A Neural Theory of Punishment and Avoidance, I: Qualitative Theory

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ABSTRACT

Neural networks are derived from psychological postulates about punishment and avoidance. The classical notion that drive reduction is reinforcing is replaced by a precise physiological alternative akin to Miller's "Go" mechanism and Estes's "amplifier" elements. Cell clusters $\mathscr{A}_f^+$ and $\mathscr{A}_f^-$ are introduced which supply negative and positive incentive motivation, respectively, for classical conditioning of sensory-motor acts. The $\mathscr{A}_f^+$ cells are persistently turned on by shock (on-cells). The $\mathscr{A}_f^-$ cells are transiently turned on by shock termination (off-cells). The rebound from $\mathscr{A}_f^+$ cell activation to $\mathscr{A}_f^-$ cell activation replaces drive reduction in the case of shock. Classical conditioning from sensory cells $\mathscr{S}$ to the pattern of activity playing on arousal cells $\mathscr{A}_f = (\mathscr{A}_f^+, \mathscr{A}_f^-)$ can occur. Sufficiently positive net feedback from $\mathscr{A}_f$ to $\mathscr{S}$ can release sampling, and subsequent learning, by prescribed cells in $\mathscr{S}$ of motor output controls. Once sampled, these controls can be reactivated by $\mathscr{S}$ on recall trials. This concept avoids some difficulties of two-factor theories of punishment and avoidance. Estes' stimulus sampling theory of punishment is neurally interpreted.

Recent psychophysiological data and concepts are qualitatively analyzed in terms of network analogs. These concepts include aspects of relaxation, or elicitation, theory, which claims that an unconditioned response of relief precedes reinforcement; the concept of "effective reinforcement," which notes that shock offset and fear of situational cues can influence reward in opposite ways, as is illustrated by one-way and two-way avoidance tasks; classical and instrumental properties of a CS+ paired with shock, a CS− paired with no-shock, and feedback stimuli contingent on the avoidance response, including transfer of their effects from classical to instrumental conditioning experiments; autonomically nonchalant asymptotic avoidance performance originally motivated by fear; forced extinction of the conditioned avoidance response (CAR) without fear extinction; response suppression without an avoidance response; relief without an avoidance response; opposite effects of contingent and noncontingent punishment on fear and suppression of consummatory responding; punishment hypothesis of avoidance learning, describing rewarding effects of terminating proprioceptive cues that correspond to nonavoidance responses; response (or no-response) generalization from one shock level to a different level; rewarding effect of response-contingent reduction in frequency of shock.

INTRODUCTION

This article derives neural networks from psychological postulates concerning punishment and avoidance. Relevant experiments and theories will be analyzed in terms of network mechanisms. These networks form part of a theory of pattern discrimination and learning which is called the theory of *embedding fields*. The equations of this theory can be derived from psychological postulates and, once derived, can be given an anatomical and physiological interpretation.

The theory introduces a particular method to approach the several levels of description that are relevant to understanding behavior. This is the method of *minimal anatomies*. At any given time, we will be confronted by particular laws for individual neural components, which have been derived from psychological postulates. The neural units will be interconnected in specific anatomies. They will be subjected to inputs that have a psychological interpretation which create outputs that also have a psychological interpretation. At no given time could we hope that all of the more than 10^{12} nerves in a human brain would be described in this way. Even if a precise knowledge of the laws for each nerve were known, the task of writing down all the interactions and analyzing them would be bewilderingly complex and time consuming. Instead, a suitable method of successive approximations is needed. Given specific psychological postulates, we derive the *minimal* network of embedding field type that realizes these postulates. Then we analyze the psychological and neural capabilities of this network. An important part of the analysis is to understand what the network cannot do. This knowledge often suggests what new psychological postulate is needed to derive the next more complex network. In this way, a hierarchy of networks is derived, corresponding to ever more sophisticated postulates. This hierarchy presumably leads us ever closer to realistic anatomies, and provides us with a catalog of mechanisms to use in various situations. The procedure is not unlike the study of one-body, then two-body, then three-body, and so on, problems in physics, leading ever closer to realistic interactions; or the study of symmetries in physics as a precursor to understanding mechanisms of symmetry breaking.

