

## CHAPTER 6

### A NEURAL MODEL OF ATTENTION, REINFORCEMENT AND DISCRIMINATION LEARNING

#### PREFACE

This article discusses several important phenomena wherein present behavior depends on how temporal and geometrical relationships among past events are influenced by competitive feedback.

The overshadowing phenomenon shows that learners are minimal adaptive predictors who change their internal representations of external events when behavior based upon these representations generates unexpected environmental feedback. These data support an epistemological framework wherein each observer can possess idiosyncratic and personal definitions of objects or events, and these definitions are refined only when they are maladaptive. Many constraints for designing self-organizing measurement systems and for assigning *a priori* optimal estimators of the future are suggested by such a framework. The overshadowing phenomenon can be derived from a thought experiment concerning parallel processing of sensory cues which are conditioned to incompatible motivational meanings.

The partial reinforcement acquisition effect shows that "less is more" in the sense that intermittent reward can have more pervasive effects on behavior than continuous reward. Chronic gambling behavior is a typical example. This phenomenon can also be traced to the action of expectancies, notably to the disconfirmation of expectancies. As in serial learning, the nonoccurrences of events are no less important than their occurrences. The theory shows that partially rewarded behavior, which can seem maladaptive or even self-destructive, occurs when several adaptive mechanisms are simultaneously probed by certain environmental events. This result suggests that to understand whether behavior is 'really' adaptive, one must distinguish those pervasive environmental constraints that influence the evolution of neural designs from adventitious environmental fluctuations that merely play upon these designs. Once one has arrived at this insight, it also seems natural to ask whether the environment itself contains internal 'contradictions' which can manifest themselves in maladaptive behavior (Chapter 9).

The peak shift and behavioral contrast effect shows that 'more is less' in

the sense that events which have never been rewarded can elicit more vigorous consummatory behavior than events which have been highly rewarded. If asked: "Will our learned preferences always be chosen from those past experiences which have been rewarded?", the answer must in general be 'no'. Internal perceptual and cognitive maps can reorganize the net effects of rewards and punishments to favor as yet unexperienced alternatives. It seems ironic to me that Skinnerians, who in their traditional garb detest the use of hidden variables, should be the scientists who have best described these phenomena. Their data base is much richer than their philosophy.

Hidden variables also play an important role in answering questions like: "What motivates avoidance of a fearful situation when the avoidance behavior persists without the experience of renewed fear? What motivates language behavior?" This article suggests some answers.

My earlier work on reinforcement (Chapters 3 and 5) recognized that competitive interactions among the internal representations of external cues are needed to normalize the total STM activity of these representations. This finite capacity constraint on STM prevents large number of active STM representations from causing the release of incentive motivation if the corresponding drive level is too low. The decisive step in this article was the realization that incentive motivational signals can feed back into the competing internal representations of external cues, and thereby bias which cues will be attended. Attentional processing was hereby associated with a feedback network wherein adaptive resonances subserve the attentional state. This feedback network idealizes interactions between cortical and hippocampal structures, and implicates the hippocampus in evaluating stimulus-reinforcement contingencies that can activate observable behavior when a resonant match occurs between the reinforcing properties of available external cues and the motivating properties of active internal drives. A separate spatial map from the hippocampus is also postulated. When this work was finished in 1973, I had not yet explicitly derived the adaptive resonance concept. When I did so during the next two years from the standpoint of code development, I could mechanistically identify a sense in which adult attentional processing is a continuation of infant critical period phenomena along a developmental continuum.

Another basic concept, both in code development and in adult attention, is also related to competition, but competition that is acting on a more global scale of system design. This concept carries the factorization notion to a global extreme. It describes the competition between the consummatory behavior that generates expected consequences and the orienting behavior that mobilizes adaptation to unexpected consequences. Dualities between the expected and the unexpected, between structure and mobile energy,

between reason and passion have been explored and debated throughout human history. A good attentional model should allow us to mechanistically contemplate how these dual principles achieve the exquisitely poised balance between stability and adaptability that, moment by moment, subserves our capacity to be human.

# A NEURAL MODEL OF ATTENTION, REINFORCEMENT AND DISCRIMINATION LEARNING\*

## I. Introduction

This paper describes a psychophysiological model aimed at discussing how animals pay attention to and discriminate among certain cues while ignoring others, based on criteria of relevance derived from past experience or innately preprogrammed in their neural apparatus. The model builds upon previous results (Grossberg, 1969a,b, 1970, 1971a,b, 1972a-d, 1973, 1974; Grossberg and Pepe, 1971) that introduce some psychophysiological mechanisms of classical and instrumental learning, and of pattern discrimination. These results include network mechanisms of drive, reward, punishment, escape and avoidance, motivation, short-term and long-term memory, serial learning, arousal, expectation, and various perceptual constancies (e.g., hue and brightness). They will be reviewed herein as needed to motivate the present work. A previous paper (Grossberg, 1974) reviews some of them more systematically.

This collection of mechanisms comprises the theory of Embedding Fields. This theory derives neural networks from simple psychological facts that are taken as fundamental postulates. The theory tries to isolate postulates that act as guiding principles of neural design during individual development and the evolution of species. The networks that are hereby derived are capable of behavior that is far more complex and subtle than the postulates themselves, and also generate various new predictions. The theory is derived by a method of successive approximations; as more postulates are imposed, the networks become ever more sophisticated and realistic. At each stage of the derivation, basic mechanisms of network organization emerge, and are preserved as new postulates are imposed. Thus, each stage of the derivation ties a definite class of psychophysiological phenomena to a fixed list of elementary postulates, and successive stages of the derivation show how various phenomena of differing sophistication are interrelated.

A central theme in the present model will be that two systems are continually readjusting each other. One system (an attentional system) strives toward an ever more stable response to patterns of fluctuating cues by focusing attention on important subclasses of cues. This system is incapable of adapting to unexpected environmental changes. The second system (an arousal system) overcomes the rigidity of the attentional system when unexpected events occur, and allows the network to adapt to new reinforcement

\* Supported in part by the Alfred P. Sloan Foundation, the Office of Naval Research (N00014-67-A-0204-0051), and the Advanced Research Projects Agency DAHC 15-73-0320) administered by Computer Corporation of America.

contingencies. The following psychophysiological themes, which clarify this situation, will be discussed in the model, among others.

#### A. BLOCKING AND OVERSHADOWING

This theme is elegantly discussed by Honig (1970), Kamin (1968, 1969), Trabasso and Bower (1968), and Wagner (1969a), who should be consulted for details. Below are tersely summarized some main experimental facts taken from these sources. We will consider a sequence of three classical conditioning experiments. In each experiment, two cues  $CS_1$  and  $CS_2$ , such as a sound and a flashing light, are the conditioned stimuli that will precede a prescribed unconditioned stimulus UCS, such as food or shock. Let the UCS be a shock of prescribed duration and intensity, for definiteness.

In experiment 1, let  $CS_1$  and  $CS_2$  be equally salient to the learning subject  $\Theta$ , and suppose that both cues are always presented together before the shock. On recall trials, will  $\Theta$  be afraid of  $CS_1$  or  $CS_2$  presented separately? The answer is "yes"; thus, cues presented together can be conditioned separately.

In experiment 2, first let  $CS_1$  be paired alone with shock, until  $\Theta$  is afraid of  $CS_1$ . Then present both  $CS_1$  and  $CS_2$  before shock during the second phase of the experiment. On recall trials,  $\Theta$  is *not* afraid of  $CS_2$ . Somehow, prior conditioning of  $CS_1$  to the UCS has "blocked," or "overshadowed," the possibility of conditioning  $CS_2$  to the UCS. This happens even though  $\Theta$  "notices"  $CS_2$ , and the amount of blocking depends on the amount of prior conditioning between  $CS_1$  and the UCS. A blocking effect can also be elicited in experiment 1 if  $CS_1$  is a more intense, or salient, cue than  $CS_2$ . In a similar direction, Bitterman (1965) discussed evidence that a CS which is paired simultaneously with a UCS does not get conditioned to the UCR.

In experiment 3, again pair  $CS_1$  with the UCS before pairing both  $CS_1$  and  $CS_2$  with the UCS; however, choose the UCS intensity at two different levels in the two phases of the experiment. Then the blocking effect is at least partially eliminated:  $\Theta$  is afraid of  $CS_2$ . (In general, one must also discuss whether a decrease in shock makes  $CS_2$  a conditioned source of relief, rather than of fear.)

These experiments can be interpreted as follows. In the second phase of experiment 2,  $CS_1$  is a perfect predictor of the event UCS that is about to follow. Since  $CS_2$  is an irrelevant cue,  $\Theta$  does not connect  $CS_2$  with the UCR even though  $\Theta$  notices  $CS_2$ . In the second phase of experiment 3, however,  $CS_1$  is not a perfect predictor of UCS intensity. Hence some conditioning of  $CS_2$  to the new UCR (or UCR-like response) occurs. In experiment 1, neither  $CS_1$  nor  $CS_2$  is initially a predictor of the UCS. Hence  $\Theta$  will learn connections from each  $CS_1$  to UCR. If  $CS_1$  is more salient or intense than

$CS_2$ , then faster conditioning of  $CS_1$  to the UCS can eventually block conditioning of  $CS_2$  to the UCR.

Such experiments suggest that various learning subjects act as minimal adaptive predictors; they enlarge the set of cues that control their behavior only when the cues that presently control their behavior do not perfectly predict subsequent events. In particular, somehow the results of  $\Theta$ 's acts can feed back in time to influence which cues will control these acts in the future. This phenomenon has broad implications, since it bears on such questions as: How do we decide which cues cause events and which are adventitious? How do we characterize the cues that define the objects with which we deal? Does the persistent unpredictability of a given source of cues increase the likelihood that this source will be treated more as a "subject" than as an "object"?

### B. FRUSTRATIVE NONREWARD

A special case of an unpredictable event is one in which an expected reward does not occur. Suppose that  $\Theta$  has learned to expect food as the end result of a particular sequence of motor acts, but that food is no longer available in the expected place. Were  $\Theta$  to continue seeking food at this place,  $\Theta$  would starve to death. How does  $\Theta$  countercondition this erroneous expectation, and thereby release exploratory behavior aimed at finding new sources of food, before starvation occurs?

An aversive state that is activated by the nonoccurrence of expected events is "frustration" (Amsel, 1958, 1962; McAllister and McAllister, 1971; Wagner, 1969b). Frustration can motivate avoidance behavior and has properties analogous to those of fear.

Frustration can follow the nonoccurrence of expected rewards other than food. Thus if a sequence of events motivated by a given positive drive is suddenly interrupted, say by nonoccurrence of the expected reward at the end of a sequence of acts aimed at getting the reward, then a negative (frustrative) reaction can occur. We will argue that this rebound effect, from positive to negative, can be given a mechanistic interpretation that is shared by rebound effects from negative to positive, such as the relief that is felt when a prolonged shock is unexpectedly terminated (Denny, 1970), or various other punishment contrast and reinforcement contrast effects (Azrin and Holz, 1966). For example, let a pigeon be trained on a VI 1 schedule to peck for food. If a maintained level of punishment is suddenly removed, the pigeon will temporarily peck faster than it did in the absence of punishment. If the frequency of reward is suddenly increased, a temporary overshoot in pecking rate will again occur. The mechanism to be discussed herein also allows comparison with the facts that classically conditioned fear can

rapidly extinguish, even though learned asymptotic avoidance behavior can be very stable (Seligman and Johnston, 1973).

#### C. PARTIAL REINFORCEMENT ACQUISITION EFFECT

Why can fearful or frustrating tasks that work out well in the end become so rewarding? What causes the extra "thrill" that some people seem to feel after successfully carrying out dangerous tasks? An analogous boost in reward value is illustrated by the following example. Consider the speed with which rats run down a straight alley to a positive goal. Compared to continuously rewarded animals, animals on a random partial reinforcement schedule run slower early in training, gradually catch up, and finally, late in training, run faster (Goodrich, 1959; Haggard, 1959). This effect has been attributed by several authors to frustration (Gray and Smith, 1969). We will suggest a property of the frustration mechanism that can formally generate this effect, and can predict a relationship between an animal's ability to carry out learned escape in the presence of fearful cues, the reinforcing effect of reducing  $J$  units of shock to  $J/2$  units of shock, the size of the partial reinforcement acquisition effect, and the animal's arousal level, suitably defined.

#### D. STEEPENING OF GENERALIZATION GRADIENTS DUE TO DISCRIMINATION TRAINING

Jenkins and Harrison (1960) showed that if pigeons are trained to peck a key in response to a 1000 cps tone (the  $S+$ ) but not to peck in the absence of the tone (the  $S-$ ), then a sharper tonal generalization gradient is found than after training to peck at the  $S+$  without discrimination training with  $S-$ .

Newman and Baron (1965) used a vertical white line on a green key as  $S+$  and the green key as  $S-$ . They tested generalization by tilting the line at various orientations. A generalization gradient was found, but no gradient occurred if the  $S-$  was a red key or if the  $S-$  was a vertical white line on a red key.

By contrast, Newman and Benefeld (Honig, 1970) used as  $S+$  a vertical white line on a green key and as  $S-$  a green background, 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 significant generalization gradient, by contrast with the case where testing used a green key. This effect was interpreted to be one of "cue utilization during testing rather than cue selection during learning,"

since somehow removing green during testing unmasks prior learning on the orientation dimension.

Honig (1969) used a blue key as S+ and a green key as S-. This was followed by dimensional acquisition with three dark vertical lines on a white key. Generalization testing was on the orientation dimension. This paradigm was called a true discrimination (TD) experiment. By contrast, another group of pigeons was rewarded half the time on the blue key and half the time on the green key before dimensional acquisition with the three vertical lines and generalization testing on the orientation dimension. This paradigm was called a pseudodiscrimination (PD) experiment. The generalization gradient was marked in the TD case, but flat in the PD case.

F. Freeman (unpublished master's thesis, Kent State University, Kent, Ohio, 1967) modified this experiment by training pigeons to peck at a vertical line on a dark key (S+) but not to peck at a line tilted at 120° on the same dark background (S-). Then dimensional acquisition with the vertical line on a green background was followed by generalization testing on the dimension of color. A steeper color gradient was found than in the absence of prior discrimination training on S-. This is an example of *enhancement* due to prior discrimination training, rather than blocking. Blocking can also be achieved, as Mackintosh and Honig showed (Honig, 1970). They trained pigeons with S+ and S- as above. Then they retrained them with two spectral values (501 and 675 nm) redundantly added after the animals had reached criterion. Control groups received only the second stage of training. A generalization test on four spectral values demonstrated steeper gradients for the control group.

#### E. PEAK SHIFT AND BEHAVIORAL CONTRAST

Let a pigeon be trained to peck at a key illuminated by a 550 nm light (S+) but not to peck at a key illuminated by a light of  $x$  nm (S-), where  $x$  is chosen greater than 550 for definiteness. If the pigeon makes some errors in learning this discrimination, then it will, on test trials, peck most vigorously at a key lit by a light of  $y(x)$  nm, where typically  $y(x) \neq 550$ ,  $y(x) < 550$  if  $|x - 550|$  is sufficiently small, and  $y(x)$  tends to increase as  $x$  increases (Hanson, 1959). This shift does not occur if the pigeon learns the discrimination without making errors (Terrace, 1966).

In the same experimental setting, the influence of error-filled training at  $x$  nm can increase the rate of pecking at 550 nm if  $|x - 550|$  is sufficiently large ("behavioral contrast") (Hanson, 1959; Bloomfield, 1966). These effects do not occur if the training is errorless (Terrace, 1966), and behavioral contrast disappears after long training sessions (Terrace, 1966).

Honig (1962) has noted that the peak shift occurs only if the S+ and S- are presented successively, but not if they are presented simultaneously. Grusec (1968) has shown that after errorless discrimination training, pairing a shock with the S- will create a peak shift. Bower (1966) has suggested that such contrast effects are due to frustration. Bloomfield (1969) has attempted to unify these results by stating that an "unexpected change for the worse" yields contrast and peak shift effects. Such changes include a sudden reduction in the frequency of reinforcement, or the introduction of shock.

#### F. ORIENTING REACTION VS DISCRIMINATIVE CUES

The frustrative reaction is but one case of a general theme; namely, why can O's responses to a fixed unexpected, or novel, event be different in different contexts? For example, suppose that a human subject sits before a lever with no prior training and that a loud noise occurs abruptly to the left of the subject. There will ensue a strong tendency for the subject to orient toward the noise by turning his head to the left (Luria and Homskaya, 1970). By contrast, suppose that the subject is taught that the noise is a discriminative cue for rapidly pressing the lever to receive a valuable reward. Then the orienting reaction can be replaced by a rapid lever press. How does conditioning redirect the internal flow of activity that would otherwise activate the orienting reaction (Lynn, 1966)?

The orienting reaction is a form of attentional mechanism, but not the only one. For example, novel stimuli can attract more attention than non-novel stimuli even if the stimuli are presented tachistoscopically (Berlyne, 1970; P. McDonnell, unpublished doctoral thesis, University of Toronto, 1968; Trabasso and Bower, 1968). We will distinguish between the two types of reaction in the mechanisms to be described below.

#### G. NOVEL EVENTS AS CONTEXT-DEPENDENT REINFORCERS

As we noted above, frustration can follow the nonoccurrence of an expected reward; thus, if a sequence of events motivated by a given positive drive is unexpectedly interrupted, say by nonoccurrence of the reward, then a negative (frustrative) reaction can ensue. By contrast, if the expected reward is replaced by an even more valued reward, then the frustrative reaction can be mitigated; for example, a check for \$1,000,000 might well eliminate the frustration one might feel after opening a refrigerator and noting the absence of an expected apple. In both cases, "surprise" might occur

owing to the unexpectedness of the outcome, but this surprise is channeled differently in the two cases. Indeed, if an event is rewarding to an animal, then the effectiveness of the reward can be increased if it is also novel.

Berlyne (1969) notes that novel events *per se* can be positively rewarding. He shows that a response-contingent change in the intensity of light in a rat's cage can be used to reward bar pressing. We will suggest that the light change enhances the positive incentive-motivation that is motivating the rat during approach and pressing of the bar. This incentive motivation is not necessarily associated with a specific drive, such as hunger, and can merely be the motor arousal mechanism that is used for general approach behavior. Berlyne also notes that an increase in light level can be less rewarding if the animal's arousal level is too high. He suggests that the rewarding value of an indifferent stimulus is an inverted *U* function of its novelty. The inverted *U* is also a function of the animal's arousal level, so that a given novel stimulus can have different reward value if the animal's arousal level is varied. Berlyne distinguishes the existence of an optimal arousal *level* from an optimal arousal *increment* and discusses the relationship between a given arousal level and its optimal arousal increment in terms of the inverted *U*. Our model discusses related mechanisms of arousal with the property that various types of abnormal behavior can be elicited by overarousal; cf. a schizophrenic's difficulty in paying attention, or seizure activity.

In summary, we will suggest that the nonspecific neural activity generated by a novel event filters through all internal drive representations. The effect of this activity on behavior will depend on the pattern, or context, of activity in all these representations when the novel event occurs. Sometimes the novel event can enhance the effect of an ongoing drive, sometimes it can cause a reversal in sign (as in the frustrative reaction), and sometimes it can introduce and enhance the effect of a different drive. We will be led to assume that every novel event has the capacity to activate orienting reactions, but whether it does or not depends on competition from the drive loci which the event also activates. The nonspecific activity generated by the novel event will also be assumed to reach internal sensory representations, where it helps determine which cues will enter short-term memory to influence the pattern of internal discriminatory and learning processes.

#### H. MOTIVATION AND GENERALIZATION

Increasing an animal's motivation during learning and performance can flatten its gradient during performance (Bersh *et al.*, 1956; Jenkins *et al.*, 1958; Kimble, 1961). By contrast, let a pigeon be trained to peck a key for food, and then trained using a 1000 cps tone as a warning for electric

shock. On testing trials, its generalization gradient for response suppression as a function of tonal frequency is steeper if the pigeon is hungrier (Hoffman, 1969). Note that in this experiment two drives (hunger and fear) compete, whereas in the experiments describing flattening of generalization gradients, only one drive is operative.

### I. PREDICTABILITY AND ULCERS

Weiss (1971a,b,c) has carefully studied the influence of several parameters on the development of stomach ulcers in rats. In his experiments, some rats can escape tail shock by turning a wheel. Each turn of the wheel delays the next onset of shock by a fixed amount of time. In some studies, each shock is preceded by a warning signal. In other studies, each wheel turn is followed either by a tone or by a brief shock, but not both. In each study, there is a control group that is not shocked, and a yoked group that is shocked whenever the animals capable of avoiding or escaping the shock are shocked. The yoked group also hears the tone whenever the avoidance-escape group does. Weiss shows that (a) avoidance-escape subjects develop less ulceration than do the yoked animals; (b) a warning signal reduces the ulceration of both groups of rats; (c) the yoked animals develop less severe ulcers than the avoidance-escape animals if both groups receive a brief shock after each avoidance-escape response; and (d) little ulceration develops in the avoidance-escape group, even if no warning signal precedes shock, if each avoidance-escape response is followed by a feedback stimulus, such as a tone.

