. Such increments are, for example, thought to occur in response to unexpected events.

Suppose that the on-channel and the off-channel have equilibrated to the input levels I and J. Now suddenly increase I to I^*, thereby changing the signals to $S_1^* = f(I^* + J)$ and $S_2^* = f(I^*)$. The transmitters z_1 and z_2 continue to obey (19) and (20) for awhile, with $S_1 = f(I + J)$ and $S_2 = f(I)$. A rebound occurs if $S_2^* z_2 > S_1^* z_1$. In general,

$$S_2^* z_2 - S_2^* z_1$$

$$= \frac{AB[f(I^*) - f(I^* + J)] + B[f(I^*)f(I + J) - f(I)f(I^* + J)]}{[A + f(I)]\,[A + f(I + J)]}$$

In particular, if $f(w) = w$, a rebound occurs whenever

$$I^* > I + A,$$

since then

$$S_2^* z_2 - S_1^* z_1 = \frac{ABJ(I^* - I - A)}{(A + I + J)(A + I)}.$$

Thus, given a linear signal function, a rebound will occur if I^* exceeds $I + A$ no matter how J is chosen. If the event is so unexpected that it increments the arousal level by more than amount A, then all on-cell–off-cell dipoles in the network will simultaneously rebound. Moreover, the size of the off-cell rebound increases as a function of the size of the on-cell input, J, as (33) shows. In particular, no rebound occurs if the on-cell was inactive before the unexpected event occurs. Thus, the rebound mechanism is selective. It rebounds most vigorously those cells which are most active ($J >> 0$) and spares inactive cells ($J \simeq 0$).

68. INVERTED U IN DIPOLE OUTPUT

The inverted U effect holds if $f(w)$ is a sigmoid function; that is, if $f(0) = df/dw(0) = 0$, $df/dw(w) > 0$ if $w > 0$, $f(\infty) < \infty$, and $d^2f/dw^2(w)$ changes sign once from positive to negative as w increases. In particular, if $f(w)$ is sigmoid, an inverted U occurs in the sustained on-output (23) as I is parametrically increased, despite the fact that an inverted U does not obtain in (25) when $f(w)$ is linear. To simplify the results, I use the signum function

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

I first consider the on-reaction in (23), which I denote by x_5 (FIGURE 24). I write the derivative of a function $g(I)$ as $g'(I)$. Then, by (23), for each fixed J,

$$\operatorname{sgn}\{x_5'(I)\} = \operatorname{sgn}\{A^2[f'(I + J) - f'(I)] + 2A[f(I)f'(I + J) - f(I + J)f'(I)] + [f^2(I)f'(I + J) - f^2(I + J)f'(I)]\}.$$

Since $f(w)$ is sigmoid,

$$f(0) = f'(0) = 0.$$

Thus, by (35) and (36),

$$\operatorname{sgn}\{x_5'(0)\} = \operatorname{sgn}\{A^2 f'(J)\} > 0.$$

At large values of I,

$$f(I + J) > f(I),$$

whereas

$$f'(I + J) < f'(I).$$

Consequently, each term in brackets on the right-hand side of (35) is negative. Thus, at large I values,

$$\text{sgn}\,\{x_5'(I)\} < 0 \tag{40}$$

The inequalities (37) and (40) show that, for fixed J, $x_5(I)$ increases and then decreases as a function of I. This is the inverted U for the on-reaction. In fact, since $f(\infty) < \infty$, (23) implies that $\lim_{I\to\infty} x_5(I) = 0$. A similar proof holds for the off-reaction.

69. HYPERSENSITIVE UNDERAROUSED REACTION TO PHASIC INCREMENTS

To illustrate why the underaroused syndrome is hypersensitive to phasic increments, suppose that I is chosen abnormally small and, consequently, that $f(I)$ is very small because of f's S-shaped graph. Let J represent the intensity of a fearful cue (*e.g.*, a shock level) and let the dipole on-output (23) be correlated with the amount of fear. Since I is so small, the "fear threshold is raised" in the sense that a larger value of J is needed to create a large net on-output than when I is chosen in the "normal" range. Although the fear threshold is high, once J is chosen sufficiently large to elicit a detectable net on-reaction, additional increments in J create larger than normal increments in fear. This is because the terms $f(I)$ in the numerator and denominator of (23) are abnormally small. More precisely, differentiating (23) with respect to J, we find the rate at which the on-output increases to unit increases in J. This rate is

$$\frac{\partial}{\partial J}(S_1z_1 - S_2z_2) = \frac{A^2Bf'(I+J)}{[A+f(I+J)]^2}.$$

If $I + J$ is chosen so that $f(I + J)$ is small but growing rapidly, then $f'(I + J)$ is relatively large when the denominator, $[A + f(I + J)]^2$, is relatively small. In other words, underaroused depression is hyperexcitable despite its high threshold.