At each stage of theory construction, formal analogs of nontrivial psychological and neural phenomena emerge. We will denote these formal properties by their familiar experimental names. This procedure emphasizes at which point in theory construction, and ascribed to which mechanisms, these various phenomena first seem to appear. No deductive procedure can justify this process of name calling; some aspects of each named phenomenon might not be visible in a given minimal anatomy; and incorrect naming of formal network properties in no way compromises the

formal correctness of the theory as a mathematical consequence of the psychological postulates. Nonetheless, if ever psychological and neural processes are to be unified into a coherent theoretical picture, such name calling, with all its risks and fascinations, seems inevitable, both as a guide to further theory construction and as a tool for more deeply understanding relevant data. Without it, each theory must remain a disembodied abstraction. The following pages will attempt to distinguish clearly among postulates, mathematical properties, factual data, and mere interpretations of network variables.

In Section 2, a terse review of relevant psychological data is given. In Section 3, a review of relevant theory is presented. Section 4 compares some results from Section 3 with Estes's stimulus sampling theory of response amplifiers to help bridge the gap between these two points of view. Section 5 describes the minimal extension of the network derived in Section 3 that is needed to achieve response suppression due to punishment. Section 6 heuristically describes the minimal network mechanism needed to reinforce avoidance responses. Then the data discussed in Section 2 are qualitatively shown to be compatible with this mechanism. Part II of the article will implement the qualitative mechanisms of this part with quantitative analysis of rigorously defined network mechanisms.

2. REVIEW OF DATA

Two main experimental themes were introduced in 1941. Estes and Skinner [25] suggested that emotional responses elicited by a punishing event are *classically* conditioned to the stimuli preceding the event. The conditioned emotional response (CER) then suppresses the punished response. Miller and Dollard [58] claimed that any response associated with the termination of a punishing stimulus is *instrumentally* conditioned by a mechanism of drive reduction. The conditioned avoidance response (CAR) then competes with the punished response. Dunham [21] and Estes [24] review the development of later two-factor theories which combine the CER and CAR mechanisms: after the CER is established by classical conditioning, termination of the aversive conditioned stimulus (CS+) can reinforce the CAR by instrumental conditioning. Estes [24] also outlines a theory of punishment within the framework of stimulus sampling theory. Our neural theory is analogous to the Estes theory in several respects, and provides a neural interpretation of the stimulus sampling formalism. Hence the Estes theory will be briefly reviewed before various data relevant to the theory are cited.

Estes notes that the CER concept accounts for response suppression due to punishment, and its relation to shock intensity, duration, delay, and differences between contingent and noncontingent punishment. It also

accounts for response recovery after punishment ceases, which shows that the time scales of response suppression and of response extinction differ. Estes critically analyzes the CAR concept. Originally the CAR was thought to maintain response suppression by competing with the punished response (see also Dinsmoor [20] and Solomon [69]). Estes notes, however, that the CAR is not a necessary condition for response suppression. Whereas response suppression is readily achieved by intense shocks, avoidance is often hard to achieve with similar shocks; the time courses of suppression and of avoidance learning are not similar; and the same punishing stimulus can have different effects, depending on whether the punished response has previously been maintained by a positive reward (for example, food) or by escape and avoidance.

Estes concludes that punishment weakens motivational support for the punished response. He states that the occurrence of a response requires summation of input from external stimulus and internal drive sources. Drives and rewards serve as response *amplifiers*. On a learning trial, the organism $\mathcal{O}$ draws a sample of available discriminative cues and scans these until an element is processed which is connected with a permissible response. This response will be evoked only if an amplifier element appropriate to the response is simultaneously scanned. Stimuli can be conditioned to amplifier elements by contiguity, and the base rate of amplifier elements associated with a given drive increases as $\mathcal{O}$'s need (for example, hunger) increases. Thus $\mathcal{O}$'s prior conditioning history and present state of deprivation interact to generate responding.

Positive and negative drives are treated symmetrically in Estes's theory. He assumes that the negative flight-attack system and the positive drive systems interact by reciprocal inhibition. Thus if a stimulus is conditioned to pain, negative amplifier elements can prevent positive amplifier elements from releasing responses. Qualitatively similar concepts have been presented by Miller [57], who calls his amplifier elements "Go" mechanisms, and by Livingston [49], who discusses "Now Print" mechanisms. Logan [50] also argues in this direction when he claims that rewards "excite" rather than "strengthen" habits by providing "incentive motivation" that favors their execution. In particular, painful stimuli produce *negative* incentive motivation, and choice is based upon the *net* incentive motivation (for example, positive versus negative) that is associated with each alternative.