Weiss concludes from these results that two main factors contribute to the development of ulcers: the number of *coping responses* that an animal makes, and the amount of *relevant feedback* that these coping responses produce. As the number of coping responses increases, the tendency to ulcerate also increases; but as the relevant feedback increases, the tendency to ulcerate decreases. For example, in (d), the avoidance-escape animals can make many coping responses, but they also receive a high level of relevant feedback, since each successful response is followed by a feedback stimulus that predicts an interval free from shock. In (c), the avoidance-escape animals receive low relevant feedback, since they are shocked for coping. We will find that the magnitude of negative incentive-motivation in our model is a monotone increasing function of the amounts of ulceration that are described in (a)-(d). A rebound from a source of net positive incentive motivation to a source of net negative incentive motivation produces the frustrative reaction in our model. This positive source is capable of motivating consummatory motor activity. The negative source linked with it is not the same as the source of fear. Thus our results do not imply that amounts

of fear equal to the amounts of negative incentive produced by the rebound will have the same effects on ulceration. They suggest, rather, that properties of the negative rebound source are triggered in parallel with, or themselves trigger, ulcerogenic agents.

#### J. ANATOMY AND PHYSIOLOGY

The networks will contain several functionally distinct regions. The interactions between these regions call to mind familiar anatomical facts. It will be apparent that the network regions are not presumed to be exact replicas of real anatomical fragments. Nonetheless, the anatomical relationships between the network regions, as well as their functional roles in total network processing, suggest natural analogs with real anatomies. These analogs will be pointed out both to suggest possible new insights about the functioning of real anatomies, and to serve as an interpretive marker for the networks that will arise in the future from additional postulates. The psychological validity of formal network interactions is, however, independent of how well we guess neuroanatomical labels for network components at this stage of theorizing, since the formal anatomy is still, at best, a lumped version of a real anatomy.

A network region of particular interest is reminiscent of the hippocampus. This region supplies motivational feedback to several other network areas (Olds, 1969). This feedback is determined by a competition between channels corresponding to different drives. Each channel is influenced by sensory and drive inputs. The sensory pathways can be strengthened or weakened by reinforcing events ("conditioned reinforcers"). If a given channel has a prepotent combination of input from conditioned reinforcers and drive, it will suppress other channels using its on-center off-surround anatomy (Anderson *et al.*, 1969; Grossberg, 1973). This feedback has at least three functions. It supplies signals to the region where the sensory pathways are being conditioned by reinforcing events. These signals help to determine the pattern of motivational activity that the sensory pathways will learn. Thus the mock-hippocampus receives input from a region that is implicated in reinforcement, and delivers feedback to this region. We therefore (undogmatically) interpret this second region as a mock-septum (Raisman *et al.*, 1966). The mock-hippocampus also supplies conditionable nonspecific feedback, in the form of a late, slow potential shift, to sensory processing areas (e.g., mock-neocortex) of the network. This feedback, which is related to the network's arousal, drive, reinforcement, and motivational mechanisms, helps to determine which cues will be attended to by the network. An analogous wave, the contingent negative variation (CNV), has been reported

*in vivo* (Walter, 1964). Finally, the mock-hippocampus controls a feedback pathway that helps to regulate the degree of motor arousal or suppression.

If the mock-hippocampus is removed, then transfer of short-term memory into long-term memory is prevented, and difficulties in paying attention will ensue (Milner, 1958).

The mock-septum is influenced by a source of drive input (mock-hypothalamus) and of nonspecific arousal (mock-reticular formation). The level of nonspecific arousal is modulated by the degree of unexpectedness of external events. A mechanism whose motor command cells can be preset to fire only in response to expected events has been synthesized and has an anatomy reminiscent of cerebellar interactions (Grossberg, 1972a). This mechanism projects to the mock-reticular formation. Thus, although the arousal itself is nonspecific, its regulation can be dependent upon specific sensory cues. The nonspecific arousal filters through the drive-representing channels, and can either contrast enhance their activity, or cause a positive (negative) motivational bias to flip into a negative (positive) motivational bias. Thus nonspecific arousal can have specific effects on the pattern of motivational feedback. The nonspecific arousal also feeds into sensory processing areas (e.g., mock-neocortex), where it influences which cues will generate enough neural activity to reverberate in short-term memory, and thereupon be able to influence processes of learning and discrimination. The nonspecific arousal that is triggered by unexpected events differs from the nonspecific conditionable feedback that is related to network drive, reinforcement, and motivational levels. Indeed, these two input sources can compete with each other in overshadowing experiments.

In summary, at least two major feedback loops exist in the network. One feeds between external sensory and internal sensory (e.g., drive) processing areas (cortex → hippocampus → cortex). The other feeds within the internal sensory processing areas (septum → hippocampus → septum).

The drive representations are organized into dipoles, such that each dipole controls a positive and a negative incentive motivational channel; e.g., relief and fear, hunger and frustration. The regulation of motivational output from the dipoles, and of learning based on this output, has been interpreted as using two distinct transmitter systems, which are presumed to be analogous to adrenergic and cholinergic transmitters (Grossberg, 1972c). The need to synchronize the activity of the two parallel channels in a given dipole, and to sample the resultant activity in both dipole channels, suggests that the two transmitter systems are also organized in parallel across the two channels. The organization of drives into dipoles can induce a formal "poker-chip" organization in the input source that feeds them non-specific arousal. A poker-chip anatomy for the reticular formation has been described (Scheibel and Scheibel, 1967).

## II. Drives, Rewards, Motivation, and Habits

The model is an extension of a previous model that has been derived from psychological postulates (Grossberg, 1969a; 1971a, 1972b,c, 1974). This extension is the result of imposing more postulates. The old postulates describe basic properties of classical conditioning, yet the mechanisms that arise can also be used to discuss aspects of instrumental conditioning. The main postulates are described in Grossberg (1974). Two of these postulates are, for example, that (1) the time lags between CS and UCS on successive learning trials can differ; and (2) after learning has occurred, the CS can elicit the UCR (or UCR-like event) in the absence of the UCS. Such obvious facts seem innocent enough; yet when several of them are taken together, and are translated into a rigorous mathematical description, the ensuing neural networks are capable of surprisingly subtle behavior. A heuristic discussion of various mathematical properties of these networks can be found in Grossberg (1974). Some mathematical theorems are proved in Grossberg (1972d, 1973). A review of relevant network properties is given below in several stages. Consider Fig. 1.

In Fig. 1a, the  $i$ th conditioned stimulus ( $CS_i$ ) among  $n$  possible stimuli excites the cell population  $U_{i1}$  of its sensory representation. In particular,  $CS_i$  has already been filtered on its way from the sensory periphery of the network to  $U_{i1}$ , so that it reliably excites  $U_{i1}$  but not irrelevant cells. Some mechanisms of sensory filtering (i.e., pattern discrimination) are derived in Grossberg (1970) and extended in Grossberg (1972a). Sensory representations will be denoted generically by  $\mathcal{S}$ . In response to the  $CS_i$  input,  $U_{i1}$  sends signals to stage  $U_{i2}$  of the  $i$ th sensory representation, as well as toward all the populations  $\alpha = (\alpha_h, \alpha_f^+, \alpha_f^-, \dots)$  of arousal cells. (In this presentation, we ignore effects due to spatial gradients in interaction strength.) Thus the

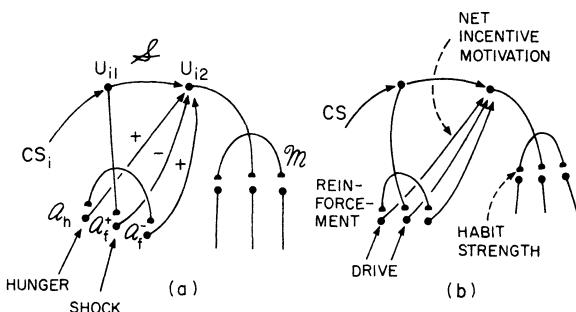


FIG. 1. Interaction of reinforcement, drive, motivation, and habit strength in minimal network.

$\mathcal{S} \rightarrow \mathcal{Q}$  pathways are “nonspecific,” whereas the  $\mathcal{S} \rightarrow \mathcal{S}$  pathways are “specific.” The arousal cells  $\mathcal{Q}_h$  subserve the hunger drive, cells  $\mathcal{Q}_f^+$  subserve fear, and cells  $\mathcal{Q}_f^-$  subserve relief from fear. The cells  $\mathcal{Q}_h$  receive an internally generated drive input that is a monotone increasing function of hunger level. The cells  $\mathcal{Q}_f^+$  receive an input that is a monotone increasing function of shock level. Offset of shock elicits transient excitatory activity in the relief center  $\mathcal{Q}_f^+$  (Denny, 1970).

Signals from the sources  $\mathcal{Q}$  are generated by activity at these sources and are, other things equal, monotone-increasing functions of this activity. The signals from  $\mathcal{Q}_h$  and  $\mathcal{Q}_f^-$  to all  $U_{i2}$  populations are excitatory, whereas the signals from  $\mathcal{Q}_f^+$  to all  $U_{i2}$  populations are inhibitory. Since a signal from a population in  $\mathcal{Q}$  is sent to *all* populations  $U_{i2}$ , the pathway  $\mathcal{Q} \rightarrow \mathcal{S}$  is non-specific.  $U_{i2}$  can send signals to  $\mathcal{M}$  only if it simultaneously receives a large signal from  $U_{i1}$  and a large net excitatory signal from  $\mathcal{Q}$ . In particular, a large excitatory  $\mathcal{Q}_h \rightarrow U_{i2}$  signal can be canceled by a large inhibitory  $\mathcal{Q}_f^+ \rightarrow U_{i2}$  signal, which thus prevents  $U_{i2}$  from firing even if  $CS_i$  is present. In this way, consummatory activity compatible with hunger can be suppressed by shock.

Suppose that shock is terminated by an avoidance response, or AR. (Learned escape responses can become avoidance responses; hence we use only the term “avoidance” below, for simplicity.) Then  $\mathcal{Q}_f^-$  is excited and creates a large, but transient, excitatory  $\mathcal{Q}_f^- \rightarrow \mathcal{S}$  signal to all sensory representations. Sensory feedback cues of the AR also excite particular sensory representations, which we denote by  $\mathcal{S}(\text{AR})$ . The  $U_{i2}$  stages of  $\mathcal{S}(\text{AR})$  cells thus receive  $U_{i1}$  and  $\mathcal{Q}_f^-$  inputs. They can therefore fire and send signals to  $\mathcal{M}$ . Cells in  $\mathcal{S}$  that receive only the  $\mathcal{Q}_f^-$  input cannot fire.

Changes in long-term memory (LTM) can occur at two locations in this picture: at the  $\mathcal{S} \rightarrow \mathcal{Q}$  synaptic knobs, and at the  $\mathcal{S} \rightarrow \mathcal{M}$  synaptic knobs. The unit of LTM is a *spatial pattern*: the relative activities of all the long-term memory traces in the synaptic knobs of a given population. The  $U_{i1} \rightarrow \mathcal{Q}$  synaptic knobs and the  $U_{i2} \rightarrow \mathcal{M}$  synaptic knobs can learn (“sample”) patterns of activity playing on the populations  $\mathcal{Q}$  and  $\mathcal{M}$ , respectively, *only* when these knobs are activated by  $U_{i1}$  or  $U_{i2}$  signals. The  $U_{i1} \rightarrow \mathcal{Q}$  synaptic knobs encode a weighted average of the “motivational” patterns that are sequentially presented to  $\mathcal{Q}$  populations when these knobs are sampling. The  $U_{i2} \rightarrow \mathcal{M}$  synaptic knobs encode a weighted average of the “motor” patterns playing on the motor control cells  $\mathcal{M}$  when these knobs are sampling. (The cells  $\mathcal{M}$  need not be motor controllers; they can also be an arbitrary collection of sensory cells, for example.) Thus when the cells in the  $\mathcal{S}(\text{AR})$  representations are active after an avoidance response, their  $U_{i1}$  stages can sample “relief” at  $\mathcal{Q}_f^-$ , and their  $U_{i2}$  stages can sample the motor pattern corresponding to the AR. On a recall trial, the presence of

avoidance or escape cues, such as seeing the wheel that delays or terminates shock when turned, activates  $\mathcal{S}(\text{AR})$ . Signals  $\mathcal{S}(\text{AR}) \rightarrow Q_f^- \rightarrow \mathcal{S}$  generate positive feedback which combines with  $\mathcal{S}(\text{AR}) \rightarrow \mathcal{S}(\text{AR})$  signals to activate  $\mathcal{S}(\text{AR}) \rightarrow \mathcal{M}$  synaptic knobs and reproduce (a frame of) the AR. In this sense, the rebound from fear to relief when shock terminates provides the positive motivation for learning escape or avoidance motor acts.

Figure 1b describes network variables in more conventional terms. Rewarding or punishing events change the pattern of activity across populations in  $\mathcal{Q}$ . These reinforcing events are encoded in LTM by the  $\mathcal{S} \rightarrow \mathcal{Q}$  synapses that are sampling at the crucial times. Drive inputs also perturb  $\mathcal{Q}$ .  $\mathcal{Q}$  cells such as  $Q_h$  can fire to  $\mathcal{S}$  only if they receive a sufficiently large drive input *and* an input from a UCS or a conditioned reinforcer. Otherwise, the network would emit persistent eating behavior in response to food in the absence of hunger. In this context, a conditioned reinforcer corresponding to a given drive is a cue whose  $\mathcal{S} \rightarrow \mathcal{Q}$  pathway to that drive locus has become relatively large owing to prior conditioning. The  $\mathcal{Q} \rightarrow \mathcal{S}$  signals provide incentive motivation for modulating the activity in  $U_{i2}$ . If the net incentive motivation to  $U_{i2}$  is sufficiently positive *and*  $\text{CS}_i$  is present, then signals from  $U_{i2} \rightarrow \mathcal{M}$  can occur, sampling at the  $U_{i2} \rightarrow \mathcal{M}$  synaptic knobs is initiated, and the habit strengths of these knobs are influenced by the patterns playing on  $\mathcal{M}$  during the sampling interval.

Given this basic network structure, several refinements must be made to guarantee that the network works well. These refinements eventually lead to mechanisms that are relevant to the phenomena described in the Introduction. The next three sections review refinements that have, at least partially, been discussed in previous papers.

### III. The Rebound from Fear to Relief

How does the offset of shock at  $Q_f^+$  generate a transient rebound of activity at  $Q_f^-$ ? A previous paper (Grossberg, 1972c) analyzes this question and constructs explicit rebound mechanisms. The simplest nonrecurrent (feed-forward) version of this mechanism is shown in Fig. 2, and is described mathematically in the Appendix.

An internally generated, tonic (i.e., persistent) input  $I$  derives both the  $Q_f^+$  and the  $Q_f^-$  channels. This input provides the activity that drives the rebound at  $Q_f^-$  when the phasic shock-derived input  $J$  is shut off. The inputs  $I$  and  $J$  add up at  $v_1$  and create signals along  $e_{13}$ . A smaller signal is created by  $I$  in  $e_{24}$ . At the synaptic knobs  $N_{13}$  and  $N_{24}$ , transmitter is produced at a fixed rate. This rate is inhibited by the transmitter end product at a rate proportional to transmitter concentration. The two processes taken together

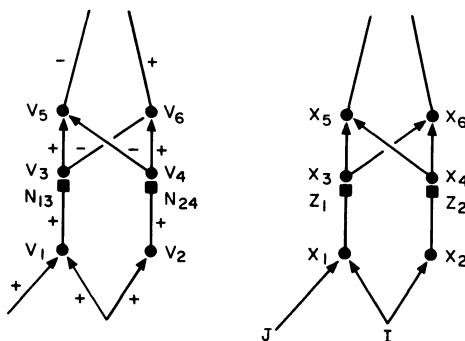


FIG. 2. Nonrecurrent rebound mechanism from fear to relief.

create the tendency for transmitter level in both  $N_{13}$  and  $N_{24}$  to approach a fixed finite upper bound. Transmitter is also removed from  $N_{13}$  at a rate jointly proportional to the signal strength in  $e_{13}$  and the amount of transmitter in  $N_{13}$ . A similar release of transmitter occurs at  $N_{24}$ . Since the signal in  $e_{13}$  is larger than the signal in  $e_{24}$  whenever  $J > 0$ , less transmitter exists in  $N_{13}$  than in  $N_{24}$  when  $J > 0$ . On the other hand, it has been proved that the multiplicative coupling of signal strength to transmitter produces a larger output from  $N_{13}$  than from  $N_{24}$  when  $J > 0$ . Thus when shock is on, the signals emitted by  $v_3$  exceed those emitted by  $v_4$ . Since these signals compete subtractively at  $v_5$  and  $v_6$ , only the output from  $v_5$  is positive when shock is on. That is, only the fear channel supplies incentive motivation to  $\S$ . When shock is turned off, both  $v_1$  and  $v_2$  are driven by the equal tonic input  $I$ . The potentials at  $v_1$  and  $v_2$  rapidly equalize, as do the signals in  $e_{13}$  and  $e_{24}$ . By contrast, the transmitters in  $N_{13}$  and  $N_{24}$  only slowly begin to readjust to the new input levels. The input to  $v_3$  is, however, determined by a multiplicative coupling of the signal in  $e_{13}$  with the transmitter level in  $N_{13}$ . A similar coupling of  $e_{24}$  signal with  $N_{24}$  transmitter determines the input to  $v_4$ . Since the transmitter level in  $N_{24}$  exceeds that in  $N_{13}$ , the input to  $v_4$  exceeds the input to  $v_3$ , and hence only  $v_6$  generates an output. Thus, after shock terminates, only the relief channel is active. Gradually the equal tonic input to  $v_1$  and  $v_2$  equalizes the amount of transmitter in  $N_{13}$  and  $N_{24}$ . The two channels then annihilate each other's equal signals, and no outputs arise from either channel. The relief response is transient because the imbalance in transmitter accumulation caused by shock is gradually eliminated by the uniformly distributed tonic input.

The identification of the accumulation-depletion-release substances in  $e_{13}$  and  $e_{24}$  as transmitters is speculative at present. Grossberg (1972c) cites compatible data. Any process with the same formal properties could do the job,

however; cf., accumulation of bound  $\text{Ca}^{2+}$  and its participation in transmitter release.<sup>2</sup>

This rebound mechanism has technical properties that are relevant to the discussion below. These are the following:

1. Both fear and relief are inverted  $U$  functions of the tonic input level  $I$ . In other words, either underarousal or overarousal depresses emotional affect and incentive motivational feedback in the network. Overaroused depression is stable with respect to sensory inputs; the network is "indifferent" to emotionally charged cues. This is because sensory inputs to the fear or relief channels create only a small asymmetry in the pattern of inputs to these channels when the equal arousal inputs to these channels are large; the large equal arousal inputs tend to saturate the response of the two parallel channels. Thus, after subtractive competition between these channels, their net output is small. Underaroused depression is unstable in the sense that, after the system's elevated thresholds are exceeded by external cues (i.e., there is not enough  $I$  input to exceed threshold in response to small  $J$  inputs), either aversive or rewarding cues can cause overreactive fear or relief responses; network response is emotionally "irritable." This is because sensory inputs to the fear or relief channels create an unusually large asymmetry in the total input to these channels, since the background arousal level is smaller than usual. This phenomenon formally illustrates the paradoxical fact that underarousal can be unusually aversive in some situations and unusually rewarding in others (Berlyne, 1969).

2. There exist levels  $I$  such that maximal relief is greater than maximal fear in response to a prolonged, but then abruptly terminated, fearful cue. In fact, the ratio of maximal relief to maximal fear grows as  $I$  increases. By property (1), however, unduly small or large  $I$  levels create small absolute values of relief or fear. There exist intermediate  $I$  values, however, such that the (relief:fear) ratio is large, and the absolute size of these reactions is also large. This property is needed to make learned avoidance or escape behavior possible in the presence of fearful cues. One needs the guarantee that, although the fear channel is on, the relief channel can be so strongly activated by avoidance or escape dues that it can generate a positive *net* incentive motivational input to  $S$ , and thereby release motor activity that leads to avoidance or escape.

3. Once the transmitter levels have adjusted to a fixed input level, either a sudden decrease in arousal input or a sudden increase in fearful cue input will cause an increase in fear. Similarly, a sudden decrease in input from irrelevant cues (i.e., cues that send equal signals to the two channels) will

<sup>2</sup> Note added in proof: C. D. Wise, B. D. Berger, and L. Stein, *Biol. Psychiatry*, 6, 1 (1973) present data suggesting that a norepinephrine reward system and a serotonin punishment system compete in parallel for relative dominance.

cause an increase in fear. By contrast, a sudden increase in arousal and/or irrelevant cue input, or a sudden decrease in fearful cue input, will tend to create a relief rebound.

4. More relief is generated by shutting off  $J$  units of shock than  $J/2$  units of shock. More relief is generated by shutting off  $J/2$  units of shock than by cutting the shock level from  $J$  units to  $J/2$  units. A relationship exists between the rewarding effect of cutting the shock level in half, the size of the (relief:fear) ratio, and the arousal level (Grossberg, 1972c). This will be extended in Section XI to include the size of the partial reinforcement acquisition effect.

This rebound mechanism is coupled to the learning mechanism in Fig. 3. Sampling by  $\mathcal{S}$  channels occurs at  $v_3$  and  $v_4$  for two reasons: (a) it must occur after the accumulation-depletion step to be able to sample the rebound; (b) it must occur before the subtractive stage in order to ensure that not both fear and relief control behavior at any instant of time. Another reason is given in Section V.