70. PARADOXICAL ON-REACTION TO UNEXPECTED EVENTS AND DIFFERENTIAL ENHANCEMENT OF OVERSHADOWED CUES

Two other properties of underaroused dipoles are related to Parkinsonian bracing. These properties, like underaroused hyperexcitability, are due to the faster-than-linear, or threshold, behavior of the S-shaped signal function, $f(w)$, at small activity values, w. Neither property holds if the signal function is linear, say $f(w) = w$. In particular, by (33), when $f(w) = w$, an arousal increment ΔI in response to an unexpected event causes a rebound whenever $\Delta I > A$. The minimal ΔI capable of causing a rebound is independent of the ambient arousal level, I. This property does not hold when $f(w)$ grows faster than linearly, say $f(w) = w^2$, which approximates the sigmoid shape of $f(w)$ at low arousal levels. By (31), a rebound occurs when $f(w) = w^2$ only if

$$\Delta I > g(I,J), \tag{42}$$

where the function

$$g(I,J) = \frac{A - I(I+J) + (A+I^2)^{1/2}[A+(I+J)^2]^{1/2}}{2I+J}$$

is a decreasing function of I. In fact, $g(I,J)$ approaches 0 as I is chosen arbitrarily large. Thus, a much larger ΔI is needed to rebound an underaroused dipole than a normally aroused dipole. Moreover, if $\Delta I < AJ^{-1}$, then when $I \simeq 0$,

$$\frac{\partial}{\partial(\Delta I)}\left[\frac{(I+\Delta I+J)^2}{A+(I+J)^2}-\frac{(I+\Delta I)^2}{A+I^2}\right]>0. \tag{44}$$

In other words, an arousal increment can actually enhance the on-output of an underaroused dipole instead of rebounding the dipole.

Use of a sigmoid function also helps explain how overshadowed cues can be enhanced even though very active cues are inhibited when an arousal burst occurs. This is because the function $g(I,J)$ is a decreasing function of J, as well as of I. This means that it is easier to rebound a more active STM representation than a less active STM representation.

71. PARADOXICAL LACK OF REBOUND TO PHASIC DECREMENT: ORDERING OF REINFORCEMENT MAGNITUDE

This section illustrates how several behavioral indices should all covary as arousal level is parametrically increased. The first index says that reducing J units of shock to $J/2$ units is less rewarding than reducing $J/2$ units of shock to 0 units, despite the fact that both operations reduce shock by $J/2$ units. This result is based on the fact that (23) and (28) include ratios of I and J effects as well as differences of I and J effects. In fact, this result can be generalized to a formula that predicts when reducing J_1 units of shock to K_1 units at arousal level I_1 is more reinforcing than reducing J_2 units of shock to K_2 units at arousal level I_2 (Grossberg, 1972b). To make these assertions, I assume that the size of the relief rebound caused by reducing the shock level is proportional to the rewarding effect of the manipulation, other things being equal (which is usually false!).

To derive these effects, it is convenient to use a signal function

$$f(w) = \max(w - C, 0). \tag{45}$$

Such a signal function has a threshold C, below which it equals 0 and above which it grows linearly. This threshold function approximates a sigmoid function in the activity range before saturation occurs. I will also use the following notation. I denote the steady-state on-reaction that occurs after a specific input of intensity J is kept on for S time units by $x_5(S, J \rightarrow K)$ and the off-rebound that occurs when intensity J is switched to K at time $t = S$ by $x_6(S^+, J + K)$. To compute $x_6(S^+, J \rightarrow K)$, I approximate the transmitters by their steady-state values at $t = S$ and the potentials by their new steady-state values in response to input K.