Maier, Seligman, and Solomon [51] review experiments confirming that fear is classically conditioned, and that Pavlovian conditioned responses motivate instrumental behavior. They show that a CS+ paired with shock elicits fear and a CS− paired with no-shock inhibits fear and depresses fear-motivated behavior. They note that whereas escape from

fear ordinarily motivates avoidance learning, escape from fear is not necessary to maintain avoidance. For example, at asymptotic avoidance performance, subjects are nonchalant responders [70]; subjects need not display a CER to the CS+ [47]; subjects need not show autonomic arousal to the CS+ [6]; and subjects can have an avoidance latency that is too short to allow autonomic arousal [71].

Various refinements in the concept of how instrumental reinforcement occurs have been noted. Many of these, in one form or another, pay homage to the influence of situational cues, and thereby emphasize the need for theories in which parallel processing of events can be conveniently treated. The punishment hypothesis of avoidance learning states that nonavoidance responses acquire conditioned aversive properties when they are paired with aversive stimuli; the avoidance response is rewarded by termination of the proprioceptive stimulation associated with nonavoidance responses [20, 56, 67]. Presentation of a nonredundant feedback stimulus (FS.) that signals a no-shock interval after the avoidance response occurs is also reinforcing; the FS can act independently of the CS+ [10, 11, 48, 68]. Similarly, a CS− that predicts a no-shock interval can acquire positive rewarding properties that antagonize the negative rewarding properties of CS+ in a symmetric fashion [21, 22, 42, 64, 65, 74]. Elicitation theory suggests that removing the aversive stimulus is not immediately rewarding; an unconditioned response of relief or relaxation must occur after the aversive stimulus or other situational cues are removed [17, 52, 66]. The relaxation concept emphasizes that fear can be conditioned both to the CS+ and to situational cues which together influence learning and performance. Thus the amount of *effective* reinforcement for avoidance learning is positively related to the amount of fear reduction occurring with CS+ termination and negatively related to the amount of fear of situational cues [54, 55]. This suggestion is related to various pain analgesic effects, including the greater reinforcing effect of reducing 200 units of shock to 0 units than 400 units of shock to 200 units, or of terminating shock in the absence, rather than in the presence, of loud noise [14, 27, 60]. Related influences of situational cues on drive reduction have been noted in a variety of experimental situations [2, 13, 19, 53].

How do the classical and instrumental conditioning mechanisms interact? Various data note that classical and instrumental contingencies *can* be manipulated independently, but in general the CER and CAR systems interact in subtle ways. For example, experiments have found transfer of CS+ and CS− effects from classical to instrumental situations [4, 18, 65, 74, 75], partial reinforcement effects familiar from instrumental conditioning in the classical conditioning of the CER [45], and forced extinction of avoidance responding in the absence of fear extinction [15].

Moreover, in a yoked design, a subject that is contingently shocked can yield less instrumental responding than one which is noncontingently shocked, although both have similar autonomic responses [77]. Contingent versus noncontingent shock can also influence choice behavior, based on fear, and degree of response suppression in opposite directions [63]. Whereas a fearful situation can yield immobile crouching, a discrete approaching fearful cue can elicit active avoidance [8, 9]. Avoidance responses are more rapidly acquired if they are unconditionally elicited by the aversive stimulus [12]. In a one-way avoidance task, prior fear conditioning can facilitate avoidance learning [4], whereas in a two-way task it can interfere with avoidance responding [76]. A bizarre interaction between CER and CAR systems arises in self-punitive avoidance or "vicious circle" behavior, which denotes delayed extinction of the avoidance response as a result of noncontingent punishment during extinction trials [5, 28].

If hypothalamic stimulation elicits a given behavior, its offset transiently elicits an opposite behavior [16, 29, 73]. A transient rebound mechanism from mutually inhibitory "on-cells" to "off-cells," in combination with a mechanism of classical conditioning at both cell aggregates, will be used to discuss both classical and instrumental, negative and positive "incentive motivational" conditioning in our networks.