A noncurrent rebound mechanism is not capable of higher-order instrumental conditioning, i.e., of instrumentally motivated "chaining" (Kelleher, 1966). For example, the offset of a cue that was previously paired with shock could not be used to reward escape behavior (Maier *et al.*, 1969). To make higher-order instrumental conditioning possible, the network must be modified so that offset of activity in a conditioned  $\mathcal{S} \rightarrow \alpha_f^+$  channel can drive a rebound from  $\alpha_f^+$  to  $\alpha_f^-$ . In the above example, a cue that was paired with shock has a strong  $\mathcal{S} \rightarrow \alpha_f^+$  pathway. Offset of this cue will reward escape behavior if it elicits a rebound at  $\alpha_f^-$ . Thus  $\mathcal{S}$  must send axons to a stage *prior* to the rebound, and these axons will have conditionable synaptic knobs.

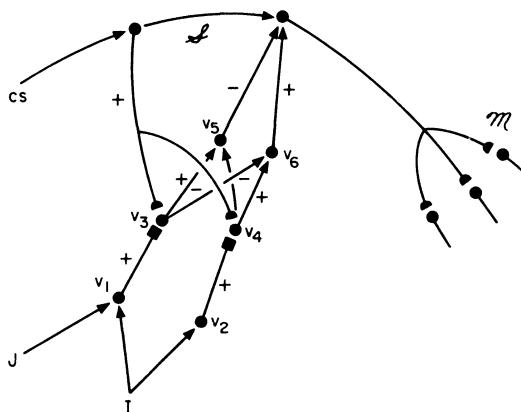


FIG. 3. Interaction of learning and rebound mechanism.

$\S$  must also send its axons to a stage *after* the rebound, so that cues can sample the fear and relief reactions. Thus the anatomy of  $(\alpha_f^+, \alpha_f^-)$  is *recurrent* to guarantee that the rebound occurs both before and after the stage where  $\S$  samples the rebound. See Fig. 4 for some recurrent anatomies.

In Fig. 4a, the cell sites  $v_1$  and  $v_3$  of Fig. 2 are identified, as are the sites  $v_2$  and  $v_4$ . Figure 4c is particularly interesting, since it permits the learning of a stable conditioned-avoidance response that is motivated on performance trials by relief rather than fear (Maier *et al.*, 1969). The tonic source  $I$  is moved downstream from the  $\S \rightarrow \alpha$  sampling axons. There it can drive the rebound, which occurs still further downstream, but it does not counter-condition patterns in the  $\S \rightarrow \alpha$  axons whenever sampling by  $\S$  occurs. The outputs from the rebound stage compete before they are fed back to be sampled by  $\S$ . This feedback is positive *only* if one of the channels is stronger than the other. Thus the tonic input  $I$  alone cannot generate any feedback, and therefore does not countercondition patterns encoded in  $\S \rightarrow \alpha$  synaptic knobs. Irrelevant cues in  $\S$  can, however, countercondition

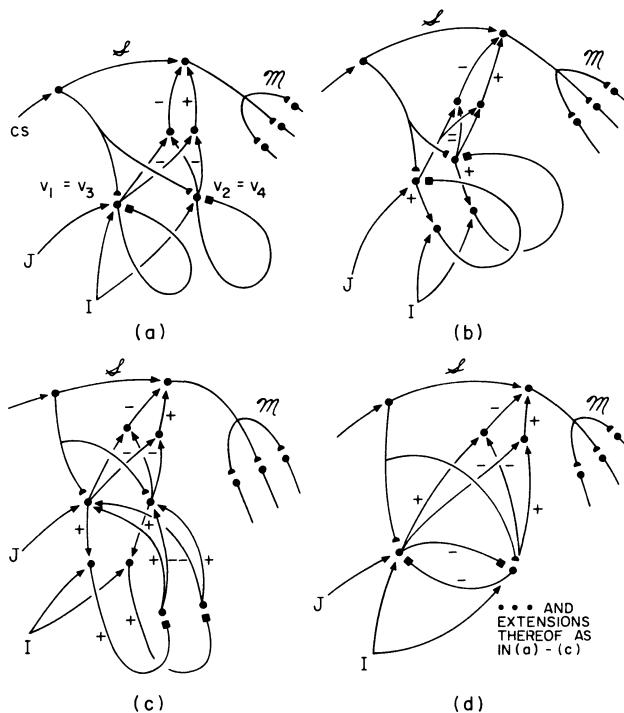


FIG. 4. Some recurrent rebound mechanisms coupled to the learning mechanism.

conditioned  $S \rightarrow Q$  pathways. Such cues send equal signals to  $Q_f^+$  and  $Q_f^-$ , and these signals can extinguish the pattern in other active  $S \rightarrow Q$  pathways by contiguity. Even this source of extinction can be removed by a slight modification of network design: require that *only* the feedback signal along the recurrent loop can cause changes in the long-term memory of the  $S \rightarrow Q$  synaptic knobs. Then neither irrelevant cues nor the tonic input can counter-condition  $S \rightarrow Q$  knobs. Section XX discusses this modification in greater detail. Section IV discusses ways to minimize the possibility of saturating the feedback loop with irrelevant cue and tonic inputs. Of course, if the avoidance or escape response does not remove  $\emptyset$  from sources of fearful cues, then the strong  $S \rightarrow Q_f^+$  connections which these cues control can counter-condition the  $S \rightarrow Q_f^-$  channels that motivate avoidance.

In Grossberg (1972c), the inputs  $I$  and  $J$  add up at  $v_1$ , and their influence decays exponentially through time. Denote the response of  $v_1$  (its activity, stimulus trace, or short-term memory trace) by  $x_1$ . There exists a signal threshold  $\Gamma$  in  $e_{13}$ , such that the signal strength in  $e_{13}$  is zero if  $x_1(t) \leq \Gamma$  and is a linear function of  $x_1(t) - \Gamma$  if  $x_1(t) > \Gamma$ . The inverted  $U$  in the relief channel, as a function of arousal level  $I$ , does not depend on the threshold  $\Gamma$ , but the inverted  $U$  in the fear channel does; in fact, the amount of fear is a decreasing function of  $I$  once  $x_1 \geq \Gamma$ .

*In vivo*, signal functions are not always linear functions of activity above a threshold cutoff. Often they are sigmoid functions of activity (Kernell, 1965a,b; Rall, 1955). Section IV discusses the importance of this property for the processing of neural signals in noise when the network is recurrent. Figure 4 shows that rebound mechanisms often have a recurrent anatomy. We therefore consider how the rebound mechanism is altered by making the output signals in  $e_{13}$  and  $e_{24}$  sigmoid functions of the activity levels in  $v_1$  and  $v_2$ , respectively. The main new effect for present purposes is that, given a fixed level  $J$  of shock, an increase in the arousal level  $I$  can potentiate the fear reaction, even if the activity of  $e_{13}$  is suprathreshold (see the Appendix). This does not happen if the  $e_{13}$  signal is linear, since equal linear increments in the activity of the parallel fear and relief channels do not cause a greater than linear response to  $J$ , but they do tend to saturate the transmitter channels. Figure 5 depicts the main difference for the case of sigmoid signal functions.

Comparing Fig. 5a with Fig. 5b, we see that even if the equal arousal inputs in the parallel channels cancel at  $v_5$  and  $v_6$ , nonetheless the response to  $J$  can be larger when it accompanies a larger arousal level. Figure 5c shows that the  $J$  response can also be smaller if the network is “overaroused.” This overarousal effect can become confounded with the saturation of transmitter levels in  $N_{13}$  and  $N_{24}$ . Saturation yields an overarousal effect even if the signals in  $e_{13}$  and  $e_{24}$  are linear. Both effects cause depression of incentive motivation due to subtractive competition of signals at  $v_5$  and  $v_6$ .

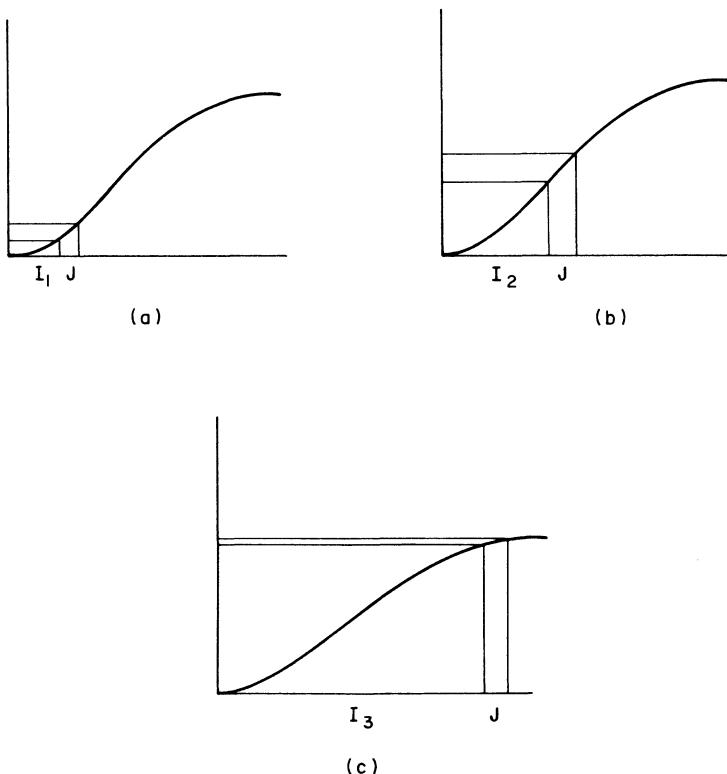


FIG. 5. Potentiation or compression of response to phasic input as arousal level varies.

The interaction of arousal level  $I$  and phasic increment  $J$  in Fig. 5 recalls Berlyne's (1969) discussion of the interaction between arousal level and optimal arousal increment. In Section XV, the effect will be used to discuss how a novel light change can reinforce lever pressing.

#### IV. Short-Term Memory and Total Activity Normalization

Several constraints must be placed on  $\mathcal{S} \rightarrow \mathcal{Q}$  signals. All these constraints can be guaranteed by the same mechanism, which is analyzed in Grossberg (1973). These constraints are listed below. They amount to predictions about the global behavior of recurrent on-center off-surround anatomies undergoing shunting interactions.

1. The *total*  $S \rightarrow Q$  output must have an upper bound that is independent of the number and intensity of  $S \rightarrow Q$  channels that are active at any time. Otherwise the  $S \rightarrow Q$  channels, in the absence of drive inputs, could activate  $Q \rightarrow S$  feedback. In such a network, persistent feeding could occur in the absence of hunger. Similarly, a *single* active  $S \rightarrow Q$  channel, combined with a prepotent drive input, should be able to activate  $Q \rightarrow S$  feedback, even though the drive input alone should not be able to do so. Thus the total activity of  $S$ , rather than just its upper bound, must be carefully regulated. Such regulation can also prevent the feedback loop in the rebound mechanism of Section III from being saturated by irrelevant cues. We call such regulation of total activity *normalization*.

2. Consider an experiment in which lever pressing preceded by a tone is rewarded, whereas lever pressing in the absence of the tone is punished. What mechanism keeps the internal representations of the tone and the lever press active in  $S$  until the reward or punishment can alter the probability of lever pressing in response to the tone-plus-cues of the lever, as opposed to cues of the lever alone? A mechanism for storing these representations in short-term memory (STM) is needed.

3. Consider the processing of a pattern of inputs delivered to an ensemble of noninteracting cell populations. Suppose that neural noise exists in these populations. In many neural systems, noise cannot be avoided, if only because they operate near the quantum range, as in the case of sensory systems. In this context, if the input signals are too small, they can be lost in the noise. If they are too large, they can saturate their respective populations, thereby creating a uniform pattern of excitation across populations and destroying all information about the input pattern. To avoid these extremes in the noninteracting case, input intensities would have to be restricted to a narrow range, and the ability to process arbitrary patterns with fluctuating input intensities would be lost.

The following mechanism regulates total network activity, is capable of STM, quenches network noise, and permits the effective processing of arbitrary input patterns without saturation. The simplest example of this mechanism will be reviewed below. It is a network with a recurrent on-center off-surround anatomy whose interactions are of shunting type. The network is defined by the system

$$\dot{x}_i = -Ax_i + (B - x_i)f(x_i) - x_i \sum_{k \neq i} f(x_k) + I_i \quad (1)$$

where  $i = 1, 2, \dots, n$ , and  $x_i (\leq B)$  is the mean activity of the  $i$ th cell, or cell population,  $v_i$  of the network. Four effects determine this system: (a) exponential decay, via the term  $-Ax_i$ ; (b) shunting self-excitation, via the term  $(B - x_i)f(x_i)$ ; (c) shunting inhibition of other populations, via the term  $-x_i \sum_{k \neq i} f(x_k)$ ; and (d) externally applied inputs, via the term  $I_i$ . The

function  $f(w)$  describes the mean output signal of a given population as a function of its mean activity  $w$ . *In vivo*,  $f(w)$  is often sigmoid function of  $w$  (Kernell, 1965a,b; Rall, 1955). This is an important property of the above model for the effective processing of signals in noise.

The system in (a) can be formally motivated as follows. Consider  $n$  states  $v_i$  whose responses  $x_i(t)$  to inputs  $I_i(t)$  are linear, return to equilibrium (say 0) in the absence of inputs, and have a finite maximum (say  $B$ ). Then  $\dot{x}_i = -Ax_i + (B - x_i)I_i$ . Term  $(B - x_i)I_i$  says that inactive sites become activated at a rate jointly proportional to the number of inactive sites and the excitatory input size. At equilibrium,  $x_i = BI_i(A + I_i)^{-1}$ , which approaches  $B$  as  $I_i$  becomes large. This system saturates and is not normalized. Both these difficulties vanish if an off-surround with shunting interaction exists. Then

$$\dot{x}_i = -Ax_i + (B - x_i)I_i - x_i \sum_{k \neq i} I_k$$

The new term says that active sites become deactivated at a rate jointly proportional to the number of active sites and the inhibitory input size. At equilibrium,  $x_i = BI_i(A + I)^{-1}$ , where  $I = \sum_k I_k$ . Letting  $\theta_i = I_i I^{-1}$ , we find  $x_i = \theta_i BI(A + I)^{-1}$ . The activity  $x_i$  is proportional to  $\theta_i$  no matter how large  $I$  becomes, and the total activity  $x = \sum_k x_k$  never exceeds  $B$ . Such a system, however, is not capable of STM. The anatomy is made recurrent by replacing each  $I_i(t)$  by  $f(x_i)$ . External inputs are then added on to get (a).

The STM capabilities of recurrent networks carry with them possible difficulties. If these networks can reverberate patterns imposed by external inputs, then why do they not also reverberate their own noise indefinitely, thereby flooding the network with its own noise? The answer is that they do, if the signal function  $f(w)$  is improperly chosen. For example, if  $f(w)$  is a linear function of  $w$ , or a function that grows slower than linearly, such as  $f(w) = w(1 + w)^{-1}$ , then noise, even in the absence of signals, will be amplified and reverberated. If  $f(w)$  grows faster than linearly, such as  $f(w) = w^2$ , then this problem is avoided. Sufficiently small noise values will dissipate through time. If a brief, but sufficiently intense, input pattern is imposed on the noise, however, then two things happen.

First, all populations which receive the largest input in the pattern will suppress the activity in all other populations, including the noise. Second, if the function  $g(w) = w^{-1}f(w)$  is convex, then *normalization* occurs: the total activity  $x(t) = \sum_{k=1}^n x_k(t)$  of all the populations approaches a fixed positive limit through time. The first property shows that an extreme form of contrast enhancement occurs: only the peaks of the input pattern survive. If one population of the network receives more input than any other, then the network “chooses” this population and quenches all others. The second property shows that the system precisely regulates its total activity and can

store the activity of certain populations indefinitely in STM by reverberating their activity through excitatory recurrent interneuronal loops.

The first property is too strong: too much of the pattern is suppressed in the attempt to suppress the noise. How can this be avoided? The way is to choose  $f(w)$  so that it grows faster than linearly for small values of  $w$ , and (approximately) linearly at larger values of  $w$ . Then noise dissipates, and there exists a *quenching threshold*. This means that, given a sufficiently energetic pattern of inputs, the activities of populations which fall below the threshold are quenched (including noise) and those which fall above the threshold are contrast enhanced and stored in STM. Speaking intuitively, this is due to three interacting effects. The pattern is contrast enhanced for activities at which  $f(w)$  grows faster than linear, but is preserved for activities at which  $f(w)$  is linear. Normalization of activity can drive the system from the first to the second region of  $f(w)$  growth. Thus, a pattern that is partially contrast-enhanced at small activity levels is preserved after it is normalized in the linear  $f(w)$  range. Various applications of this system, and its mathematical properties, are found in Grossberg (1973). An important property of the system is shown in Fig. 6.

The function  $g(w)$  alluded in Fig. 6 is defined by  $g(w) = w^{-1}f(w)$ ;  $g(w)$  tests how the signal  $f(w)$  deviates from a linear function as the activity level  $w$  increases. For example, if  $g(w)$  increases for small values of  $w$  and decreases for large values of  $w$ , then  $f(w)$  will be a sigmoid function of  $w$ . The arrows in Fig. 6 depict the direction in which the total activity  $x(t) = \sum_{k=1}^n x_k(t)$  will change if it falls in a given region between two adjacent vertical dotted lines. In Fig. 6a,  $g(w)$  is convex, and only one stable equilibrium point exists. Either  $x(t) \rightarrow 0$  as  $t \rightarrow \infty$  if  $x(0)$  is small, or  $x(t)$  approaches the unique limit  $E_1$ . In Fig. 6b,  $g(w)$  is not convex, and two positive limits  $E_1$  and  $E_2$  for  $x(t)$  exist. This property motivates the following possibility, which is illustrated in Fig. 7.

In Fig. 7, a nonspecific arousal input  $J_A$  combines with a specific input  $J_i$  at each population  $v_i$ . Two important cases arise. In case 1,  $J_A$  and  $J_i$  combine multiplicatively to influence the activity level  $x_i$ . Input  $J_A$  is said to shunt the activity level (Grossberg, 1973b). In case 2,  $J_A$  and  $J_i$  combine additively to influence the activity level  $x_i$ . Consider case 1 for definiteness. Then the input  $J_A$  does not change the *relative* input levels to the various populations. (In case 2, a large  $J_A$  tends to uniformize any pattern of  $J_i$ 's.) Let  $J_A$  be parametrically increased to ever higher levels. One hereby increases the number of populations that receive enough input to exceed the quenching threshold and are stored in STM. Conversely, reducing  $J_A$  decreases the number of populations that will be stored. Thus, given an input pattern in which many inputs are close to each other in relative size, one way to "make a choice" between populations is to lower the arousal level

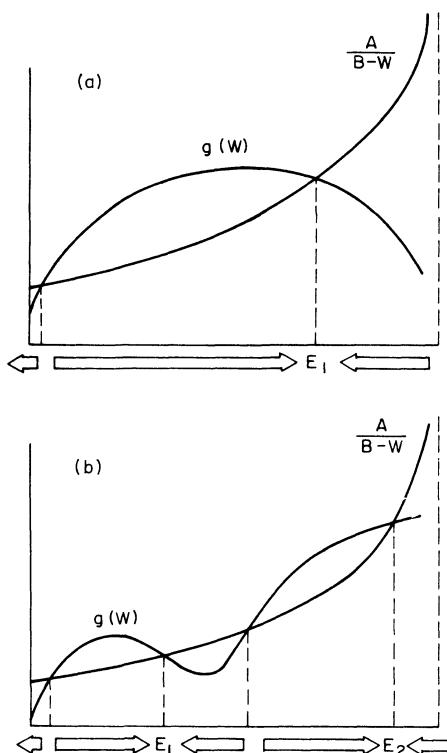


FIG. 6. Stable limit points of total activity.

of the input until only one population exceeds the quenching threshold; in common parlance, put the network in a quiet place. By contrast, one way to make as many cues as possible relevant to further network processing is to substantially increase the arousal level. Thus, suppose that a "novel" stimulus excites the network's nonspecific arousal source. Then all recently presented cues can have their network representations brought into STM to play a part in further network processing, including the sampling and subsequent learning of motor responses (Grossberg, 1972b). In this way, novel or unpredictable events can bring all possible information about presently available cues into an active state, to enhance the network's ability to deal with the unexpected situation.

A similar effect could be achieved if an increase in arousal lowered the quenching threshold by, say, decreasing the signal strength in the inhibitory

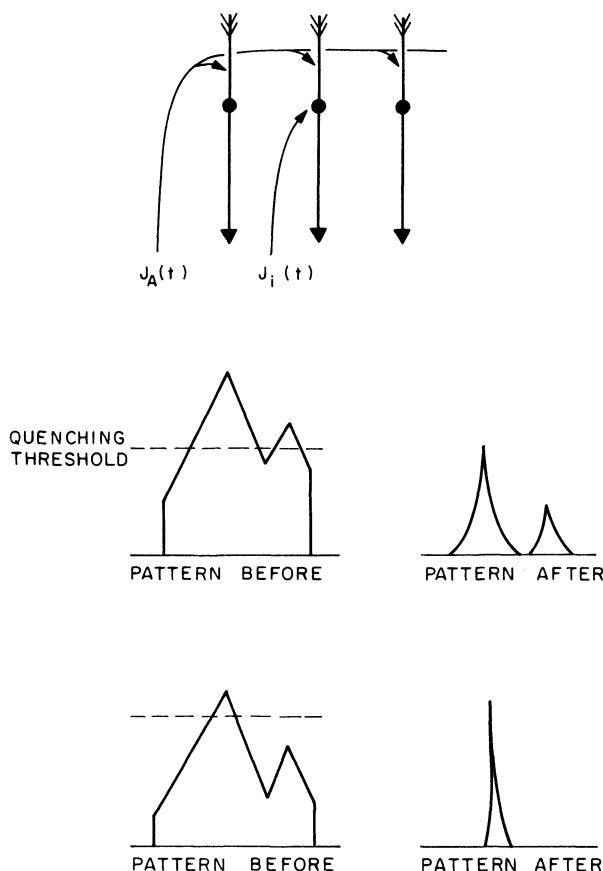


FIG. 7. Influence of arousal on final pattern stored in short-term memory.

off-surround using arousal-initiated inhibition of the inhibitory interneurons. Grossberg (1973) discusses how such a network can be thrown into "seizure" under abnormal circumstances.