Let us choose an arousal level I that exceeds the threshold, C. Then, proceeding as in our previous computations, we find that

$$x_6\left(S^+, J \rightarrow \frac{J}{2}\right) = \frac{AB\frac{J}{2}(I-A-C)}{(D+I)(D+I+J)}$$

where $D = A - C$. By comparison, (23) and (28) imply that

$$x_5(S, J \rightarrow 0) = \frac{A^2BJ}{(D+I)(D+I+J)}$$

and

$$x_6(S^+, J \to 0) = \frac{ABJ(I - C)}{(D + I)(D + I + J)}, \tag{48}$$

from which it also follows that

$$x_6\left(S^+, \frac{J}{2} \to 0\right) = \frac{AB\frac{J}{2}(I - C)}{(D + I)\left(D + I + \frac{J}{2}\right)} \tag{49}$$

and

$$\frac{x_6(S^+, K \to 0)}{x_5(S, K \to 0)} = A^{-1}(I - C)$$

for any $K > 0$. Comparing (46) and (49), we find that

$$x_6\left(S^+, \frac{J}{2} \to 0\right) > x_6\left(S^+, J \to \frac{J}{2}\right),$$

or that cutting J units in half is less rewarding than shutting off $J/2$ units. We also confirm that the ratio (50) increases with I, as in the more general equation (29). We can now substitute (50) into (46) to find that

$$x_6\left(S^+, J \to \frac{J}{2}\right) = \frac{A^2B\frac{J}{2}[x_5^{-1}(S, K \to 0)\, x_6(S^+, K \to 0) - 1]}{(D + I)(D + I + J)} \tag{52}$$

By (52), an arousal level that favors the possibility of learned avoidance in the presence of fearful cues also favors a large rewarding effect when the shock level is halved. If I is chosen to be small (underarousal), then x_6 in (46) can be negative (no rebound occurs) even if x_6 in (49) is positive (a rebound occurs).

72. INHIBITING EXCITATORY RESISTANCE VS. EXCITING INHIBITORY CONDUCTANCE IN DISINHIBITORY INCENTIVE MOTIVATIONAL PATHWAYS

Thus far, our discussion of gating effects has ignored the fact that the post-synaptic target cells possess only a finite number of excitable sites that can be turned on and off. This fact is, however, an important constraint on the design of cellular mechanisms, since, after all the sites are turned on, the target cell is insensitive to later input fluctuations; in other words, the target cell saturates. As I noted in Section 14, this noise-saturation dilemma is overcome by competitive interactions among cells that undergo mass action, or shunting, dynamics. Shunting dynamics occur, for example, in the membrane equation that is a cornerstone of experimental neurophysiology. Such an equation takes the form

$$C\frac{dV}{dt} = (V^+ - V)g^+ + (V^- - V)g^- + (V^p - V)g^p \tag{53}$$

for the time rate of change dV/dt of a cell potential $V(t)$. The constant C is a capacitance; the constants V^+, V^-, and V^p are saturation points for the excitatory, inhibitory, and passive channels, respectively; and the terms g^+, g^-, and g^p are conductances for the excitatory, inhibitory, and passive channels, respectively. I will assume by convention that $V^+ > V^p = 0 \geq V^-$. Then $V^+ \geq V(t) \geq V^-$ at all times t. Our previous discussion tacitly assumed that inputs that change g^+ or g^- are sufficiently small to prevent $V(t)$ from saturating at V^+ or V^-. Thus, the $V(t)$ terms in the excitatory and inhibitory channels of (53) have a negligible effect and we have until now ignored them. In general, however, this is false. Let us now study how including the saturation terms can sharpen our concepts.

To fix ideas, let $g^p = A$ (a positive constant), let $g^- = 0$, and let $g^+ = Sz$, where z is a slow transmitter that gates the input S. Also choose $C = 1$ for simplicity and write $V^+ = B$. Then

$$\frac{dV}{dt} = -AV + (B - V)Sz.$$

The equilibrium potential of (54) is found by setting $dV/dt = 0$. It is

$$V_\infty = \frac{BSz}{A + Sz}$$

Had we ignored the excitatory term $-VSz$ of (54) to study

$$\frac{dV}{dt} = -AV + BSz$$

instead, we would have found the equilibrium potential $BA^{-1}Sz$. This is what we tacitly did to derive x_3 and x_4 in FIGURE 24. The steady-state potential V_∞ resembles Sz in that both increase as a function of Sz so both will habituate if either one does. However, (54) differs from (56) in two crucial ways. (1) Automatic Gain Control: The gain, or averaging rate, of $V(t)$ in (54) is $-(A + Sz)$, which increases as Sz increases. In (56), the gain is the constant A. (2) Saturation: V_∞ saturates at B as Sz becomes large.

Often it is desirable to prolong cell response, rather than speed it up, as input size increases. One way to accomplish this is to increase the resistance of the excitatory channel rather than its conductance. This operation hyperpolarizes (inhibits) rather than depolarizes (excites) the cell potential (Baylor and Hodgkin, 1974; Baylor *et al.*, 1974a, b), but this reversal in sign is unimportant if the inhibited cell is part of a two-state disinhibitory pathway.