Many of the foregoing experiments consider the influence of external environmental cues. Internal "environmental" inputs, due to interactions between several drive states, are also of great importance, as was noted by Estes [24] and Logan [50] in their discussions of the competition between positive and negative drive states to form a consensus on which overt responding is based. Hull [46] originally argued for the additivity of drives: if a response is reinforced under one drive and if a second relevant drive is later substituted, then response strength under the second drive benefits from prior training under the first drive. Porter and Miller [62] demonstrated such an effect with alternate day training using food and water. Mixtures of shock and food or water yield suppressive rather than additive effects, however [3, 7, 59, 78]. Such results have provided the rationale for reciprocally inhibiting unwanted behavior traits in psychotherapy [26, 79]. They show that the global anatomy of drive states is no less important than the global anatomy of sensory filters in determining which responses will be released. Related data will also be discussed as the theory is developed.

3. THEORETICAL REVIEW

Two stages of the theory have been derived elsewhere. The present stage builds on these stages. Hence they will be reviewed as needed herein.

Stage one is derived in [31] and reviewed in [37]. Stage two is derived in [38]. Both derivations are based on familiar psychological facts taken as fundamental postulates. In this sense the theory is deductive and attempts to show that various nonobvious phenomena are manifestations of familiar facts taken together in a proper formal setting.

The main postulates used to derive stage one are rudimentary facts about Pavlovian conditioning. These postulates are briefly stated here for completeness. The references contain complete details.

Postulate I: Presentations Induce Perturbations. This postulate addresses the question: How does $\mathcal{O}$ internally represent the presentation of a given behaviorally indecomposable stimulus at a prescribed time?

Postulate II: Distinguishing Order. How does $\mathcal{O}$ learn that a given unconditioned response (UCR) follows a prescribed conditioned stimulus (CS), and not some other response?

Postulate III: Reproducing Order. How does the distinguished (learned) CS→UCR pathway elicit the proper output in response to a given CS input?

Postulate IV: Independence of Lists in First Approximation. How does $\mathcal{O}$ prevent massive response interference from unpresented stimuli that are internally represented in $\mathcal{O}$ when short lists are being learned?

The other conditions are either formal consequences of these or are general constraints to make the mathematics as simple, continuous, and linear as possible. These postulates generate surprisingly powerful neural networks, which for example can discriminate, learn, remember, and perform arbitrarily complex sequences of events [32, 35] and give rise to analogs of various serial learning phenomena [33, 40, 41].

In their simplest form, the networks are defined as follows.

$$\dot{x}_i(t) = -\alpha_i x_i(t) + \sum_{k=1}^{n} [x_k(t-\tau_{ki}) - \Gamma_{ki}]^+ \beta_{ki} z_{ki}(t) - \sum_{k=1}^{n} [x_k(t-\sigma_{ki}) - \Omega_{ki}]^+ \gamma_{ki} + C_i(t) \tag{1}$$

and

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

where $i, j, k = 1, 2, \ldots, n$ and $[\xi]^+ = \max(\xi, 0)$ for any real number ξ. $x_i(t)$ denotes the stimulus trace (or average membrane potential) at time t of the cell body (or cell body cluster) v_i, and $z_{jk}(t)$ denotes the memory trace (or associational strength, or excitatory transmitter production activity) at time t of the synaptic knob (or knobs) N_{jk} found at the end of the axon(s) e_{jk} from v_j to v_k. The term $-\alpha_i x_i$ in (1) represents a passive

exponential decay of potential. The term $[x_k(t - \tau_{ki}) - \Gamma_{ki}]^+\beta_{ki}$ in (1) is proportional to the spiking frequency released into e_{ki} in the time interval $[t - \tau_{ki}, t - \tau_{ki} + dt]$. Γ_{ki} is the spiking threshold, β_{ki} is proportional to the excitatory axonal connection strength from v_k to N_{ki}, and τ_{ki} is the time required for spikes to travel from v_k to N_{ki}. The term $\Sigma_{k=1}^n[x_k(t - \tau_{ki}) - \Gamma_{ki}]^+\beta_{ki}z_{ki}(t)$ in (1) is the total excitatory input from other cells to v_i at time t. At an excitatory synapse ($\beta_{ki} > 0$), spiking frequency couples multiplicatively to transmitter $z_{ki}(t)$ to release transmitter that perturbs $x_i(t)$, and all such signals combine additively at v_i. The term $\Sigma_{k=1}^n[x_k(t - \sigma_{ki}) - \Omega_{ki}]^+\gamma_{ki}$ is the total inhibitory input from other cells to v_i at time t, with γ_{ki} the inhibitory axonal connection strength from v_k to v_i. The term $C_i(t)$ is the experimental input (or stimulus) to v_i at time t.