Thus, we suppose that the cell populations in § are interconnected by a recurrent on-center off-surround anatomy whose interactions are of shunting type. This anatomy does not, of course, accomplish all the tasks that living sensory processors undertake. For example, it does not generate a sequential STM buffer (Atkinson and Shiffrin, 1968); it has no hierarchical structure. A later paper will investigate this extension in a setting that preserves properties (1)-(3) above.

### V. Sensory-Drive Hierarchy

Several constraints must be placed on  $\alpha \rightarrow \beta$  feedback. These constraints are the following:

1. The *total*  $\alpha \rightarrow \beta$  output must have an upper bound that is independent of the number and intensity of  $\alpha \rightarrow \beta$  channels that are active at any time. Otherwise, since the  $\alpha \rightarrow \beta$  channel is nonspecific, it could activate  $\beta \rightarrow \mathcal{M}$  sampling by  $\beta$  channels that receive no sensory inputs. Precise regulation of total  $\alpha \rightarrow \beta$  output would also provide a steady baseline of incentive motivation to activate compatible motor activity.
2. Consider the situation in which a student regularly eats meals in spite of the prolonged absence of a sexual partner. A positive, but nonprepotent, drive can control motor behavior in the presence of compatible sensory cues (e.g., eating food if hungry), if cues compatible with the prepotent drive are unavailable (e.g., absence of a sexual partner).
3. Simultaneous  $\alpha \rightarrow \beta$  feedback from two or more incompatible drives must be prevented to avoid the occurrence of incompatible motor commands.

Property (3) can be achieved by any mechanism that can make a choice among  $n$  populations. For example, a recurrent on-center off-surround network with shunting interactions can achieve this if its quenching threshold is sufficiently large. A nonrecurrent on-center off-surround network with additive interactions can also achieve this (Grossberg, 1970). The system

$$\dot{x}_i = -Ax_i + I_i - \sum_{k \neq i} I_k$$

with inputs  $I_i$ ,  $i = 1, 2, \dots, n$ , is of this type. At any given time, only the population  $v_i$  whose input  $I_i$  is maximal receives a net positive total input ( $I_i - \sum_{k \neq i} I_k$ ) =  $2I(\theta_i - \frac{1}{2})$ . All other populations receive negative inputs that drive their activity to subthreshold values. The nonrecurrent mechanism is incapable of STM. It is driven, however, by signals from  $\beta$  which are capable of STM. Thus, the outputs from a nonrecurrent mechanism that chooses among  $\alpha \rightarrow \beta$  signals can be sustained by the STM reverberation in  $\beta$  that drives  $\beta \rightarrow \alpha$  signals.

Property (2) requires that signals from sensory cues and from drive inputs combine before the choice mechanism of property (3) determines  $\alpha \rightarrow \beta$  feedback.  $\alpha \rightarrow \beta$  feedback is then determined by the dominant combination of sensory-plus-drive cues (Fig. 8a) rather than by the dominant drive level (Fig. 8b). Figure 8a shows that a sensory-drive hierarchy can be established if the normalizer occurs after the stage at which sensory and drive inputs mix, after the stage at which  $\beta \rightarrow \alpha$  sampling occurs, after the stage at which a relief rebound occurs, and before  $\alpha \rightarrow \beta$  feedback signals can influence  $\beta$ . The normalizer determines a consensus between *all* the possible  $\alpha \rightarrow \beta$  feed-

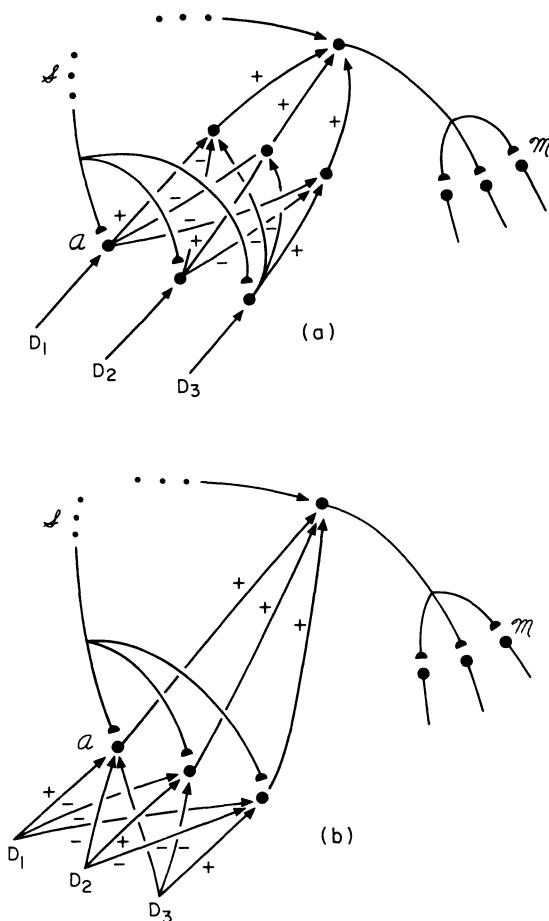


FIG. 8. Sensory-drive heterarchy vs drive hierarchy.

back channels, and thus its inhibitory interactions cut across the channels controlled by different drives.

Property (1) can be achieved by a recurrent on-center off-surround network with shunting interactions. This mechanism also regulates total activity of  $\alpha \rightarrow \gamma$  feedback, and therefore provides a steady baseline of incentive motivation to activate compatible motor activity. Given a nonrecurrent choice mechanism, total activity is bounded from above, but its precise regulation can be achieved only indirectly by the regulation of total  $\gamma \rightarrow \alpha$  output.

### VI. Conditionable $\alpha \rightarrow \beta$ Feedback and Psychological Set

The following unfortunate phenomenon can occur in the network thus far discussed. Suppose that a particular UCR (e.g., salivation) is encoded in  $\beta \rightarrow \mathcal{N}$  synaptic knobs when a given CS (e.g., ringing bell) and a UCS<sub>1</sub> (e.g., smell of food) compatible with drive  $D_1$  (e.g., hunger) is active. Suppose on performance trials that a different drive  $D_2$  (e.g., sex) is stronger than  $D_1$ ; indeed, suppose that  $D_1$  has been satisfied, for definiteness. Then the UCR can be released if a UCS<sub>2</sub> (e.g., smell of mate) compatible with  $D_2$  is presented along with the CS. Such a network can release persistent eating behavior in the absence of hunger if it is sexually aroused by sexual cues other than a mate.

This difficulty is due to the fact that  $\alpha \rightarrow \beta$  feedback in response to any given drive is nonspecific. On performance trials, UCS<sub>2</sub> supplies sensory input that combines with  $D_2$  drive input to release nonspecific  $\alpha \rightarrow \beta$  feedback. In particular, the  $\beta$  channels activated by the CS receive this feedback, and their  $\beta \rightarrow \mathcal{N}$  pathways are activated, in spite of the fact that drive  $D_2$  and the CS are unrelated.

This difficulty can be cured by letting the  $\alpha \rightarrow \beta$  pathways have conditionable synaptic knobs. Then the nonspecific  $\alpha \rightarrow \beta$  signals that are released by a given drive will become conditioned to the  $\beta$  representations that are compatible with (i.e., persistently contiguous in time with) this drive. See Fig. 9. In particular, if a cue compatible with drive  $D_j$  is activated and drive

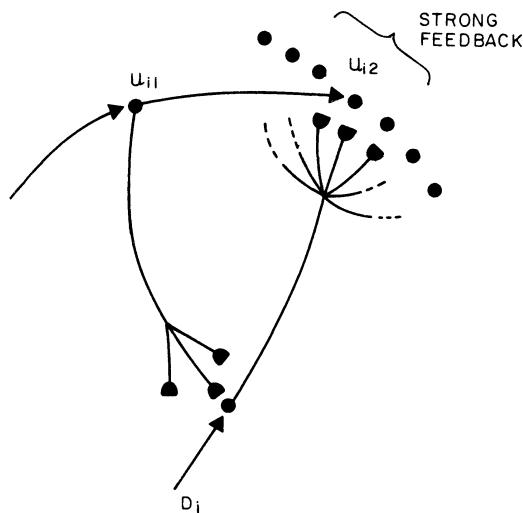


FIG. 9. Conditionable  $\alpha \rightarrow \beta$  feedback establishes psychological set.

$D_j$  is unsatisfied, then  $\mathcal{Q} \rightarrow \mathcal{S}$  feedback controlled by this cue will activate the stages  $U_{i2}$  of all  $\mathcal{S}$  representations that are compatible with  $D_j$ . In this way, motor activity compatible with the cue can be released, and a “psychological set,” or predisposition to fire, is established in all the  $U_{i2}$  stages that are compatible with  $D_j$ .

Making  $\mathcal{Q} \rightarrow \mathcal{S}$  synapses conditionable increases the symmetry of the network. Now both  $\mathcal{Q} \rightarrow \mathcal{S}$  and  $\mathcal{S} \rightarrow \mathcal{Q}$  synapses are conditionable, and these pathways fire only in response to a specific cue plus a nonspecific arousal input. Do both types of cells have a common phylogenetic ancestor?

We want conditioning of synaptic knobs to occur in both excitatory and inhibitory  $\mathcal{Q} \rightarrow \mathcal{S}$  pathways. Then inhibitory and excitatory psychological sets can be switched on separately by different cues, but an  $\mathcal{S}$  representation that often samples when both sets are on will control a mixture of excitatory and inhibitory feedback, and hence its net feedback can be small. The next section shows how to accomplish this without preventing attentiveness to fearful cues.

## VII. The Persistence of Learned Meanings

Now we are ready to begin a study of attentional factors in these networks. The networks have the following unfortunate property.

Let  $CS_i$  be a conditioned reinforcer for drive  $D_i$ ,  $i = 1, 2$ . For example, let  $CS_1$  be a roast turkey,  $CS_2$  be one's lover,  $D_1$  be hunger, and  $D_2$  be sex. Consider the situation of having roast turkey for dinner with one's lover. At dinner, both  $CS_1$  and  $CS_2$  are scanned intermittently in rapid succession, or even simultaneously. In daily life, we do not come away from the dinner table labeling our lover as a source of food and the turkey as a source of sex, as would happen if all contiguous cues were always mutually conditioned to their respective responses. Fortunately, the learned meanings of cues can endure in spite of parallel presentation of cues with different drive representations. Of course, if the turkey is persistently and consistently paired with all of our sexual encounters, then turkey can become a discriminative cue for sex, just as pairing turkey which shock can make us afraid of turkey. The above example distinguishes the forced pairing of events from the free reorganization of attention through time.

How can persistence of learned meanings be achieved in these networks? Let the  $\mathcal{S}$  representations that are activated by  $CS_i$  be denoted by  $\mathcal{S}_i$ ,  $i = 1, 2$ . We want  $\mathcal{Q}$  to be able to “notice” each  $CS_i$  as it is scanned. Thus each  $CS_i$  should be able to activate its  $\mathcal{S}_i$ . We also want to prevent *sustained simultaneous sampling* of  $\mathcal{Q}$  by  $\mathcal{S}_1$  and  $\mathcal{S}_2$ . Otherwise,  $\mathcal{S}_1$  would activate the  $\mathcal{Q}_1$  channel, and  $\mathcal{S}_2$  would sample this channel and strengthen its  $\mathcal{Q}_1$  connec-

tion. Simultaneously  $\mathcal{S}_2$  would activate  $\alpha_2$ , and  $\mathcal{S}_1$  would strengthen its  $\alpha_1$  connection. In effect, the turkey would become a cue for sex and the lover would become a cue for eating.

Our task is to prevent sustained simultaneous sampling of  $\alpha$  by  $\mathcal{S}_1$  and  $\mathcal{S}_2$  if  $\mathcal{S}_1$  and  $\mathcal{S}_2$  project to incompatible drives. To achieve this in the present context, at least three stages of processing are needed:

1.  $\mathcal{S}_1$  and  $\mathcal{S}_2$  send signals to  $\alpha$  in order to test which  $\alpha$  channels they control (e.g., do  $\mathcal{S}_1$  and  $\mathcal{S}_2$  control incompatible drives?).
2. The  $\alpha \rightarrow \mathcal{S}$  feedback measures which  $\alpha_i$  channel is stronger at any time, via the sensory-drive heterarchy.
3.  $\mathcal{S}_i \rightarrow \alpha$  sampling is shut off in the weaker channel.

Stage 1 is accomplished when the  $CS_i$  inputs activate  $\mathcal{S}_i \rightarrow \alpha_i$  signals. Stage 2 is accomplished by the sensory-drive heterarchy. How is stage 3 accomplished? Suppose for definiteness that the  $\mathcal{S}_1$  channel is stronger; that is, the strength of sensory-plus-drive inputs to  $\alpha_1$  exceeds that to  $\alpha_2$ , so that only  $\alpha_1 \rightarrow \mathcal{S}$  feedback is positive. Somehow this feedback must suppress  $\mathcal{S}_2 \rightarrow \alpha$  sampling. By Section VI,  $\alpha_1 \rightarrow \mathcal{S}$  feedback will be received only by the  $\mathcal{S}_1$  representation. How does this feedback suppress  $\mathcal{S}_2$  reverberation and sampling? An answer is suggested by Section IV. The total activity of the  $\mathcal{S}$  representations is normalized, and a quenching threshold exists. We want strong  $\alpha_1 \rightarrow \mathcal{S}_1$  feedback to enhance the activity of the  $\mathcal{S}_1$  representation and, as a consequence of normalization, to thereby, at least partially, suppress the activity, and hence the sampling, of the  $\mathcal{S}_2$  representation. The minimal way to accomplish this is to require that *specific*  $U_{i2} \rightarrow U_{i1}$  signals exist in each  $\mathcal{S}$  representation (see Fig. 10a). In Fig. 10a, strong  $\alpha_1 \rightarrow U_{12} \rightarrow U_{11}$  feedback increases the strength of activity in the  $U_{11}$  population relative to the activity in the  $U_{21}$  population. The  $U_{21}$  activity is thereupon suppressed by inhibitory signals from  $U_{11}$  to  $U_{21}$ .

The above argument holds if the drives in question control positive  $\alpha \rightarrow \mathcal{S}$  feedback. The case of drives, such as fear and frustration, which control negative feedback requires further argument. The problem is this. If the conditioned feedback is negative, then it will tend to differentially suppress activity in the controlling  $\mathcal{S}$  representation, rather than to enhance it. This would have the following maladaptive effect on behavior. Increasing the learned fearfulness of a given cue, in a fixed context of other cues, would decrease the attention paid to it. Jumping ahead in our discussion for a moment, we also would note that fearful cues could not overshadow or block learning in response to other cues, which is false (Kamin, 1968, 1969).

Hence a distinction must be made between mechanisms for learned persistence of negative meanings and for negative incentive motivation. See Figs. 10b and 10c. The former feedback channel helps to focus attention on particular cues. The latter feedback channel suppresses motor activity. The

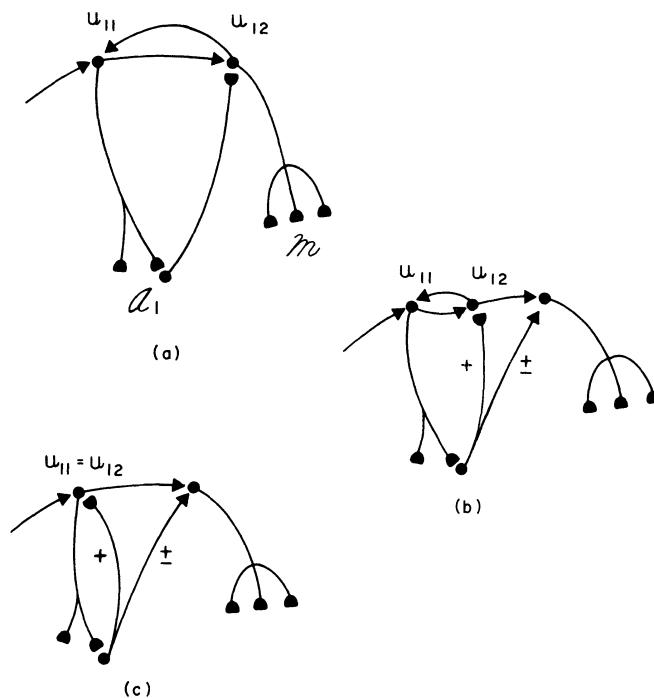


FIG. 10. Attentional feedback and motor arousal occupy different channels.

attentional feedback is always positive, even if the drive in question controls negative incentive motivation. The synapses of this feedback channel are, moreover, conditionable, so that the feedback can enhance the activity of particular representations on which attention will focus.

Given that attentional feedback is conditionable, is it also necessary to make the  $\alpha \rightarrow \mathcal{S}$  incentive motivational synapses conditionable? At any given time, the conditionable attentional feedback will guarantee that only certain  $U_{i1}$  stages will send signals to their respective  $U_{i2}$  stages. Can an irrelevant drive release  $\mathcal{S} \rightarrow M$  sampling in the manner described by Section VI? If the irrelevant drive creates conditioned attentional feedback to its "psychological set" in  $\mathcal{S}$ , then this set will tend to quench other sources in  $\mathcal{S}$ , and therefore to prevent their firing. Thus the  $\alpha \rightarrow \mathcal{S}$  incentive motivational feedback is not necessarily conditionable, although making it conditionable could only improve network efficiency.

The two kinds of feedback can be interpreted as slow potential shifts. The conditionable attentional feedback is reminiscent of the contingent negative variation, or CNV (Cohen, 1969). Such a wave has been associated

with an animal's expectancy, decision (Walter, 1964), motivation (Irwin *et al.*, 1966; Cant and Bickford, 1967), volition (McAdam *et al.*, 1966), preparatory set (Low *et al.*, 1966), and arousal (McAdam, 1969). Walter (1964) hypothesized that the CNV is a conditionable shift in the average baseline of the cortex, acting to depolarize its apical dendritic potentials and to thereby prime the cortex for action by reducing its overt response threshold to other inputs.

The incentive motivational feedback acts more as a form of motor arousal or suppression, since it controls whether or not the  $\mathcal{S} \rightarrow \mathcal{M}$  channels will fire.

Thus, the above model suggests that at a stage following the sensory-drive heterarchy, feedback channels to sensory-motor areas should bifurcate; one channel, as in the case of the CNV, should be related to an animal's attentional state, and is influenced by drives, motivation, arousal, etc. The second channel should be capable of enhancing or depressing motor output.

### VIII. Overshadowing and the Triggering of Arousal by Unexpected Events

Adding the feedback connections  $U_{i2} \rightarrow U_{i1}$ , or more generally from  $\mathcal{Q}$  to  $U_{i1}$  (cf. Fig. 10c), gives rise to phenomena like those reported in Section I,A. At the outset of Experiment 1 in that Section, neither  $CS_1$  nor  $CS_2$  projects to any particular drive representation in  $\mathcal{Q}$ . Thus both  $CS_1$  and  $CS_2$  can sample the fear representation when shock is on. Since the total  $\mathcal{S} \rightarrow \mathcal{Q}$  output is normalized, the strength of  $\mathcal{S}_i \rightarrow \mathcal{Q}$  signals depends on how many  $\mathcal{S}_i$  channels are active at any time. Thus learning by  $\mathcal{S}_1$  and  $\mathcal{S}_2$  activated together will be slower than learning by  $\mathcal{S}_1$  activated alone, unless there exists more than one limit point for  $x(t)$ , as in Fig. 6b.

In experiment 2,  $CS_1$  first becomes conditioned to fear, which we will call the  $\mathcal{Q}_1$  channel. The channels  $\mathcal{S}_1 \rightarrow \mathcal{Q}_1$  and  $\mathcal{Q}_1 \rightarrow \mathcal{S}_1$  both become conditioned during the first phase of this experiment. When  $CS_1$  and  $CS_2$  are presented in phase 2 of the experiment,  $U_{11} \rightarrow \mathcal{Q}_1 \rightarrow U_{12} \rightarrow U_{11}$  feedback suppresses sampling in the  $U_{21} \rightarrow \mathcal{Q}_1$  channel before  $CS_2$  can become conditioned to fear.  $CS_2$  is hereby overshadowed by prior fear conditioning to  $CS_1$ .

Suppose in experiment 1 that  $CS_1$  is more salient than  $CS_2$ . Then the sampling signals from  $\mathcal{S}_1$  to  $\mathcal{Q}$  will initially be larger than those from  $\mathcal{S}_2$  to  $\mathcal{Q}$ . Consequently learning in  $\mathcal{S}_1 \rightarrow \mathcal{Q}_1$  synaptic knobs will occur faster than learning in  $\mathcal{S}_2 \rightarrow \mathcal{Q}_2$  synaptic knobs. Similarly, learning in the feedback channel  $\mathcal{Q}_1 \rightarrow \mathcal{S}_1$  will occur faster than learning in the  $\mathcal{Q}_2 \rightarrow \mathcal{S}_2$  channel. The  $U_{11} \rightarrow \mathcal{Q}_1 \rightarrow U_{12} \rightarrow U_{11}$  feedback therefore grows faster than the  $U_{21} \rightarrow$

$\alpha_1 \rightarrow U_{22} \rightarrow U_{21}$  feedback. Sampling by  $U_{21}$  is hereby gradually suppressed as learning trials proceed, and  $CS_2$  is gradually overshadowed by  $CS_1$ . Similarly, if a CS and UCS are simultaneously presented, then the UCS can overshadow the CS via  $\$ \rightarrow \alpha \rightarrow \$$  feedback. If the CS occurs shortly before the UCS, then its sampling channels are active in the time interval after the UCS occurs and before  $\$ \rightarrow \alpha \rightarrow \$$  feedback can quench their activity. Hence  $CS \rightarrow UCR$  conditioning is possible in this latter case.