To slow down cell response in this way, let

$$\frac{dV}{dt} = -AV + (B - V)G, \tag{57}$$

as in (54), where $G(t)$ is the number of open membrane "gates" at time t. Also let

$$\frac{dG}{dt} = \alpha(\beta - G) - SzG. \tag{58}$$

Equation 58 says that G obeys an accumulation-depletion equation just like (7) with signal Sz. The term $\alpha(\beta - G)$ says that the system strives to keep β gates open. The term SzG says that open gates, which number G, are closed by the signal Sz. Suppose

can treat G as if it is always in equilibrium with respect to Sz and, by (58),

$$G \simeq \frac{\gamma}{\alpha + Sz}, \tag{59}$$

where $\gamma = \alpha\beta$. By (59), when $S = 0$, the number of open gates, G, approaches $G_0 = \beta$ and the potential, V, approaches $V_0 = BG_0(A + G_0)^{-1}$. We are interested in the quantity $x = V_0 - V$, where V is the equilibrium potential induced by a prescribed input, S. The function x measures how much V is hyperpolarized by the gated signal Sz.

We find that

$$x = \frac{USz}{V + Sz} \tag{60}$$

where $U = \beta B(1 + \beta A^{-1})^{-1}$ and $V = \alpha + \gamma A^{-1}$. Note that V_∞ in (55) exhibits the same steady-state dependence on Sz as does x in (60). Nonetheless, the gain of (57) is $A + (\gamma/(\alpha + Sz))$, which is a decreasing function of Sz rather than an increasing function of Sz. All in all, (57) and (58) describe a tonically active cell ($V_0 > 0$) whose rate of inhibition by gated inputs Sz decreases as Sz increases.

To elicit a prolonged response to inputs in a disinhibitory pathway, suppose that the output $f(V)$ of (57) inhibits the conductance of the potential $W(t)$, as in the equation

$$\frac{dW}{dt} = -AW + (B - W)C - f(V)W.$$

The term $(B - W)C$ says that the cell maintains a tonic excitatory conductance. The term $-f(V)W$ says that the excitatory tonic activity is counteracted by inhibitory tonic activity. As V decreases due to an increase in Sz, $f(V)$ also decreases. The gain $A + f(V)$ of W is thus decreased as the asymptote of W is increased. All in all, a two-stage disinhibition in which the first stage increases excitatory resistance and the second stage decreases inhibitory conductance significantly prolongs the effect of an input pulse. Moreover, if $f(V) = kV$, then the steady-state depolarization, $W - W_0$, again has the form $USz(V + Sz)^{-1}$.

The design of this disinhibitory pathway raises the experimental question: Do (for example) the DA-GABA feedback loops between the neostriatum and the substantia nigra (Groves *et al.*, 1978) contain cells whose increase in excitatory resistance causes a decrease in the inhibitory conductance of their target cells?

73. INTRACELLULAR DIPOLES

Part of understanding a design principle is being able to recognize how different anatomies can compute the same functional properties. For example, if both excitatory and inhibitory conductances are nonzero in a cell membrane, and if at least one of these conductances is altered by a gated signal, then the cell potential can generate both on-overshoot and off-rebound effects. This system acts like an intracellular dipole. In particular, suppose that

$$\frac{dV}{dt} = -AV + (B - V)\frac{\gamma}{\alpha + Sz} - (V + C)D \tag{62}$$

and

$$\frac{dz}{dt} = E(F - z) - Sz.$$

The term $(B - V)(\gamma/(\alpha + Sz))$ in (62) acts like the on-channel of the dipole and the term $-(V + C)D$ acts like the off-channel. In response to a sustained increment in S, the on-channel overshoots, then habituates, as $z(t)$ in (63) is slowly depleted by the larger value of S. A sudden decrement in S causes the potential to rapidly decrease and then to slowly increase as $z(t)$ accumulates in response to the smaller value of S. Thus, a dipole can be realized by a single cell, rather than by two parallel competing pathways, as occurs in *Gekko gekko* rods (Carpenter and Grossberg, 1981; Kleinschmidt and Dowling, 1975).