In (2), the memory trace cross-correlates the presynaptic spiking frequency which reaches N_{jk} from v_j at time t with the value $x_k(t)$ of average potential at v_k at this time. Passive exponential decay of memory, due to the term $-\delta_{jk}z_{jk}$, can also occur. Other decay laws have also been analyzed [36].

The notion that synapses are facilitated by joint presynaptic and postsynaptic activity goes back to Hebb [43], but the details of learning in the heuristic Hebbian nets and our rigorously defined systems are very different. This is due to the combined effect of *all* terms in Eq. 1 and 2, which cannot be analyzed by heuristic definitions and arguments. Indeed, elementary properties of learning due to alterations in synaptic weights of the present type seem to have eluded heuristic thinking on the subject. Even in the simplest systems, learning can be "nonlocal" in the sense that a physiological experimentalist could not find out what was being learned at a given cell by measuring the processes going on at that cell.

Note by Eq. 2 that no learning occurs in N_{jk} if $\delta_{jk} = \varepsilon_{jk} = 0$, since then z_{jk} is constant. β_{jk} can nonetheless be chosen positive. Then signals can flow from v_j to v_k if also z_{jk} is positive. We will draw N_{jk} as an arrowhead if learning cannot occur in N_{jk}, and as a filled half-circle if learning can occur in N_{jk}. By (1), no learning occurs in inhibitory synaptic knobs (at least for present purposes). Thus inhibitory axons always terminate in an arrowhead (see Fig. 1).

Stage two of the theory invokes more sophisticated properties of Pavlovian conditioning. A formal representation of these properties includes influences of motivation and reward. Thus the theory suggests that important aspects of classical and instrumental conditioning share common local mechanisms at individual cells, even though different cell aggregates—including different discrimination mechanisms—can be activated by different types of conditioning experiments. The derivation of stage two will be reviewed as a point of departure for the present work.

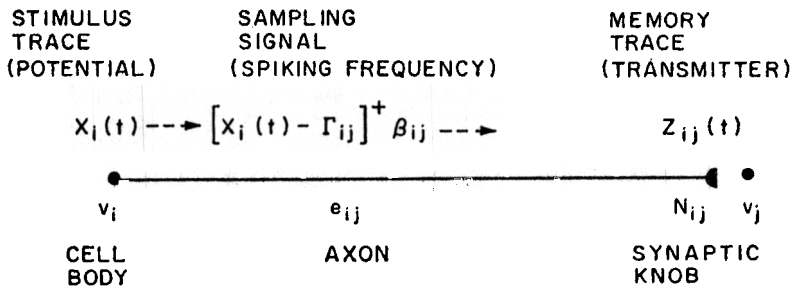

FIG. 1. Psychophysiological interpretation of network variables.

Five postulates are the basis of stage two:

Postulate V. Practice makes perfect.

Postulate VI. The time lags between CS and unconditioned stimulus (UCS) on successive trials can differ.

Postulate VII. The UCR can be elicited by the CS alone on recall trials.

Postulate VIII. A given CS can be conditioned to UCRs corresponding to any of several drives (for example, bell→salivation or bell→fear).

Postulate IX. Rate of consummatory responding is influenced by the state of deprivation.