Experiment 3 is not so easily approached. Somehow, the occurrence of an unexpected UCS must prevent  $CS_1$  from overshadowing  $CS_2$ . Either the  $U_{11} \rightarrow \alpha_1 \rightarrow U_{21} \rightarrow U_{11}$  feedback must be weakened, or an independent nonspecific (e.g., "arousal") input to  $\$$  must keep activity at  $\$$  in the suprathreshold range.

Weakening  $U_{11} \rightarrow \alpha \rightarrow U_{21} \rightarrow U_{11}$  feedback does not seem to be a physically plausible way to overcome overshadowing. To see this, change experiment 3 as follows: in phase 1 of the experiment ( $CS_1 \rightarrow$  shock), use 40 units of shock, and in phase 2 of the experiment ( $CS_1 + CS_2 \rightarrow$  shock), use 80 units of shock. The increase in shock level is unexpected, but it should surely be accompanied by an *increase* in  $\alpha_1 \rightarrow U_{12}$  feedback. Indeed, the very survival of an animal can depend on its ability to process the reinforcing characteristics of unexpected events. The increase in  $\alpha_1 \rightarrow U_{12}$  feedback would increase the overshadowing of  $CS_2$  by  $CS_1$ , other things equal, but just the reverse occurs *in vivo*.

Overshadowing can be eliminated, or at least reduced, if unexpected events transiently increase the nonspecific arousal of  $\$$ , and thus the number of  $\$$  representations whose activity exceeds quenching threshold. This increase in overall arousal of  $\$$  competes with overshadowing tendencies controlled by motivational  $\alpha \rightarrow \$$  channels. Alternatively, it is possible that unexpected events transiently decrease the quenching threshold of  $\$$ . The latter effect could be achieved, say, by letting an unexpected event trigger shunting inhibition of the inhibitory interneurons in the off-surround of each population in  $\$$ . The triggering of arousal by unexpected events will be seen to be a basic feature of the model for dealing with a variety of phenomena (see Fig. 11). For example, the Appendix derives a formula showing that  $CS_2$  can become a learned source of relief, rather than of fear, if the shock level that follows  $CS_1 + CS_2$  is lower than the shock level that follows  $CS_1$ . This can be achieved using the increase in tonic arousal input to  $\alpha$  that accompanies the unexpected change in shock level (cf Section IX). The increase in arousal at  $\alpha$  enhances the tendency for a relief rebound to occur, whereas the increase of arousal at  $\$$  overcomes overshadowing and enables  $\$ \rightarrow \alpha$  sampling of this rebound to occur. By contributing an increase in irrelevant cue input to  $\alpha$ , the increase in arousal at  $\$$  can also enhance the relief rebound at  $\alpha$ .

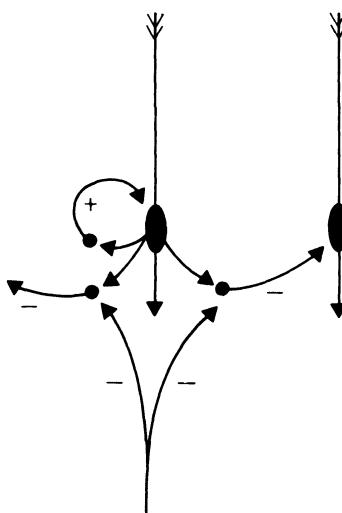


FIG. 11. Arousal-initiated inhibition of inhibitory interneurons.

Section XXI notes some possible clinical differences that would arise due to overactivity of nonspecific arousal to  $\S$  vs overactivity of conditionable  $\alpha \rightarrow \S$  feedback, especially with regard to the hypothesis that certain schizophrenic symptoms are due to imbalances in catecholamine production.

The above conclusions can be phrased in a way that emphasizes the adaptability of a network to changing environmental demands as a fundamental principle of its design. The mechanism for preserving learned meanings of cues is an adaptive attentional mechanism that permits parallel processing of cues without spurious cross-conditioning of the learned meanings of all cues. Overshadowing is a consequence of this mechanism. Overshadowing can, however, yield maladaptive network performance if the environment changes, or is only partially understood, since then the cues that presently control network output will be imperfect predictors of environmental response to this output. The property of persistence, by itself, creates too rigid a network. Taken together with the liberating effect of unexpected events on nonspecific arousal (or the quenching threshold), it can achieve a stable, but adaptively changeable, attentional mechanism.

The above discussion reduces the overshadowing problem to the problem of how arousal is triggered by an unexpected, but *not* by an expected, event. This latter problem can be restated in an informative way: how does a network *habituate* (Grossman, 1967) to a repetitively presented, and therefore increasingly expected, event? A mechanism whereby network output

is regulated by the expectedness of an event is described in Grossberg (1972a), and will be applied to the present case in Section XIX.

### **IX. Pavlovian Fear Extinction vs Persistent Learned Avoidance**

The above results suggest a mechanism for the fact that classically conditioned fear can rapidly extinguish, whereas learned asymptotic avoidance behavior can be very stable. An explanation that uses the concept of expectation in a descriptive psychological theory has been given by Seligman and Johnston (1973). Our neural explanation will use the arousing effect of unexpected events on the fear-relief dipole of Section III. Figure 4c illustrates a mechanism in which avoidance is stable, if it does not confront the network with a new source of fearful cues. To approach the fear extinction problem we suppose that an unexpected event transiently increases not only the arousal input to  $\mathcal{S}$ , but also the arousal input  $I$  to the fear-relief dipole; e.g., imagine that both regions receive arousal from a common source, such as reticular formation. Using this hypothesis, a mechanism of fear extinction is the following.

Suppose that a  $CS_1$  (e.g., bell) has persistently been paired with a shock UCS. Eventually  $\mathcal{S}_1$  will project strongly to the fear channel  $\alpha_f^+$ , and will be capable of generating a conditioned emotional response (cf. Grossberg, 1972b). If on a performance trial, the  $CS_1$  is not followed by the expected shock, then a transient increase in  $I$  occurs and causes a rebound at  $\alpha_f^-$ . This rebound is sampled by  $\mathcal{S}_1$ . The  $\mathcal{S}_1 \rightarrow \alpha_f^+$  channel is hereby counterconditioned by the increase in relative strength of the  $\mathcal{S}_1 \rightarrow \alpha_f^-$  channel, since the *net* positive feedback controlled by  $\mathcal{S}_1$  decreases. If the fear has been suppressing consummatory activity based on a positive drive, then spontaneous recovery of this activity can occur; the  $\mathcal{S} \rightarrow \mathcal{M}$  synapses which encoded the activity were not counterconditioned by fear suppression, and the positive incentive motivation that originally activated these synapses is no longer inhibited by fear (Grossberg, 1972b).

A similar rebound effect, triggered by arousal subsequent to an unexpected event, can be used to approach a neural mechanism of frustration.

### **X. Frustration**

Let a  $CS_1$  (e.g., bell) supported by drive  $\mathcal{D}_1$  (e.g., hunger) be conditioned to a response (e.g., lever press) to satisfy  $\mathcal{D}_1$  (e.g., with food that appears after the lever is pressed). Suppose that the expected food does not appear. How does the network prevent itself from persistently responding to this

$CS_1$  with lever pressing for food? This problem can be phrased in a more general way as follows: How does an organism stop persistently performing learned motor acts which no longer satisfy its needs, and free itself to seek new sources of gratification before it suffers irreversible damage due to prolonged deprivation?

To accomplish this in the networks which have already been derived, we want the nonoccurrence of the expected event to create a negative incentive-motivational output that can be sampled by  $\S_1$ . Thereafter, the occurrence of  $CS_1$  will create signals from  $\S_1$  both to the positive incentive-motivational source that used to support the motor act, as well as to the negative incentive-motivational source. The *net* incentive-motivation will decrease until  $CS_1$  no longer elicits the erroneous response.

Clearly this situation is analogous to that involving fear and relief. This analogy is depicted graphically in Fig. 12. In Figure 12a, a sudden reduction in shock or  $\S \rightarrow Q_f^+$  input, or a sudden increase in irrelevant  $\S \rightarrow Q$  or  $I$  input tend to cause a rebound at  $Q_f^-$ . In Fig. 12b, suppose that the network is engaged in a sequence of behaviors compatible with hunger. Then a persistent, large  $\S \rightarrow Q_h^+$  input drives positive  $Q_h^+ \rightarrow \S$  feedback that supports this behavior. Regulating this input through time is one of the tasks of the recurrent normalizer in  $\S$ . Suppose that the nonoccurrence of the expected event follows this sequence of acts. Because the expected event does not occur,  $\S \rightarrow Q_h^+$  input can suddenly decrease. (Temporarily ignore the case in which an unexpected event projects to  $Q_h^+$ .) Simultaneously the non-occurrence creates an increase in  $I$ . Both these factors conspire to create a negative incentive-motivational rebound at  $Q_h^-$  that  $\S_1$  can sample. Also note that an increase of arousal to  $\S$  can also create an increase in the total  $\S \rightarrow Q$  input due to cues that are "irrelevant" with respect to the  $(Q_h^+, Q_h^-)$  dipole. This input can also contribute to the rebound at  $Q_h^-$ .

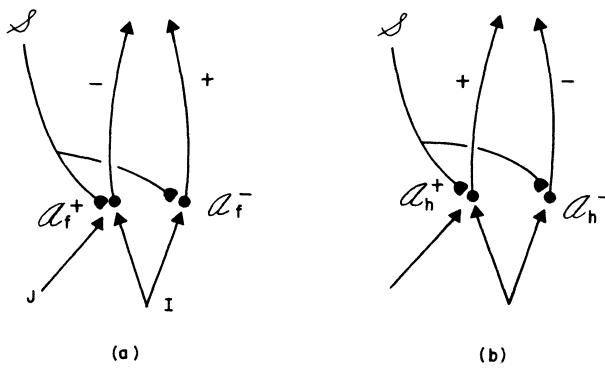


FIG. 12. Fear-relief (a) and hunger-frustration (b) dipoles.

We hereby assume, as in the case of fear and relief, that *every* drive capable of generating incentive motivational feedback has a complementary drive capable of generating incentive motivational feedback of the opposite sign. In short, drives are organized in dipoles. The properties of the fear-relief dipole are assumed to occur also in all other dipoles; e.g., a sudden reduction in expected reward can cause frustration, just as a sudden reduction in expected shock can cause relief. We also imagine that all negative incentive motivational sources are grouped together anatomically, and that all positive incentive motivational sources are grouped together. The dipole organization then becomes a *universal* feature of network design. The different behavioral meanings of particular dipoles are determined by the particular input sources that perturb them (e.g., shock at a given negative source, or metabolic levels parameterizing deprivation states at various positive sources). This anatomical grouping into positive and negative sources is reminiscent of the organization of lateral hypothalamus as a reward center and of ventromedial hypothalamus as a punishment center (Grossman, 1967) (see Fig. 13).

In Fig. 13, each dipole receives a tonic arousal input whose size through time is influenced by the unexpectedness of events. If the dipoles are arranged in a regular fashion (e.g., a row), then these arousal sources can also be regularly laid out in the network. A plausible candidate for these arousal sources is the reticular formation (Thompson, 1967). Perhaps the "poker chip" organization of the dipoles is one reason for the "poker chip" organization of reticular formation anatomy that has been so elegantly investigated by the Scheibels (1967).

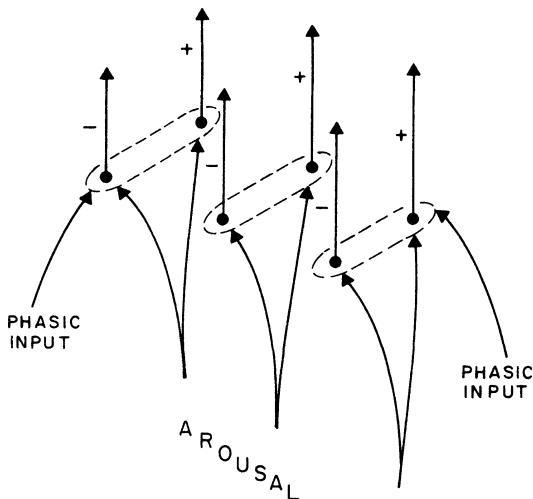


FIG. 13. Regular organization of dipoles and supportive arousal sources.

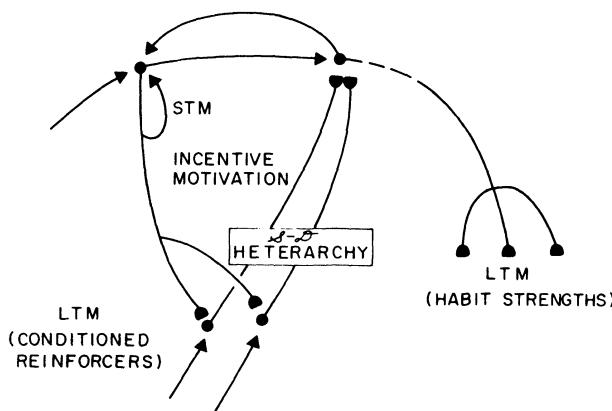


FIG. 14. Influence of motivational feedback on transfer of short-term memory (STM) to long-term memory (LTM).

The normalizer that determines the final sensory-drive heterarchical feedback to  $\S$  must come after the dipole stage. Various data suggest the plausibility of interpreting this normalizer as an idealized analog of hippocampus. It can, for example, maintain a baseline of learned incentive motivational feedback (Olds, 1969), its elimination can prevent transfer of short-term memory to long-term memory (Milner, 1958), and it is organized as a recurrent on-center off-surround anatomy (Anderson *et al.*, 1969) (see Fig. 14). Thus rather simple behavioral considerations generate a subdivision of network anatomy into components that are, at least in a broad qualitative way, suggestive of significant neuroanatomical structures.

### XI. Partial Reinforcement Acquisition Effect

We analyze this effect by noting an analog in the fear-relief dipole. The relief rebound that occurs after termination of a fixed shock level typically has a maximum larger than the maximum fear level produced by the shock. This permits learned avoidance or escape cues to create positive net incentive motivation in the presence of fearful cues, and to thereby activate avoidance or escape behavior. The (relief:fear) ratio is, moreover, an increasing function of arousal level.

The same mathematical properties hold for the hunger-frustration dipole, but with different labels. If a network is rewarded with food for running down an alley, the cue representations  $\S(R)$  which control this behavior will become conditioned to the hunger arousal cells  $G_h^+$ . If the network is rewarded on a random schedule, then the nonoccurrence of expected food

can create a frustrating rebound at  $\alpha_h^-$ , both by decreasing  $\$ \rightarrow \alpha_h^+$  input and by increasing nonspecific arousal due to the unexpected event. The  $\$R$  and other  $\$$  representations that are active during the  $\alpha_h^-$  rebound will therefore be conditioned to  $\alpha_h^-$ . The net feedback from  $(\alpha_h^+, \alpha_h^-)$  to  $\$$  activated by these representations will therefore be smaller than in the case of a continuously rewarded network, for which only  $\$R \rightarrow \alpha_h^+$  conditioning occurs. In particular, early in training, while the  $\$R \rightarrow \alpha_h^+$  connection and the  $\alpha_h^-$  rebound develop, the partially rewarded network will find the goal box less attractive than the continuously rewarded network. As training proceeds, an ever stronger  $\$R \rightarrow \alpha_h^-$  projection develops. The frequency of reward must be adjusted to prevent the  $\$R \rightarrow \alpha_h^+$  channel from dominating the  $\$R \rightarrow \alpha_h^+$  channel; otherwise the network will eventually stop seeking the goal. Suppose that the frequency of reward has been suitably adjusted. Then what happens on a reward trial late in the training of a randomly rewarded network? First, the usual boost in  $\$ \rightarrow \alpha_h^+$  connection strength will occur. Second, there will be a sudden reduction in activity of the cues that are conditioned to frustration. Third, owing to the partial unexpectedness of reward, there can be a transient increase in arousal. The second and third effects both tend to create a rebound from  $\alpha_h^-$  to  $\alpha_h^+$  due to "frustration reduction," just as reduction of shock intensity tends to produce a relief rebound. This rebound combines with the usual rewarding effect of food to produce an enhancement of the desirability of the goal late in training. In short, the enhancement can be analyzed as a double rebound, with conditioning in between, from  $\alpha_h^+$  to  $\alpha_h^-$ , and then back to  $\alpha_h^+$ .

It is ironic that the persistence of this kind of frustrating behavior can be analyzed as a composite of two effects that have a manifestly adaptive biological value. The rebound from positive to negative allows an organism to countercondition erroneous expectations before they do irreversible damage. The rebound from negative to positive allows an organism to learn escape from or avoidance of noxious events. The rebound from positive to negative to positive can, however, generate a maladaptive persistence in seeking an unlikely goal; e.g., gambling. Moreover, while cues that are conditioned to frustration are active, they can negatively bias the interpretation of other cues as possible alternatives, and can suppress exploratory behavior by inhibiting positive incentives. The network can "fall into a rut," and might tenaciously await the elusive frustration reduction that can give it some relief.

## XII. Generalization Gradients in Discrimination Learning

Our discussion of discrimination learning will try to show how various mechanisms fit together to qualitatively generate empirical effects. A more

quantitative analysis will appear in another place. The main new assumption is that a sensory input will excite not only its own internal representation in  $\S$ , but also the representations of closely related inputs, e.g., the auditory system in which peripheral auditory cells have "tuning curves" that include a connected band of frequencies (see Fig. 15). Given this elementary fact, the model already at our disposal generates various nontrivial effects.

The populations in  $\S$  will also be interconnected by a recurrent on-center off-surround field of shunting type for the reasons cited in Section IV. There exist variations on this theme which will not be considered here, but which are being studied. For example, let each input perturb only its own  $\S$  representation, but let the on-center and off-surround of each representation have a generalization gradient that includes related populations; or let the inputs, the on-center, and the off-surround have such a generalization gradient. Before considering particular experiments, we now note various general properties of this system. Let  $CS_i$  be an  $X_i$  cps tone for definiteness. Suppose that  $CS_i$  activates  $\S_i$  while  $\alpha_i^+$  is the dominant active arousal source. Then all representations in  $\S$  which are sufficiently excited by  $CS_i$  to exceed the quenching threshold of  $\S$  will sample  $\alpha_i^+$  with an intensity that increases as a function of input strength. If  $i = h$ , then  $\S_h$  eventually controls positive  $\alpha \rightarrow \S$  feedback. If  $i = f$ , then  $\S_f$  eventually controls negative  $\alpha \rightarrow \S$  feedback (see Fig. 16).

Let a test CS ( $CS_T$ ) have an  $\S$  representation ( $\S_T$ ) that lies within the generalization gradients of both  $\S_h$  and  $\S_f$ .  $\S_T$  will become conditioned to both  $\alpha_i^+$  during presentations of  $CS_h$  or  $CS_f$ , with a relative strength that depends in part on how close  $\S_T$  lies to these foci of input activity. Thus the net  $\alpha \rightarrow \S$  feedback controlled by  $\S_T$  will be a mixture of positive and negative signals. How strong will the feedback be when  $CS_T$  is presented on test trials?

$CS_T$  also has a generalization gradient that excites a band of  $\S$  representations, including  $\S_h$  and  $\S_f$ . Some of these  $\S$  representations lie in the generalization gradients of  $\S_f$  and/or  $\S_h$ , and will therefore be conditioned to

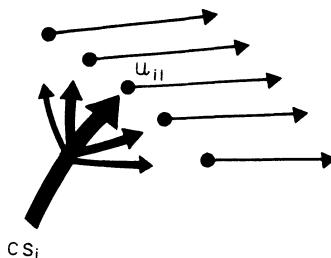


FIG. 15. Tuning curves underlying generalization gradients.

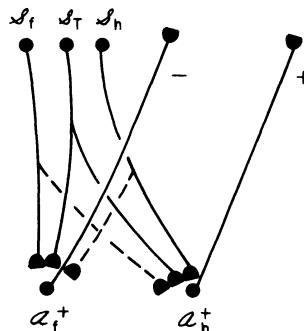


FIG. 16. Net feedback varies along generalization gradient.

$\alpha_f^+$  and/or  $\alpha_h^+$  with a relative strength depending on how close they lie to these foci. Thus when  $CS_T$  is presented, it will excite a band of  $\mathcal{S}$  representations that project with differing patterns to  $\alpha_f^+$  and  $\alpha_h^+$ . The net  $\alpha \rightarrow \mathcal{S}$  feedback is a composite of all these patterns after they filtered through the sensory-drive heterarchy. Recall also, however, that there is conditionable  $\alpha \rightarrow \mathcal{S}$  feedback, specific  $U_{i2} \rightarrow U_{i1}$  feedback, and normalization within  $\mathcal{S}$  to contend with.