74. PRESYNAPTIC NORMALIZATION BY TRANSMITTER DIFFUSION AND FEEDBACK INHIBITION

When diffusion and reuptake of released transmitter can occur between synapses, the total amount of transmitter can be controlled by the extra feedback inhibition that is due to reuptake. Without reuptake, the simplest transmitter law at the ith synapse is

$$\frac{dz_i}{dt} = A(B - z_i) - S_i z_i$$

where S_i is the input and z_i is the transmitter at the synapse. With reuptake, and ignoring time-delay effects, the simplest transmitter law becomes

$$\frac{dz_i}{dt} = A\left(B - z_i - \sum_{k=1}^{n} S_k z_k C_{ki}\right) - S_i z_i,$$

where the term $-AS_k z_k C_{ki}$ describes the extra feedback inhibition due to uptake of released transmitter $S_k z_k$ from the kth synapse at the ith synapse. To understand the normalizing effect of intercellular diffusion, consider the steady-state rate $T = \Sigma_{k=1}^{n} S_k z_k$ of total transmitter release in two extreme cases: (1) where no diffusion occurs (all $C_{ki} = 0$) and (2) where long-range diffusion occurs (all $C_{ki} = C > 0$). Suppose for definiteness that all inputs are equal, say $S_i = S$. By (65), in case (1),

$$T = \frac{nABS}{A + S} \leq ABn, \tag{66}$$

which increases linearly with the number of cells, n. In case (2),

$$T = \frac{nABS}{A + S + nCS} \leq ABC^{-1}, \tag{67}$$

whose maximum is independent of n. This result helps to explain the experiments of Stricker and Zigmond (1976) on recovery from damage to the dopaminergic synapses of the nigrostriatal bundle, since reducing the feedback inhibition from damaged cells allows undamaged calls to produce more transmitter in a compensatory fashion.

75. PARADOXICAL INHIBITORY ACTION OF EXCITATORY TRANSMITTER ON TONICALLY AROUSED CELLS

A tonically active cell that uptakes extracellular transmitter can generate paradoxical responses when the transmitter is extracellularly applied by an experimenter. In particular, extracellular release of an excitatory transmitter can have a net inhibitory effect on a postsynaptic cell that is normally excited by the transmitter. If the presynaptic cell uptakes extracellular transmitter at a rate proportional to transmitter concentration, then the net effect of applying extracellular transmitter will be inhibitory at all concentrations if it is inhibitory at any concentration. The critical level of tonic activity needed to cause net inhibition decreases as a function of the uptake rate. This possibility can create difficulties in interpreting central actions of those transmitters, such as catecholamines, which can be tonically activated by an animal's state of deprivation and arousal.

To prove these properties, denote the habituated, or steady-state, rate of transmitter release in response to the tonic signal S in (7) by

$$T_\infty = Sz_\infty = \frac{ABS}{A + S}. \tag{68}$$

Let a quantity J of transmitter be injected into a region near this synapse. Let the transmitter uptake rate by the synaptic knob equal the fraction θJ, and let the rate with which transmitter directly excites the postsynaptic cell equal the fraction ϕJ. By (7) the uptake of transmitter inhibits the rate of transmitter production *via* feedback inhibition. Then (7) becomes

$$\frac{dz}{dt} = A(B - \theta J - Z) - Sz,$$

whose habituated transmitter level is

$$z_\infty^* = \frac{A(B - \theta J)}{\quad}$$

The habituated rate of transmitter release is, therefore,

$$T_\infty^* = Sz_\infty^* = \frac{A(B - \theta J)S}{A + S} \tag{71}$$

and the total postsynaptic signal is

$$T_\infty^* + \phi J.$$

The transmitter J will have a net inhibitory effect on the postsynaptic cell if

$$T_\infty > T_\infty^* + \phi J, \tag{73}$$

which is true if

$$\frac{AS}{A + S} > \phi\theta^{-1}$$

The most important property of (74) is that it does not depend on the transmitter

concentration, J. It depends only on the tonic level, S, and on the uptake fraction, θ. If θ is close to 1, then a small tonic level, S, suffices to convert any concentration of excitatory transmitter into a net inhibitory response. This inhibitory effect can be eliminated either by pharmacologically blocking presynaptic transmitter uptake or by transecting the source of tonic input. The critical uptake fraction at which reversal of the inhibitory effect occurs depends on the size of the tonic postsynaptic signal $T_\infty(S)$ in (**68**), according to an equation of the form

$$\theta_{\text{crit}} = \frac{C}{1 + BT_\infty(S)}$$

where B and C are positive constants. These results indicate that paradoxical transmitter sign reversals might seem to occur in the tonic cells of networks whose arousal level cannot be independently calibrated.

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