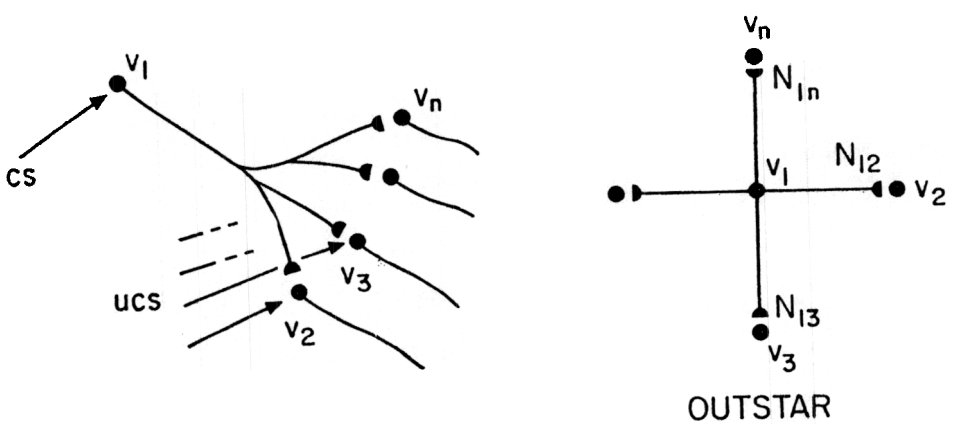

FIG. 2. An outstar.

Postulate V is a truism that will be implemented in conjunction with Postulate VI. Postulates VI and VII are observations about the Pavlovian conditioning paradigm. Postulates VIII and IX are obvious. Such trivialities would yield little directive in a theoretical vacuum. Applied to the theory available from stage one, however, they are powerful guides to constructive theorizing.

Stage one helps us because its mathematical analysis reveals unsuspected formal properties. These properties include a concrete physiological interpretation of stimulus sampling theory. To illustrate this, we consider the simplest embedding field that can learn by Pavlovian conditioning;

namely, an *outstar* [36, 37]. Let one CS-activated cell v_1 send equal signals to its synaptic knobs N_{1i} which abut the UCS-activated cells $V = \{v_i: i = 2, 3, \ldots, n\}$. See Fig. 2.

Mathematical analysis of the outstar reveals the following properties, among others [36]. v_1 can learn and perform at V a *spatial pattern*; that is a UCS input to V of the form $C_i(t) = \theta_i C(t)$, where θ_i is the fixed, but otherwise arbitrary, relative pattern intensity at v_i and $C(t)$ is the total pattern intensity, which can fluctuate wildly in time. In particular, $\theta_i \geqslant 0$ and $\Sigma_{k=2}^{n} \theta_k = 1$. The relative memory trace $Z_{1i} = z_{1i}(\Sigma_{k=2}^{n} z_{1k})^{-1}$ is attracted toward ("encodes") the pattern weight θ_i at a rate that depends on CS and UCS input rate, intensity, relative timing, and related factors. The sizes of the absolute memory traces z_{1i} also depend on these factors.

The relative memory traces $Z = (Z_{12}, Z_{13}, \ldots, Z_{1n})$ are attracted toward the pattern weights $\theta = (\theta_2, \theta_3, \ldots, \theta_n)$ only at times when the synaptic knobs N_{1i} receive CS-activated spikes from v_1. This is the property of "stimulus sampling" in an outstar: v_1 samples the patterns playing on V by emitting signals at prescribed times. The relative memory traces Z, which form a probability distribution at each time t, are the "stimulus sampling probabilities" of an outstar [36]. Whenever v_1 samples V, the memory traces in its synaptic knobs begin to learn the spatial pattern playing on V at this time. If a sequence of patterns (that is, a space–time pattern) plays on V while v_1 is sampling, then v_1's synaptic knobs learn a weighted average of all the patterns, rather than any single spatial pattern. Thus if an outstar samples V while a long sequence of spatial patterns reaches V, then after sampling terminates, the sampling probabilities Z can be different from any one of the spatial patterns. On recall trials, a CS input to v_1 creates equal signals in the axons e_{1i}. These signals flow down to the N_{1i}. In N_{1i}, the signal interacts with the memory trace z_{1i} to reproduce at the cell v_i an output proportional to Z_{1i}. In this way, recall trials reproduce at V the weighted average of sampled patterns that was encoded on learning trials.