To understand what happens in a qualitative way, we first make an unsatisfactory approximation, and then improve it step-by-step. First, ignore the generalization gradient in  $\mathcal{S}$  of  $CS_T$  and compute the net feedback that would occur in response to activating any  $S_T$  when this feedback is just the resultant of the relative  $S_T \rightarrow \alpha_h^+$  and  $S_T \rightarrow \alpha_f^+$  path strengths, and the total path strength is normalized (see Fig. 17a). The boldface curve in Fig. 17a shows the resultant at any  $\mathcal{S}$  representation  $S_T$  of the gradients centered at  $S_f$  and  $S_h$ . Note that the resultant gradient always is less than the  $S_h$  gradient, but that its slope is steeper than the  $S_h$  gradient. What is the effect of normalization by the on-center off-surround field? The normalized  $S_T$  gradient is shown in Fig. 17b. Its maximum is higher than the  $S_h$  gradient because the positive part of the  $S_T$  gradient in Fig. 17a is narrower and steeper than the  $S_h$  gradient. Thus normalization of the resultant gradient produces behavioral contrast. Also there is a peak shift away from  $S_f$ , and a steepening of the generalization gradient due to discrimination training. The need for normalization, in turn, can be traced back to the need to prevent  $\mathcal{S} \rightarrow \alpha$  signals from creating  $\alpha \rightarrow \mathcal{S}$  feedback in the absence of supporting drives. The various other mechanisms at work in the network can now be switched in without changing these qualitative conclusions.

Why does a pronounced peak shift not occur if the training is errorless? In our networks, errorless training means that there is no fear or frustration (Bower, 1966), hence no negative gradient to interact with the positive

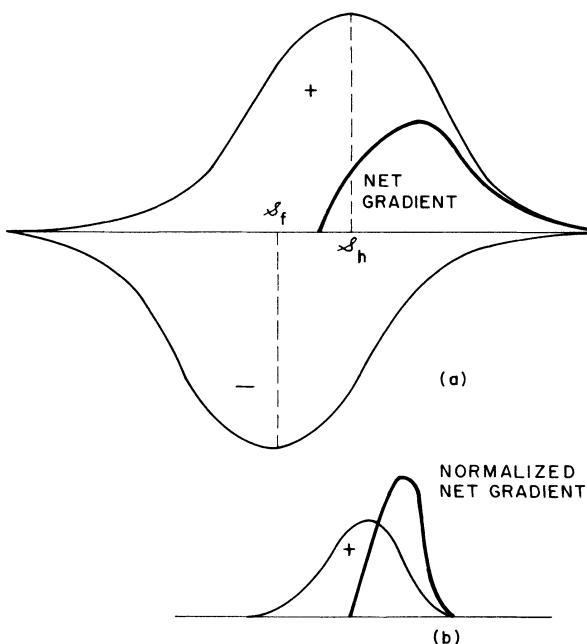


FIG. 17. Normalization yields behavioral contrast based on net generalization gradient.

gradient to cause a shift. If, however, a shock is paired with the S- after errorless discrimination training, then  $S- \rightarrow \alpha_r^+$  conditioning will occur (along with  $\mathcal{S} \rightarrow \alpha_r^+$  conditioning of all the  $\mathcal{S}$  representations in the S-gradient) and a peak shift will develop (Grusec, 1968). Bloomfield's (1969) remarks about "an unexpected change for the worse" can readily be interpreted in this context. A sudden reduction in reinforcement frequency generates a frustrative rebound, and hence sampling by  $\mathcal{S}$  of a negative feedback source, as does introduction of shock, etc. Honig's (1962) suggestion that the peak shift occurs only if the S+ and S- are presented successively, but not if they are presented simultaneously, can also be discussed. One possible reason for the latter fact is that rewarding S+ gradually gives it control over a powerful positive  $\mathcal{S} \rightarrow \alpha \rightarrow \mathcal{S}$  feedback channel. This feedback enables S+ to overshadow S-, so that as the animal's expectation of reward develops, the tendency to approach S+ simultaneously dominates the tendency to approach S-. Moreover, before this expectation develops, responding to S- will not generate a large negative rebound. In the successive paradigm, approach to S- is not inhibited by the presence of S+, so

that more occasions occur after the expectation of reward develops that generate a frustrative negative rebound. Consequently, in the simultaneous paradigm S— does not gain (as much) control over a negative feedback channel, and the peak shift does not develop.

Normalization can also be used to interpret the Newman and Baron (1965) and Newman and Benefeld (Honig, 1970) studies. These studies suggest that the color dimension can mask the orientation dimension in pigeons, but that some conditioning is occurring in the orientation dimension nonetheless. In the Newman and Benefeld study, a vertical line on a green background during training is replaced by a vertical line on a black background during testing, and a generalization gradient is demonstrated even in the absence of discrimination training with an S—. We suppose that removal of the green background has three effects. It (1) eliminates the strong color → orientation inhibition due to the off-surround. Thus (2) the vertical orientation representation, and its generalization gradient, become more active owing to a shift of activity in the normalized field. This gradient can thereupon sample  $\alpha$  and release its learned (but previously weak)  $\alpha \rightarrow \S$  feedback. Removal of the green background also (3) causes surprise by changing the expected line-color combination used during training, and thereby enhances activity both of the orientation representation in  $\S$  and of the sampled drive representations in  $\alpha$  by triggering a transient increase in nonspecific arousal.

### XIII. Habituation and the Hippocampus

The Newman and Baron study shows that a white line on a green key as S+ and a green key as S— produces a generalization gradient on the orientation dimension. Why does color not overshadow orientation in this situation? A phenomenon of this type exists in our networks. It is due to the interaction of several mechanisms, namely, (1) normalization and the quenching threshold, (2) conditioned arousal, (3) conditionable net incentive motivation, and (4) feedback of net incentive motivation to the normalization stage. On the initial S+ trials, color will partially overshadow orientation via the off-surround in  $\S$ . Denote the relevant color representations in  $\S$  by  $\S(C)$  and the orientation representations by  $\S(O)$ . Both  $\S(C)$  and  $\S(O)$  will sample  $\alpha_h^+$  on these trials, but  $\S(C)$  will build up a stronger connection since its activity is greater. As this occurs,  $\alpha_h^+ \rightarrow \S$  feedback paths will become conditioned to  $\S(C)$  and  $\S(O)$  with a similar difference in relative strength. In the usual overshadowing experiment, this initial advantage of  $\S(C)$  over  $\S(O)$  will be progressively enhanced as training continues until  $\S(C)$  completely overshadows  $\S(O)$ . In the present context, however, S— trials occur. On these trials,  $\S(O)$  is inactive.  $\S(C)$  is active,

but the expected reward does not occur. A frustrative rebound is therefore generated.  $\S(C)$  thereupon samples  $\alpha_h^+$ . Simultaneously,  $\alpha_h^+$  samples the second stages  $U_{i2}$  of  $\S(C)$ . Thus the *net* incentive motivation controlled by  $\S(C)$  is progressively diminished by frustrative nonreward. On S+ trials, the net feedback from  $(\alpha_h^+, \alpha_h^-)$  to  $\S(C)$  is cut down, owing to competition between these two channels before they release incentive motivation, but the feedback to  $\S(O)$  comes only from  $\alpha_h^+$ , and increases through time. Asymptotically, the  $\S(O)$  activity, bolstered by  $\S(O) \rightarrow \alpha_h^+ \rightarrow \S(O)$  feedback, can dominate the  $\S(C)$  activity. In this limited sense, the network has habituated to  $\S(C)$ , even as it grows ever more attentive to  $\S(O)$ . This habituation mechanism has several interesting properties. First, the sensory channel itself does not habituate; habituation is an active process based on interpretive feedback of sensory information via the drive representations (Grossman, 1967; Sharpless and Jasper, 1956). Second, suppose that the normalizer which creates a sensory-drive heterarchy is indeed interpreted as a simplified hippocampus. Then the hippocampus becomes involved in attentional control and the habituation of attention, but only indirectly via its determination of which motivational channel will be active in response to particular cues.

Section XX describes another habituation mechanism with the property that increasingly expected, and in particular repetitively presented, events elicit progressively smaller orienting reactions.

Why does a generalization gradient not occur if the S— is a red key or if the S— is a vertical white line on a red key? Then the color dimension is not habituated by frustrative rebound. Indeed, in the latter case, the orientation dimension might habituate, although perhaps at a slow rate because it is overshadowed by the dominant color dimension.

Note that expectation mechanisms can interact with habituation mechanisms in two opposing ways in the above experiment. First, they contribute to the frustrative rebound during S— trials, by altering the uniform (e.g., arousal) input to  $\alpha_h^+$  and  $\alpha_h^-$  on these trials. Second, they work against habituation by creating nonspecific arousal in  $\S$  that tends to overcome the reduction of  $\S \rightarrow \alpha \rightarrow \S$  feedback in particular channels, and allows them to once again reverberate in STM.

#### XIV. Overshadowing vs Enhancement

We now interpret and contrast the Honig (1969) experiments with the F. Freeman (unpublished master's thesis, 1967) experiment. Honig used TD and PD training sessions, followed by dimensional acquisition and finally testing on an orientation dimension. In the TD experiment, the pigeons

were trained to make a discrimination on a dominant (namely, color) dimension. In particular, lesser dimensions were overshadowed, and the pigeon acquired a strong expectation and a positive conditioned arousal path in response to the S+ color cues. On dimensional acquisition trials, the color cues were not present, so that the orientation dimension, no longer overshadowed, could be trained, given that the pigeon still maintains general approach tendencies to the lever.

The PD training session, by contrast, frustrates the pigeon on the dominant color dimension. Yet the reinforcement schedule has been chosen so as to overcome frustration and yield a net approach tendency. The cues that elicit learned approach are not the frustrated cues of the color dimension. These cues develop their own powerful positive feedback paths. It is reasonable to assume that these cues are also present on the orientation dimensional acquisition trials. If they are, they will (at least partially) overshadow the orientation dimension both on orientation training and testing trials.

By contrast, Freeman trained pigeons to peck at a vertical line on a dark key (S+) but not to peck at a line tilted at 120° on the same dark background (S-). A generalization gradient is hereby established on the orientation dimension. Then dimensional acquisition occurs with the vertical line on a green background, and one finds a nontrivial chromatic generalization gradient on testing trials. Why does the orientation dimension not overshadow the green background during dimensional acquisition? One wants to say that surprise, and hence arousal, is triggered by changing the black background to green. Then the green  $\mathcal{S}(C)$  representations will be able to sample the positive net incentive motivation controlled by the vertical  $\mathcal{S}(O)$  representations. This explanation works, however, only if one first can answer the question: why does the orientation dimension not overshadow the dark background during discrimination training? And if the dark background is overshadowed, and therefore irrelevant, why is the pigeon surprised if it is removed on dimensional acquisition trials? The importance of these questions is perhaps better seen when they are phrased as follows: If the pigeon *does* get surprised when the dark background is replaced by green, then why does this not happen in *all* overshadowing experiments when the CS<sub>1</sub> is replaced by the CS<sub>1</sub> + CS<sub>2</sub>, thereby preventing overshadowing from occurring?

To answer these questions, we seek differences in how the expectation mechanism (and thus arousal) responds in the Freeman experiment as opposed to the usual overshadowing experiment. We want to say that introducing green in the Freeman experiment is more surprising, say, than introducing a tone as CS<sub>2</sub> after prior CER training with a flashing light as CS<sub>1</sub> in a Kamin-type overshadowing experiment. A difference of degree is sought

in the two experiments, rather than the operation of different mechanisms. We suggest that this difference exists, in part at least, because the pigeon can develop an expectation of a vertical line on a particular visual background more easily than it can develop an expectation of a flashing light in a prescribed combination of events involving nonvisual modalities. In other words, a learned expectation can be at least partially localized to a given cluster of features or events, and features which stream into the same modality in close physical contiguity can be more easily grouped together as a coherent expectation than features which enter through different modalities, other things equal. If this is true, then it might be easier to eliminate overshadowing of CS<sub>2</sub> by CS<sub>1</sub> in a Kamin-type experiment if the CS<sub>1</sub> is a vertical line and the CS<sub>2</sub> is a green background, than if the two events involve different modalities. This kind of prediction is hard to analyze completely because inputs to two different modalities are hard to equate psychophysically, and can activate orienting reactions that need not be activated by two inputs to the same modality.

The Freeman experiment demonstrates enhancement due to prior discrimination training. The closely related Mackintosh and Honig experiment (Honig, 1970) demonstrates blocking. We suggest that blocking occurs because the surprise that is triggered during redundant spectral discrimination training, after orientation discrimination training has been completed, only partly overcomes overshadowing. When no prior orientation discrimination exists, and only spectral discrimination training is given, there is no overshadowing to overcome.

If the above analysis is accurate, then one might be able to create a transition from overshadowing to enhancement in a given experimental setup by varying the relative strength of the attentional and surprise channels, say by drugs.

#### XV. Novelty and Reinforcement

Berlyne (1969) showed that a novel light change, contingent on lever pressing, can reinforce lever pressing. We suggest that the novelty of the light change, as usual, triggers nonspecific arousal which, as usual, filters through all drive representations. If a positive incentive motivational source is active when arousal occurs, and this source dominates other drive representations at that time, then the arousal will enhance the amount of positive motivation. The lever press cues  $\mathcal{S}(L)$  can become differentially conditioned to the positive source, which also supplies enough incentive to trigger  $\mathcal{S}(L) \rightarrow \mathcal{M}$  sampling of the motor commands that control the lever press. We suggest that the source of positive incentive in this case is the motor

arousal source for exploratory approach and pressing of the lever, rather a specific drive representation.

We can now provide an answer to a related question: Why is the approach incentive motivation not *usually* the motivational source for learned goal objects? One reason is that, unless the approach source is *differentially* strengthened by arousal enhancement or other means, then *all* meaningless objects in the environment can be approached, and none will be approached more frequently than any other, other things equal. A second reason is that, when a specific drive is rewarded, then the source of positive incentive tends to shift from general exploration and approach to the specific drive representation that was rewarded.

The enhancing effect of arousal on the pattern of activity at drive representations can also generate incentive motivational feedback to sensory representations in the absence of external sensory cues. For example, if the hunger drive is prepotent, and all drive representations are aroused, then  $\alpha_h^+$  can generate feedback to its psychological set  $S_h$  in  $\mathcal{S}$ , leading, say, after further enhancement through the feedback loop  $S_h \rightarrow \alpha_h^+ \rightarrow S_h$ , to the motor output "I want food." More generally, the network can ask itself "how it feels" by arousing its drive representations. The resulting motivational feedback from  $\alpha$  to  $\mathcal{S}$  can establish a psychological set that is capable of generating compatible motor activity. This possibility is a special case of the "two-thirds rule" discussed in Section XXII.

## XVI. Motivation and Generalization

How does increased drive flatten generalization gradients? A formal answer exists in the networks. Increasing the drive increases positive incentive motivational signals in  $\alpha \rightarrow \{U_{i2}\}$  synapses. Increasing these signals has two effects. It speeds up conditioning in the  $\alpha \rightarrow \{U_{i2}\}$  synapses, and it increases the signals from  $\{U_{i2}\}$  to  $\{U_{i1}\}$ . At  $\{U_{i1}\}$ , the increased input allows more  $\mathcal{S}$  representations to exceed quenching threshold, and faster conditioning occurs in the  $\mathcal{S} \rightarrow \alpha$  synapses of these representations.

How does this mechanism affect generalization gradients? If a particular  $\mathcal{S}$  representation is activated by external cues, its generalization gradient of  $\mathcal{S}$  representations will also be activated, albeit to a lesser extent. Increasing  $\alpha \rightarrow \{U_{i2}\} \rightarrow \{U_{i1}\}$  signals permits more of these representations to sample drive representations in  $\alpha$ . Thus, on testing trials, more cues in the generalization gradient can generate the type of feedback that was elicited on training trials. The generalization gradient is hereby flattened. Section IV also shows that if the nonspecific feedback is additive at  $\mathcal{S}$  sites, then it will tend to flatten the gradient by uniformizing the pattern of activity in  $\mathcal{S}$ .

If two drives compete, then increasing one drive can steepen the general-

ization gradient on the other drive (Hoffman, 1969). Let the two drives be hunger and fear for definiteness, and consider the generalization of fear conditioned to a 1000 cps tone. Let the  $\mathcal{S}$  representation  $\mathcal{S}(X)$  of an  $X$  cps tone be activated by the tone on testing trials. Choose  $X$  in the generalization gradient of the 1000 cps tone. On training trials,  $\mathcal{S}(X)$  sampled  $\alpha_f^+$ , and possibly also  $\alpha_h^+$ . Thus the strength of  $\alpha_h^+ \rightarrow \mathcal{S}$  feedback is increased relative to the strength of  $\alpha_f^+ \rightarrow \mathcal{S}$  feedback, which is driven by  $\mathcal{S}(X) \rightarrow \alpha_f^+$  signals. The suppressive effect of fear is hereby reduced by increasing the hunger level.

Why does this mechanism steepen the fear generalization gradient? A formal reason is that a fixed increment in positive feedback can totally overcome the suppressive effect of a sufficiently small amount of negative feedback, but has only a small relative effect on large amounts of negative feedback. An  $X$  cps generalization gradient controls large amounts of negative feedback, but tones near the edge of the 1000 cps generalization gradient control only small amounts of negative feedback. Hence the increase in hunger narrows and steepens the fear gradient.

### XVII. Predictability and Ulcers

If Weiss's experiments (1971a,b,c) on the development of stomach ulcers in rats are performed on our networks, then the net incentive motivation in the networks is a monotone increasing function of the degree of ulceration in his experiments. This analysis does not give a physiological explanation of the ulcerogenic process, but it does suggest that the frustrative sources of negative incentive are also triggered at the same time as sources of ulcer-inducing agents.

Why do avoidance-escape networks develop less ulceration than yoked networks? Avoidance-escape networks have been trained to respond to cues which activate positive incentive motivation that supports avoidance and/or escape activity. The positive incentive competes with the negative incentive generated by shock, and thereby reduces the net negative incentive motivation.

Why does a warning signal reduce the ulceration of both groups of networks? It can do so by reducing the novelty of the shock. By Section VIII, this will reduce the arousal level that accompanies the shock, and thus the net negative incentive that the shock produces. In the avoidance-escape networks, the warning signal can also be used as a discriminative cue for activating avoidance-escape cues that switch on positive incentive motivation.

Why do the yoked networks develop less severe ulcers than the avoidance-escape networks if both groups receive a brief shock after each avoid-

ance-escape response? Three effects in the network conspire to produce this result. First, the network is motivated by positive incentive in making the avoidance-escape response; this motivational source is abruptly terminated. Second, the network expects relief after performing the response, but does not get it; this unexpected event triggers nonspecific arousal. Third, a negative, or punishing, event occurs instead of the expected relief. The first effect tends to produce a positive-to-negative rebound. The third effect creates a second source of negativity. And the second effect enhances the total negative tendency. The first and second effects are absent, or at least much weaker, in yoked networks.

Why does little ulceration develop in avoidance-escape networks if each avoidance-escape response is followed by a feedback stimulus, such as a tone? Three effects are operating in our networks. First, the avoidance-escape response produces relief, as in Section III. Second, the novel tone, of itself, produces nonspecific arousal. As in the analyses of the fear-relief dipole in Section III, and of the Berlyne (1969) experiments in Section XV, this arousal enhances the relief rebound that is produced. Third, these effects speed up the conditioning of avoidance-escape cues to the positive incentive motivational source, and therefore reduce the net negative incentive that is produced even before the coping response is made.

Is this analysis compatible with Weiss's idea that no ulcers can develop in the absence of a coping response? It is compatible with a weaker statement: that coping responses can enhance or suppress ulceration, but that any mechanism that produces negative incentive in the rebound mechanism creates a predisposition to ulcerate. A deeper analysis of the way in which positive and negative incentive actually regulate muscular contraction might refine this view at a later time.

### XVIII. Orienting Reaction

We will show below that some properties of this reaction can formally be represented within the networks that are already at our disposal. We will invoke psychophysical examples to illustrate the formal meaning of the mechanisms, but do not presume that they are given a complete physiological explanation. Consider Fig. 18.

In Fig. 18, different paths  $P_i$  are differentially excited by different peripheral events, e.g., retinal loci, positions on the skin, auditory inputs. Suppose that  $U_{i2}$  can fire only if orienting arousal combines with a signal from  $U_{i1}$ . Let the axon collaterals from  $U_{i1}$  to  $\mathfrak{N}$  have relative strengths that determine a final orienting position for the muscles that they control. Different  $P_i$  paths will determine different orienting positions by having dif-

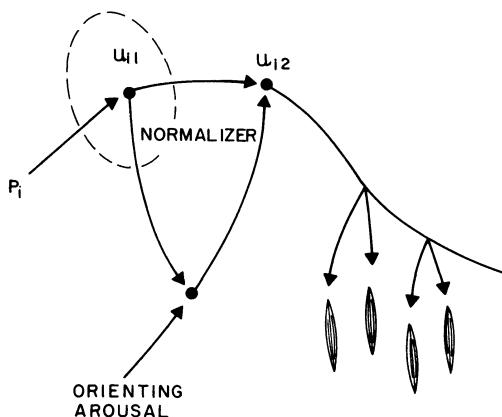


FIG. 18. Orienting arousal activates position codes for motor control.

ferent patterns of axon strength. Let a recurrent normalizer interconnect the  $\{U_{i1}\}$  populations. This establishes a “position code” in the field  $\{U_{i1}\}$  of populations by normalizing the total activity of the field (e.g., normalizing the effect of variations in the total light energy hitting the retina), and letting the  $U_{i2} \rightarrow \mathcal{M}$  axon strengths determine the terminal muscle positions. For example, if two  $U_{i1}$  are simultaneously and equally active, then a position will be determined that lies between the positions determined by each  $U_{i1}$  separately. As the relative activity of one  $U_{i1}$  increases, the terminal position will approach the position controlled by this  $U_{i1}$  alone. In the case of vision, for example, if the arousal level of the field is tuned so that only one population can reverberate in STM, then only one retinal light source can attract the eyes. If arousal permits several populations simultaneously to reverberate, then weighted averages of the retinal positions can attract the eyes.

Withdrawing orienting arousal prevents the release of signals from any  $U_{i2}$ . We assume that this arousal can be inhibited by activity from competing arousal sources, such as drive representations. Thus, before training, a loud noise in the direction of a subject's left side can elicit an orienting reaction toward the left. Suppose, however, that the noise is used in learning trials as a discriminative cue for rapid lever pressing for food. Then on testing trials, the noise can differentially excite the  $G_h^+$  representation, which can inhibit the source of orienting arousal via, say, the sensory-drive hierarchy.

The source of orienting arousal is triggered by unexpected events. Minimality bids that we identify this arousal source with the arousal source, also triggered by unexpected events, that overcomes blocking and triggers enhancement or rebound in the various drive representations. A plausible candidate for this arousal source is the reticular formation.

There exist variations on this anatomical theme, such as an orienting arousal source supplying *shunting* excitation that permits the cells which carry the position code to fire. Such an arousal source can also act at the synaptic knobs, or to inhibit tonic presynaptic inhibition of these knobs (disinhibition). In all the above anatomies, excitation and disinhibition can have similar functional effects. Disinhibition has the disadvantage of requiring an extra processing step, but it has the advantage that it permits sustained activity of cells, which prevents them from undergoing a chemical degradation due to disuse.

### XIX. A Learned Expectation Mechanism

An expectation mechanism is described below to help fix ideas in the above discussion. We wish to prevent orienting arousal if an expected event occurs, and to permit it if an unexpected event occurs. The first part of the construction synthesizes a network which can learn to expect a given event subsequent to the occurrence of another event. Several variations of this construction appear in Grossberg (1972a). This construction will be supplemented herein to guarantee additional properties of the expectation mechanism. The output cells  $U$  of the network will fire only if the learned expected event occurs. The construction in Grossberg (1972a) is reviewed below for completeness.

The learned input pattern (or class of patterns) which can fire the cell (or cells)  $U$  is controlled by presetting cells  $P$ . The cells  $P$  send axons to the filtering mechanism (e.g., inhibitory interneurons and dendrites) that processes inputs to  $U$ . Each  $P$  cell can learn a particular pattern that will bias  $U$ 's filter when  $P$  is active. For example, consider an animal  $\Theta$  that learns to lever press for food. On a testing trial,  $\Theta$  "expects" food when it lever presses in response to hunger. We suppose that lever press cues also preset consummatory controls which can be released by expected sensory cues of the food reward. Similarly, suppose that one goes to the refrigerator expecting to find orange juice, which one loves, in a transparent container, but instead one finds tomato juice, to which one is indifferent. The same motor sequence of reaching, pouring, and drinking suffices for imbibing either the orange juice or the tomato juice. The orange fluid releases this sequence, but the red fluid does not; indeed, the red fluid can release a frustrative rebound. The consummatory controls have been preset by the expectation of an orange fluid.

How do the  $P$  cells learn the patterns on training trials that will bias the  $U$ -cell filter on testing trials? Consider the anatomy of Fig. 19, in which interacting signals combine additively. In Fig. 19, the cells  $V_1 = \{v^{(j)}\}$ :

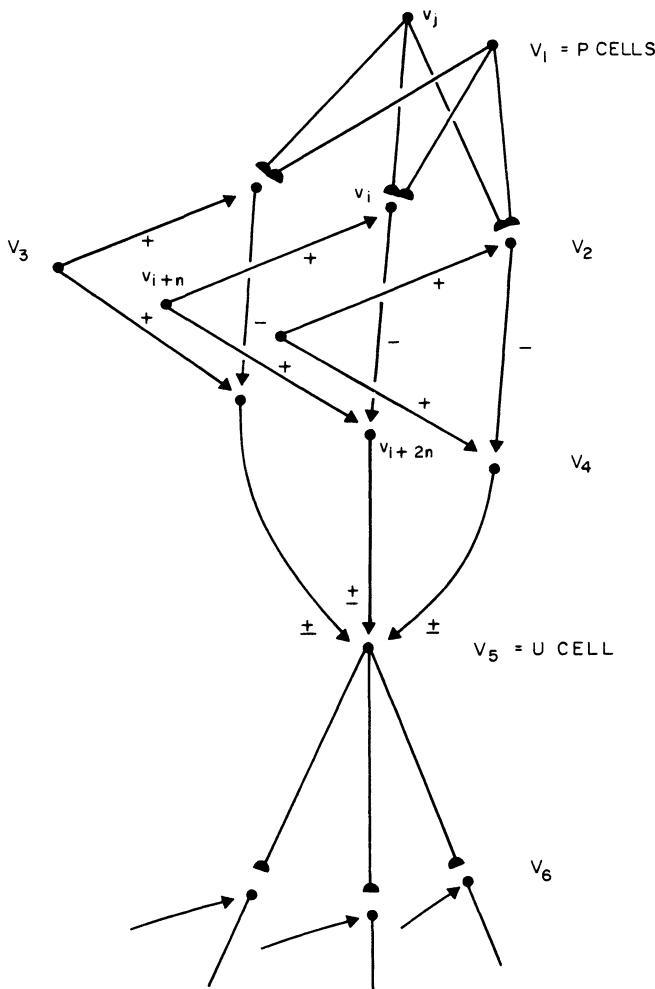


FIG. 19. Subtractive preset mechanism.

$j = 1, 2, \dots, n\}$  are  $P$  cells. These cells sample patterns playing on the cells  $V_2 = \{v_i: i = 1, 2, \dots, m\}$  when they are active. The cells  $V_3 = \{v_{i+m}: i = 1, 2, 3, \dots, m\}$  receive the test patterns that will fire the cell  $U$  if they are expected by  $P$ . On training trials,  $V_3 \rightarrow V_2$  signals reproduce these test patterns at  $V_2$ , where they can be sampled by  $V_1$ . Signals in  $V_3 \rightarrow V_4$  axons, where  $V_4 = \{v_{i+2m}: i = 1, 2, \dots, m\}$ , also reproduce the test patterns at  $V_4$ . On testing trials, activity in a  $P$  cell generates a pattern  $\theta$  of activity in  $V_2$ , which is transferred to  $V_4$  as inhibitory signals by  $V_2 \rightarrow V_4$

axons. The test pattern  $\tilde{\theta}$  at  $V_3$  is also sent along to  $V_4$ , where it can be compared with  $\theta$ . Simultaneously,  $\tilde{\theta}$  is sent to  $V_2$  to be sampled by  $V_1$ . At  $V_2$ ,  $\tilde{\theta}$  is transferred to  $V_4$  as inhibitory signals, just as the  $V_1 \rightarrow V_2$  presetting signals are. The inhibitory  $V_3 \rightarrow V_4$  signals are chosen weaker than the excitatory  $V_3 \rightarrow V_4$  signals, so that the test pattern appears at  $V_4$  with a net excitatory strength. The inhibitory preset pattern  $\theta$  and the excitatory test pattern  $\tilde{\theta}$  are thereupon compared at  $V_4$ .

The above transformations can be defined in greater detail as follows. Let the strength of the excitatory  $v_{i+m} \rightarrow v_{i+2m}$  signal be  $\tilde{\theta}_i I$ , and of the inhibitory  $v_{i+m} \rightarrow v_i \rightarrow v_{i+2m}$  signal be  $\tilde{\theta}_i \eta I$ ,  $0 < \eta < 1$ . Then the net signal to  $v_{i+2m}$  from  $v_{i+m}$  is  $\tilde{\theta}_i(1 - \eta)I$ , which is nonnegative. Let the  $v^{(j)} \rightarrow v_{i+n} \rightarrow v_{i+2m}$  inhibitory signal from the  $j$ th preset cell be  $-\theta_i K$ . If only  $v^{(j)}$  in  $V_1$  is active, the total signal to  $v_{i+2m}$  is  $\tilde{\theta}_i(1 - \eta)I - \theta_i K$ . Under these circumstances,  $v_{i+2m}$  will fire only if

$$\tilde{\theta}_i(1 - \eta)I > \theta_i K \quad (2)$$

This constraint shows that all cells in  $V_4$  can fire only if all the relative pattern activities in  $\tilde{\theta}$  are not too much smaller than the relative pattern activities in  $\theta$ . Since  $\sum_k \theta_k = \sum_k \tilde{\theta}_k = 1$ , simultaneous firing in all channels is possible only if  $(1 - \eta)I > K$ . Thus the total activities of  $V_1$  and of  $V_3$  must be carefully regulated.

Inequalities (2) do not suffice to prevent firing of a discriminative cell further downstream to patterns some of whose  $\tilde{\theta}_i$  are much larger than  $\theta_i$  (Grossberg, 1970, 1972a). To prevent this, the output signal from each  $v_{i+2m}$  in  $V_4$  excites both an excitatory and an inhibitory pathway. The inhibitory pathway (which can, in principle, be just a high threshold inhibitory ionic channel in the same axon pathway as the excitatory channel) overcomes the excitatory pathway if the signal from  $v_{i+2m}$  is too large. When this happens, the net output from  $v_{i+2m}$  to  $V_5$  is negative, so that not all channels are simultaneously excitatory. Thus the net signal from  $v_{i+2m}$  to  $V_5$  is derived from two successive inhibitory mechanisms. It is positive at  $v_{i+2m}$  only if the relative pattern activity  $\tilde{\theta}_i$  is not too much smaller than the relative pattern activity  $\theta_i$ . This positive activity is inhibited, however, if  $\tilde{\theta}_i$  is too much larger than  $\theta_i$ . All channels in  $V_4$  contribute a positive signal to  $V_5$  only if the pattern  $\tilde{\theta}$  is close, in every component, to the pattern  $\theta$ . The signal threshold of  $V_5$  is adjusted once and for all so that  $V_5$  will fire only if it receives (nearly) simultaneous positive signals from all  $V_4$  channels. Hence the cell  $U = V_5$  fires only in response to the expected pattern. Grossberg (1972a) shows that this anatomy has formal properties that are reminiscent of cerebellar anatomy, and thereby illustrates the anatomical pausibility of this expectation mechanism.

The same principles have been used to synthesize a class of networks with a suggestive retinal analog (Grossberg, 1972a). These networks are

capable of discriminating the relative figure-to-ground of spatial patterns (i.e., their  $\theta_i$ 's) but do not have a learnable expectation mechanism. Here also two successive inhibitory mechanisms are needed. If the receptor cells of this network are interpreted as light receptors, then the first inhibitory layer is reminiscent of retinal horizontal cells. Speaking functionally, this layer produces a form of light adaptation; cf. unicellular recordings in the mudpuppy retina (Werblin, 1971). The second layer is reminiscent of amacrine cells. The output cells (cf. ganglion cells) are then capable of hue constancy (including a lightness scale), brightness constancy, velocity detection, etc., depending on which receptors are hooked into the network, and on how the anatomical connection coefficients are chosen.

The expectation mechanisms defined above has two deficiencies: (1) It does not automatically regulate the total activities of  $V_1$  and  $V_3$ ; and (2) if no presetting cell in  $P$  is active, then *every* pattern presented to  $V_3$  can fire  $V_5$ , since no net inhibitory signal is produced at  $V_4$ . The first deficiency can be overcome by introducing recurrent on-center off-surround anatomies with shunting interactions into  $V_1$  and  $V_3$ . Section IV indicates the need for such mechanisms within sensory processors, so that their use here does not impose a new constraint. The second deficiency can be overcome by assuming that uniformly distributed *tonic* inhibitory signals are somehow generated from  $V_2$  to  $V_4$  in the absence of presetting signals, and that the onset of presetting signals supplants the tonic inhibition with learned patterns of  $V_2 \rightarrow V_4$  inhibition. A simple way to do this is to assume that tonically active cells exist in  $V_1$  and send uniformly distributed inputs to  $V_2$ ;  $V_2$ , in turn, generates inhibitory signals to  $V_4$  that prevent inputs to  $V_3$  from firing  $V_4$ . When a presetting cell in  $V_1$  becomes sufficiently active, it suppresses the activity in the tonic cells via the recurrent off-surround in  $V_1$ , and substitutes its own patterned signals to  $V_2$ . Tonically active cells that are suppressed by the onset of phasic afferents are known to exist in various neural structures; in the frog retina, for example, there are dimming cells whose tonic activity in the dark is suppressed by light (Chung *et al.*, 1970). Note also that the distribution of tonic inputs to  $V_2$  can be uniform even if no tonic cell is connected to all cells in  $V_2$ ; only the *distribution* of activity across all tonically active cells needs to be uniform, and this distribution can be suppressed uniformly by widely dispersed off-surround signals within  $V_1$ .

## XX. Regulation of Orienting Arousal

The output cells  $U$  fire only if their expected event occurs. If any unexpected event occurs, we want it to generate orienting arousal. It seems very

unlikely that a brain contains internal models of the infinitely many events that are unexpected at any time, and that it generates orienting arousal whenever there is a match between one of these events and its internal model. By contrast, given the above construction, it is easy to devise a network that inhibits orienting arousal only if the expected event occurs. Thus, we assume that *every* event which is processed by the network's sensory mechanisms can, in principle, activate orienting arousal using as a source the neural activity which it generates as it is processed. The output from the expectation mechanism can, however, inhibit orienting arousal (cf. Sokolov, 1960) (see Fig. 20). In Fig. 20, the output from the cells  $U$  bifurcates. One channel inhibits orienting arousal and the other channel samples the drive representations in  $\mathcal{Q}$ . For example, suppose that the expected event is a loud noise to the left of the network, and that the noise has been trained as a discriminative cue for lever pressing. When the noise occurs, it generates activity that can drive the orienting reaction. This activity is, however, inhibited by the output from  $U$ . The  $U$  output also generates positive  $\mathcal{S} \rightarrow \mathcal{Q} \rightarrow \mathcal{S}$  feedback that elicits the lever press. The orienting reaction can be inhibited by this mechanism even if  $U$  controls no other motor reaction. The construction can be modified to change this conclusion. If the orienting arousal channel is included in the on-center off-surround anatomy of the

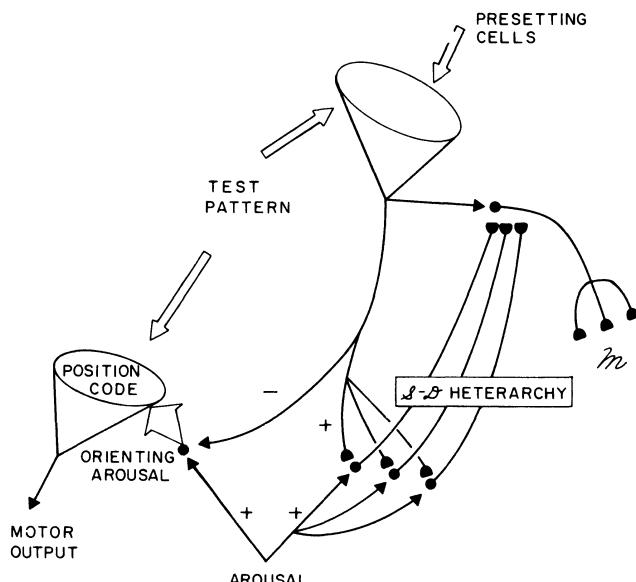


FIG. 20. Inhibition of orienting arousal by expected events.

sensory-drive heterarchy, then the orienting arousal can occur unless it is supplanted by strong competing  $S \rightarrow G \rightarrow S$  feedback in a specific drive channel.

The relationship between presetting inputs and test inputs will be more completely studied in another place. In particular, one must note that the events which excite  $P$  cells and those which excite  $V_3$  cells need differ only in their onset times; the  $P$  events occur earlier than the  $V_3$  events. Thus,  $V_3$  events gain control over  $P$  cells as new events intervene. This shift in the spatial locus of an event's internal representation can be subsumed under the study of sequential short-term memory buffers (Atkinson and Schiffrin, 1968). It is schematically represented in Fig. 21. Given such a shift in representational locus, one can see how this network becomes habituated to a repetitively presented event. As the event is repeated, it serves as a source of  $P$ -cell activity in its "past" mode, and as a source of test inputs in its "present" mode. The event samples itself, in short. As the event is repeated, it samples itself repeatedly via  $P \rightarrow V_2$  axons which build up the strength of the expectation. As the event becomes more expected, the output from  $U$  increases and progressively inhibits orienting arousal, but does not prevent conditioned responses from occurring. For example, young foxes quickly habituate orienting reactions to the sound of mouse squeaking, but once they have eaten a mouse, the squeaks become conditioned stimuli and the orienting reactions do not readily habituate (Biryukov, 1958; Lynn, 1966). One can also see how the network can become habituated to a learned set

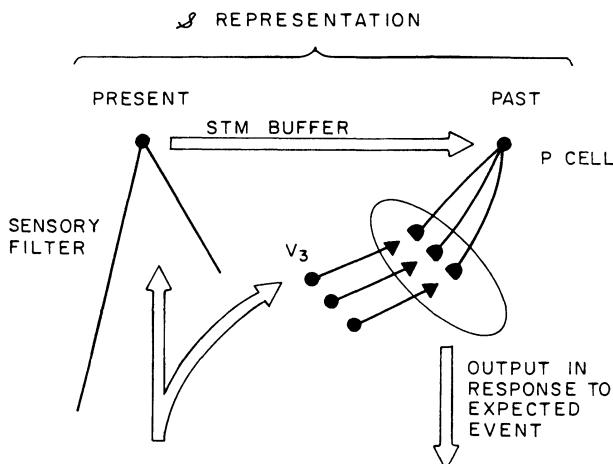


FIG. 21. Habituation to repeated event as its past-representation samples its present-representation in the expectation mechanism.

of events via conditioning of the  $P \rightarrow V_2$  synapses of the  $P$  representations corresponding to this set. Indeed, if the sensory filter is capable of grouping peripheral events in classes that fall along unconditioned or conditioned generalization gradients, then these gradients will be transferred to the  $P$  cells via the sequential STM buffer.

### XXI. Hippocampal Feedback, Conditioning, and Dendritic Spines

This section provides a way to implement three formal requirements in the network using a common mechanism. The mechanism has a suggestive anatomical analog in terms of hippocampus, septum, hypothalamus, reticular formation, and neocortex. In this analog, the hippocampus receives input from neocortex (*in vivo*, via the entorhinal cortex) and septum (Raisman *et al.*, 1966). The mock-hippocampal output trifurcates and eventually feeds back to septum as signals conditionable at  $\alpha \rightarrow \alpha$  synapses, and to neocortex as nonspecific attentional or motor feedback, possibly via the anterior thalamic nuclei (Raisman *et al.*, 1966). The mock-hypothalamus prepares drive inputs to this system, and the reticular formation provides nonspecific arousal, which can be triggered by specific events, and which is filtered through the sensory and drive representations to enhance or rebound their activity.

The three formal requirements are these:

1. Consider Fig. 22. In the figure,  $\$1$  is conditioned to  $\alpha_1$ , but all  $\$i$ ,  $i \neq 1$ , are irrelevant cues; i.e., they project equally to  $\alpha_1$  and  $\alpha_2$ . Suppose that many of these irrelevant cues are active when  $\$1$  is active. Then the  $\$1 \rightarrow \alpha_2$  synapses will become progressively stronger and eventually  $\$1$  will approach irrelevancy also. That is, the act of performing in response to a relevant event can countercondition the event simply because irrelevant events exist. Part of this difficulty can be overcome by  $\alpha \rightarrow \$$  attentional feedback, which tends to quench irrelevant cues. This does not, however, prevent counterconditioning of the  $\alpha_1 \rightarrow \$1$  channel by irrelevant cues that are active before the feedback occurs, however, just as a CS that is presented

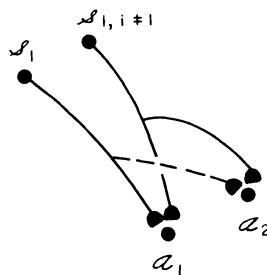


FIG. 22. Counterconditioning by irrelevant cues.

before a UCS can sample the UCS-controlled representations without being totally overshadowed by the UCS.

To prevent counterconditioning by irrelevant cues, the uniform part of the total input to a dipole's channels must be inhibited before substantial  $S \rightarrow Q$  sampling of these channels occurs. This can occur only at a stage after the  $S \rightarrow Q$  synaptic knobs, because one only knows that the  $S \rightarrow Q$  signals are uniform after they are emitted at their respective synapses. Also the resultant of this inhibition must feed back in a form that can be sampled by  $S \rightarrow Q$  synaptic knobs. How can the bulk of the conditionable signal be due to  $Q \rightarrow Q$  feedback, and not to the  $S \rightarrow Q$  signals that are in spatial contiguity with other  $S \rightarrow Q$  synapses?

2. The existence of higher-order instrumental conditioning implies that  $S \rightarrow Q$  sampling can occur both before and after the stage of drive rebounds; hence there exists a recurrent loop from sampling mechanism, to rebound mechanism, to choice-among-drives mechanism, and back to sampling mechanism.

3. What kind of feedback should be conditionable? Should the feedback be from the resultant of each dipole separately, or from the resultant of all competing drives? In the latter case, conditioning is possible only with respect to the drive that supplies incentive motivation for regulating attention, motor performance, and the transfer of STM into LTM. We exhibit a system of the latter type for definiteness. Variations on the theme are then readily constructable.

To achieve (2) and (3), we use a mechanism as in Fig. 23. Note that the output of the sensory-drive heterarchy trifurcates: it is fed back to "neocortex" as attentional feedback and as motor arousal, and it is fed back to "septum" as conditionable signals.

To achieve (1), we must somehow allow  $S \rightarrow Q$  signals to influence events further downstream without allowing these signals to be substantially conditioned to anything but sensory-drive heterarchical output. One way to do this is suggested in Fig. 24. The  $S \rightarrow Q$  signals reach "dendritic spines." Here they produce local potentials that propagate to the cell body where they influence axonal firing. We assume that the resistances in spines are such that it is much harder for a signal to pass between spines than from a spine to the cell body. Alternatively, one can assume that the threshold for the post- to presynaptic signals that are needed to change transmitter levels in  $S \rightarrow Q$  synapses are too high for spine-to-spine interactions to overcome them. By contrast, heterarchical feedback from  $Q$ , energized by nonspecific arousal (e.g., from reticular formation) causes a spike potential, or similar global potential change, throughout the dendritic column. This spike invades all the spines in its path and is sufficiently strong to induce transmitter level changes in active  $S \rightarrow Q$  channels. Thus a mechanism using

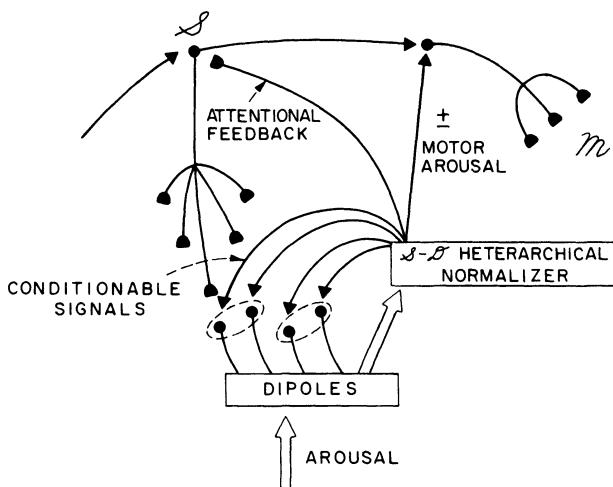


FIG. 23. Conditionable heterarchical feedback signals sampled by  $\mathcal{S}$ .

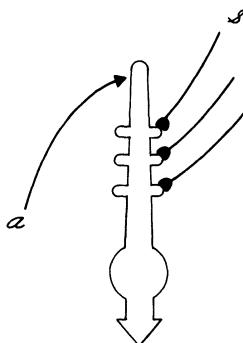


FIG. 24. Heterarchical feedback causes global potential change that invades individual  $\mathcal{S}$  channels.

dendritic spines and dendritic spike generators (or some formally analogous mechanism) can allow  $\mathcal{S} \rightarrow \mathcal{Q}$  signals to occur without major changes in  $\mathcal{S} \rightarrow \mathcal{Q}$  synaptic transmitter levels unless feedback invades the entire dendritic apparatus.

## XXII. Nervous Eating and Attentional Deficits Modulated by Arousal

Section III pointed out that the existence of higher-order instrumental conditioning implies the existence of feedback loops in the rebound mecha-

nism. Section IV showed that the signal function in a recurrent anatomy must be carefully chosen to avoid amplification of noise. Faster than linear growth of signals at small values of cell activity, followed by (approximately) linear growth at larger values, achieves this goal. Since saturation of signal response at very high activity levels is easily assured, it suffices to impose faster than linear growth of signals at small values of cell activity; by continuity, there will exist an approximately linear range between the faster-than-linear growth region and the slower-than-linear saturation region of signal response. The width of the linear region is an important parameter in determining the short-term memory characteristics of the system. Indeed, the slower-than-linear saturation region tends to create a uniform distribution of activity across cell populations, starting with any initial input pattern.

Given such a nonlinear signal function, the Appendix shows that an increase in arousal can potentiate the system's response to phasic sensory inputs. Thus, a novel event can overcome overshadowing, thereby freeing its  $S$  representation to send signals to  $G$ . In  $G$ , these signals contribute to the sensory-plus-drive combinations that are struggling to gain control over  $G \rightarrow S$  feedback via the sensory-drive hierarchy. Simultaneously, the arousal triggered at  $G$  can cause a rebound and/or enhance the  $G \rightarrow S$  feedback from the dominant sensory-plus-drive combination. Similarly, the novelty of an indifferent event can make it rewarding, if the network is engaged in approach behavior when the event occurs (Berlyne, 1969).

In effect, enhancement by arousal produces an extra source of input to  $G$ . Using this new input source, the network can, in principle, generate  $G \rightarrow S$  feedback in the absence of drive inputs, thereby yielding the following interesting possibilities: (1) Motor activity initiated by an internally generated arousal pulse in the absence of external sensory cues; e.g., by testing its drive states with an arousal pulse, the network can generate a hunger-related output, such as the statement "I want food," if the hunger drive is dominant but no cues of food are present. (2) If the arousal level is high, it can elicit consummatory activity compatible with sensory cues or drives that are too low to otherwise initiate consummation; e.g., "nervous eating." If, for example, there is a damming up of motor activity in the absence of an appropriate goal, and this activity feeds into  $G$  as arousal, then potentiation by arousal can discharge the motor activity through the heterarchical channel that is dominant at that time. (3) An unexpected event, even a frustrating one, can elicit transient motor activity via heterarchical feedback, even if the heterarchical feedback is not related in a simple way to the unexpected event.

These remarks illustrate a so-called "two-thirds rule." Namely, at least two channels from among  $S$ ,  $D$ , and  $G$  are needed to elicit  $G \rightarrow S$  feedback:  $S + D$  is the usual heterarchical constraint on  $G \rightarrow S$  firing;  $D + G$  is illus-

trated by the "I want food" example; and  $\mathcal{S} + \mathcal{Q}$  is illustrated by "nervous eating." One can also imagine, in principle, the perhaps pathological case in which intense  $\mathcal{S} + \mathcal{D} + \mathcal{Q}$  activity allows more than one heterarchical channel to be active at a time, by driving the activity of more than one channel above quenching threshold; cf. the remarks about quenching threshold relevant to determining the asymptotic "eye position" in Section XVIII.

The mixing of channel activities at the  $\mathcal{Q}$  cells has an analog at  $\mathcal{S}$  cells. Here converge arousal inputs that are triggered by unexpected events, and which consequently tend to overcome overshadowing, as well as arousal inputs from  $\mathcal{Q} \rightarrow \mathcal{S}$  channels that contribute motivational feedback, and can sustain overshadowing. Under pathological circumstances, either channel can become persistently overaroused. One possible consequence of overarousal is "seizure activity" (cf. Grossberg, 1973). Another is the inability to pay attention (cf. Grossberg and Pepe, 1971). Either of the two arousal sources can cause such difficulties, in principle, but the overall "clinical syndrome" that the network would undergo could be quite different in the two situations.  $\mathcal{Q} \rightarrow \mathcal{S}$  overarousal can cause, in addition, emotional depression (Grossberg, 1972c), as well as pathological changes in the network's "psychological sets" (Section VI). If, for example, one heterarchical  $\mathcal{Q} \rightarrow \mathcal{S}$  feedback channel became dominant, then it could bias all the network's sensory processing in a direction that is compatible with the dominant drive, or more precisely, the dominant "psychological set." Such an effect need not occur if the source of novelty-bound arousal is overaroused, since this arousal source is truly nonspecific. Previous work (Grossberg, 1972c) suggests than an analogy can be drawn between  $\mathcal{Q} \rightarrow \mathcal{S}$  channels and midbrain channels influenced by catecholamine production. Thus, imbalances in catecholamine production might produce an overaroused syndrome ("simple schizophrenia"?) which is different from an overaroused syndrome produced by malfunction of the reticular formation, both in symptomatology and in proper treatment.

SUBMITTED FOR PUBLICATION: March, 1973.

## Appendix

$CS_2$  can become a learned source of relief, rather than of fear, if the shock level that follows  $CS_1 + CS_2$  is sufficiently small compared to the shock level that follows  $CS_1$ . This happens if the increase in tonic input  $I$  that follows the unexpected change in shock level  $J$  is sufficiently great compared to the change in  $J$ . Moreover, for sufficiently small values of  $I$  and  $J$ , an increase in  $I$  can potentiate the fear produced by a fixed level of  $J$ , if the signal function is sigmoid.

Consider the following system for definiteness (see Grossberg, 1972c, for details).

$$\begin{aligned}\dot{x}_1 &= -\alpha x_1 + I + J \\ \dot{x}_2 &= -\alpha x_2 + I \\ \dot{z}_1 &= \beta(\gamma - z_1) - \delta f(x_1(t - \tau))z_1 \\ \dot{z}_2 &= \beta(\gamma - z_2) - \delta f(x_2(t - \tau))z_2 \\ \dot{x}_3 &= -\epsilon x_3 + \xi f(x_1(t - \tau))z_1 \\ \dot{x}_4 &= -\epsilon x_4 + \xi f(x_2(t - \tau))z_2 \\ \dot{x}_5 &= -\eta x_5 + \kappa[x_3(t - \sigma) - x_4(t - \sigma)] \\ \dot{x}_6 &= -\eta x_6 + \kappa[x_4(t - \sigma) - x_5(t - \sigma)]\end{aligned}$$

First, constant levels  $I_1$  and  $J_1$  of tonic input and shock are switched on until after the potentials  $x_i$  and transmitters  $z_j$  adjust to their new levels. Then new levels  $I_2$  and  $J_2$  are imposed. The potentials adjust much quicker than the transmitters. Hence a measure of the maximum response to the change in levels is computed by maintaining the transmitters at the steady-state levels determined by  $I_1$  and  $J_1$ , and the potentials at the new steady-state levels imposed by  $I_2$  and  $J_2$ . Fear is produced if  $x_5 > 0 > x_6$ ; relief is produced if  $x_5 < 0 < x_6$ . The function  $f(w)$  computes the signal in response to the potential  $w$ . In Grossberg (1974), the function  $f(w) = \max(0, w - \Gamma)$  is used, where  $\Gamma$  is a signal threshold. Fear is produced only if

$$f\left(\frac{I_2 + J_2}{\alpha}\right)\left[1 + \mu f\left(\frac{I_1}{\alpha}\right)\right] > f\left(\frac{I_2}{\alpha}\right)\left[1 + \mu f\left(\frac{I_1 + J_1}{\alpha}\right)\right]$$

where  $\mu = \delta\beta$ , and relief is produced only if the reverse inequality holds. For example, if  $f(w) = w$ , then fear is produced only if

$$J_2/J_1 > I_2/(v + I_1)$$

where  $v = \alpha\beta/\delta$ .

The steady-state fear response to constant  $I$  and  $J$  is given by

$$x_5(\infty) = \frac{\omega \left[ f\left(\frac{I+J}{\alpha}\right) - f\left(\frac{I}{\alpha}\right) \right]}{\left[1 + \mu f\left(\frac{I}{\alpha}\right)\right]\left[1 + \mu f\left(\frac{I+J}{\alpha}\right)\right]}$$

where  $\omega = \kappa\xi\gamma\eta^{-1}\epsilon^{-1}$ . A sigmoid  $f$  yields potentiation of  $x_5(\infty)$  in response to an increase of  $I$ , if  $I$  and  $J$  are sufficiently small. To see this is the case that  $f''$  exists, we compute  $[\partial x_5(\infty)]/\partial I$ , and note that this function is positive if and only if the function

$$h(w) = f'(w)/[\mu f(w) + 1]^2$$

is strictly monotone increasing in the desired region of  $I$  and  $J$  values. If  $f''$  exists, this condition becomes

$$[\mu f(w) + 1]f''(w) > 2\mu[f'(w)]^2$$

which is true for small values of  $w$ , since  $f(0) = f'(0) = 0 < f''(0)$ .

#### REFERENCES

- Amsel, A. (1958). *Psychol. Bull.* **55**, 102.
- Amsel, A. (1962). *Psychol. Rev.* **69**, 306.
- Anderson, P., Gross, G. N., Lomo, T., and Sveen, O. (1969). In "The Interneuron" (M. Brazier, ed.), p. 415. Univ. of California Press, Los Angeles.
- Atkinson, R. C., and Shiffrin, R. M. (1968). In "The Psychology of Learning and Motivation," (K. W. Spence and J. T. Spence, eds.), Vol. 2, p. 89. Academic Press, New York.
- Azrin, N. H., and Holz, W. C. (1966). In "Operant Behavior" (W. K. Honig, ed.), p. 380. Appleton, New York.
- Berlyne, D. E. (1969). In "Reinforcement and Behavior" (J. T. Tapp, ed.), p. 179. Academic Press, New York.
- Berlyne, D. E. (1970). In "Attention: Contemporary Theory and Analysis" (D. E. Mostofsky, ed.), p. 25. Appleton, New York.
- Bersh, P. J., Notterman, J. M., and Schoenfeld, W. N. (1956). Air University, School of Aviation Medicine, U.S.A.F., Randolph AFB, Texas.
- Biryukov, D. A. (1958). The nature of orienting reactions. In "The Orienting Reflex and Orienting-Investigating Activity" (L. G. Voronin *et al.*, eds.), Acad. Pedag. Sci., Moscow.
- Bitterman, M. E. (1965). In "Classical Conditioning" (W. F. Prokasy, ed.), p. 1. Appleton, New York.
- Bloomfield, T. M. (1966). *J. Exp. Anal. Behav.* **9**, 155.
- Bloomfield, T. M. (1969). In "Animal Discrimination Learning" (R. M. Gilbert and N. S. Sutherland, eds.), p. 215. Academic Press, New York.
- Bower, G. H. (1966). In "Theories of Learning" (E. R. Hilgard and G. H. Bower, eds.), Appleton, New York.
- Cant, B. R., and Bickford, R. G. (1967). *Electroencephalogr. Clin. Neurophysiol.* **23**, 594.
- Chung, S.-H., Raymond, S. A., and Lettvin, J. V. (1970). *Brain Behav. Evol.* **3**, 72.
- Cohen, J. (1969). In "Average Evoked Potentials" (E. Donchin and D. B. Lindsley, eds.), p. 143. Nat. Aeron. Space Admin., Washington, D.C.
- Goodrich, K. P. (1959). *J. Exp. Psychol.* **57**, 57.
- Grastyan, E. (1959). In "The Central Nervous System and Behavior" (M. A. Brazier, ed.) Josiah Macy, Jr. Found., New York.
- Gray, J. A., and Smith, P. T. (1969). In "Animal Discrimination Learning" (R. M. Gilbert and N. S. Sutherland, eds.), p. 243. Academic Press, New York.
- Grossberg, S. (1969a). *J. Math. Psychol.* **6**, 209.
- Grossberg, S. (1969b). *Math. Biosci.* **4**, 201.
- Grossberg, S. (1970). *J. Theor. Biol.* **27**, 291.
- Grossberg, S. (1971a). *J. Theor. Biol.* **33**, 225.
- Grossberg, S. (1971b). *Proc. Nat. Acad. Sci. U.S.* **68**, 828.

- Grossberg, S. (1972a). *Kybernetik* 10, 49.
- Grossberg, S. (1972b). *Math. Biosci.* 15, 39.
- Grossberg, S. (1972c). *Math. Biosci.* 15, 253.
- Grossberg, S. (1972d). In "Delay and Functional Differential Equations and their Applications" (K. Schmitt, ed.), p. 121. Academic Press, New York.
- Grossberg, S. (1973). *Stud. Appl. Math.* 52, 213.
- Grossberg, S. (1974). In "Progress in Theoretical Biology" (F. M. Snell, ed.), p. 51. Academic Press, New York.
- Grossberg, S., and Pepe, J. (1971). *J. Statist. Phys.* 1, 319.
- Grossman, S. P. (1967). "A Textbook of Physiological Psychology." Wiley, New York.
- Grusec, T. (1968). *J. Exp. Anal. Behav.* 11, 239.
- Haggard, D. F. (1959). *Psychol. Rec.* 9, 11.
- Hanson, H. M. (1959). *J. Exp. Psychol.* 58, 321.
- Hoffman, H. S. (1969). In "Animal Discrimination Learning" (R. M. Gilbert and N. S. Sutherland, eds.), p. 63. Academic Press, New York.
- Honig, W. K. (1962). *J. Exp. Psychol.* 64, 239.
- Honig, W. K. (1969). In "Animal Discrimination Learning" (R. M. Gilbert and N. S. Sutherland, eds.), p. 35. Academic Press, New York.
- Honig, W. K. (1970). In "Attention: Contemporary Theory and Analysis" (D. I. Mostofsky, ed.), p. 193. Appleton, New York.
- Irwin, D. A., Rebert, C. S., McAdam, D. W., and Knott, J. R. (1966). *Electroencephalogr. Clin. Neurophysiol.* 21, 412.
- Jenkins, H. M., and Harrison, R. H. (1960). *J. Exp. Psychol.* 59, 246.
- Jenkins, W. O., Pascal, G. R., and Walker, R. W., Jr. (1958). *J. Exp. Psychol.* 56, 274.
- Kamin, L. J. (1968). In "Miami Symposium on the Prediction of Behavior 1967: Aversive Stimulation" (M. R. Jones, ed.), Univ. of Florida Press, p. 9. Coral Gables.
- Kamin, L. J. (1969). In "Punishment and Aversive Behavior" (B. A. Campbell and R. M. Church, eds.), p. 279. Appleton, New York.
- Kelleher, R. T. (1966). In "Operant Behavior" (W. K. Honig, ed.), p. 160. Appleton, New York.
- Kernell, D. (1965a). *Acta Physiol. Scand.* 65, 65.
- Kernell, D. (1965b). *Acta Physiol. Scand.* 65, 74.
- Kimble, G. A. (1961). "Conditioning and Learning." Appleton, New York.
- Low, M. D., Borda, R. P., Frost, J. D., and Kellaway, P. (1966). *Neurology* 16, 771.
- Luria, A. R., and Homskaya, E. D. (1970). In "Attention: Contemporary Theory and Analysis" (D. I. Mostofsky, ed.), p. 303. Appleton, New York.
- Lynn, R. (1966). "Attention, Arousal, and the Orientation Reaction." Pergamon, Oxford.
- McAdam, D. W. (1969). *Electroencephalogr. Clin. Neurophysiol.* 26, 216.
- McAdam, D. W., Irwin, D. A., Rebert, C. S., and Knott, J. R. (1966). *Electroencephalogr. Clin. Neurophysiol.* 21, 194.
- McAllister, W. R., and McAllister, D. E. (1971). In "Aversive Conditioning and Learning" (F. R. Brush, ed.), p. 105. Academic Press, New York.
- Maier, S. F., Seligman, M. E. P., and Solomon, R. L. (1969). In "Punishment and Aversive Behavior" (B. A. Campbell and R. M. Church, eds.), p. 299. Appleton, New York.

- Milner, B. (1958). In "The Brain and Human Behavior" (H. C. Solomon, S. Cobb, and W. Penfield, eds.), p. 244. Williams & Wilkins, Baltimore, Maryland.
- Newman, F. L., and Baron, M. R. (1965). *J. Comp. Physiol. Psychol.* **60**, 59.
- Newman, F. L., and Benefield, R. L. (1968). *J. Comp. Physiol. Psychol.* **66**, 101.
- Olds, J. (1969). *Amer. Psychol.* **24**, 114.
- Raisman, G., Cowman, W. M., and Powell, T. P. S. (1966). *Brain* **89**, 83.
- Rall, W. (1955). *J. Cell. Comp. Physiol.* **46**, 413.
- Scheibel, M. E., and Scheibel, A. B. (1967). In "The Neurosciences: A Study Program" (G. C. Quarton, T. Melnechuk, and F. O. Schmitt, eds.), p. 577. Rockefeller Univ. Press, New York.
- Seligman, M. E. P., and Johnston, J. C. (1973). In "Contemporary Prospectives in Learning and Conditioning," Scripta, Washington.
- Sharpless, S., and Jasper, H. (1956). *Brain* **79**, 655.
- Sokolov, E. N. (1960). In "The Central Nervous System and Behavior" (M. A. Brazier, ed.), Josiah Macy, Jr. Found., New York.
- Terrace, H. S. (1966). In "Operant Behavior" (W. K. Honig, ed.), p. 271. Appleton, New York.
- Thompson, R. F. (1967). "Foundations of Physiological Psychology." Harper, New York.
- Trabasso, T., and Bower, G. H. (1968). "Attention in Learning: Theory and Research." Wiley, New York.
- Wagner, A. R. (1969a). In "Punishment and Aversive Behavior" (B. A. Campbell and R. M. Church, eds.), p. 157. Appleton, New York.
- Wagner, A. R. (1969b). In "Animal Discrimination Learning" (R. M. Gilbert and N. S. Sutherland, eds.), p. 83. Academic Press, New York.
- Walter, W. G. *Arch. Psychiat. Nervenkr.* **206**, 309.
- Weiss, J. M. (1971a). *J. Comp. Physiol. Psychol.* **77**, 1.
- Weiss, J. M. (1971b). *J. Comp. Physiol. Psychol.* **77**, 14.
- Weiss, J. M. (1971c). *J. Comp. Physiol. Psychol.* **77**, 22.
- Werblin, F. S. (1971). *J. Neurophysiol.* **34**, 228.