Given these facts, stage two considers the typical situation in which a space–time pattern is the UCS input to V on a sequence of N learning trials. In other words, on each trial a sequence $\theta^{(1)}, \theta^{(2)}, \theta^{(3)}, \ldots, \theta^{(N)}$ of spatial patterns with weights $\theta^{(i)} = (\theta_2^{(i)}, \theta_3^{(i)}, \ldots, \theta_n^{(i)})$ is the UCS delivered to V, $i = 1, 2, \ldots, N$. In this situation, an outstar anatomy does not suffice to achieve Postulate V if Postulate VI also holds. In other words, a given cell v_1 cannot learn a definite spatial pattern $\theta^{(i)}$ chosen from the UCS sequence if the CS alone can fire v_1 on successive learning trials. To see this, consider sampling by v_1 of $\theta^{(1)}$ for definiteness. v_1 can learn $\theta^{(1)}$ only if v_1 fires briefly a *fixed* time before the onset on $\theta^{(1)}$ on *every* trial, and if the signals from v_1 reach V only when $\theta^{(1)}$ plays on V. This will not

happen if the CS alone can fire v_1 while Postulate VI holds, since signals from v_1 will reach V on successive trials while spatial patterns $\theta^{(i)}$ other than $\theta^{(1)}$ play on v_1. Thus Z will learn a weighted average of the patterns $\theta^{(i)}$ rather than $\theta^{(1)}$.

To avoid noisy sampling, the outstar must be embedded in a larger network. v_1 must be prevented from firing unless it simultaneously receives a CS input *and* an input controlled by the UCS which signals that the UCS will arrive at V a fixed time interval later. This is accomplished in two steps: let the UCS activate axons leading to v_1 that deliver an input to v_1 a fixed time before the UCS arrives at V; and set the common spiking threshold Γ_1 of all v_1's axon collaterals so high that v_1 can fire only if it simultaneously receives large CS and UCS-controlled inputs. Then, on every trial, v_1 can fire and begin to sample the spatial pattern $\theta^{(1)}$ as it arrives at V, if also the CS has been presented. Grossberg [35] discusses some inhibitory mechanisms that guarantee brief v_1 outputs in response to even prolonged CS plus UCS inputs.

All cells in the network which can sample V receive UCS-activated axons, for the reasons given above. In other words, there exists a UCS-activated nonspecific arousal of CS-activated sampling cells. These cells are polyvalent cells, or cells that are influenced by more than one modality, such as the sound of a bell (CS) and the smell of food (UCS). The polyvalent cells fire only if the sum of CS and UCS inputs is sufficiently large. Grossberg [38] reviews physiological data relevant to this concept.

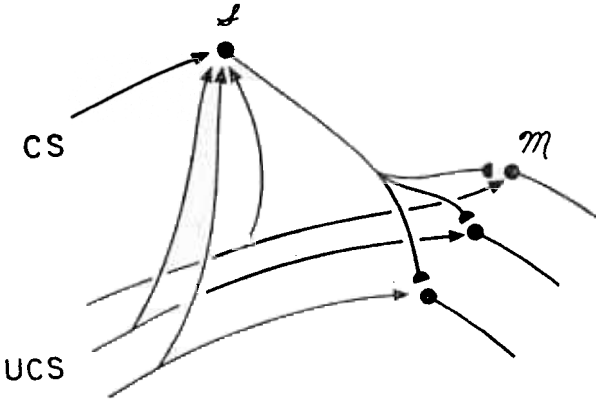

FIG. 3. UCS-activated arousal of sampling cells

Some suggestive terminology is now introduced by denoting v_1-type cells generically by $\mathcal{S}$, for "sensory cells" or "sensory representation," and V-type cells by $\mathcal{M}$ for "motor cells" or "motor representation." This distinction, of course, has no absolute significance, since both v_1 and V contribute to sensory and motor processing. It is nonetheless convenient (see Fig. 3).

Postulate VII is invoked on recall trials. After learning has taken place, the CS alone can elicit performance on recall trials. Thus the CS alone can fire cells in $\mathscr{S}$ on recall trials. But $\mathscr{S}$ cells can only fire if inputs along two axon paths converge simultaneously on them. The UCS is not available on recall trials to activate one of these paths. Only the CS is available. How does CS–UCS pairing on learning trials enable the CS to gain control over the UCS$\rightarrow\mathscr{S}$ pathway on recall trials? This dilemma imposes the concept of "conditioned arousal," which will later be specialized as "conditioned incentive motivation." Namely, CS–UCS pairing during learning trials allows the CS to gain control over the nonspecific arousal channel via Pavlovian conditioning (that is, by cross-correlating presynaptic spiking frequencies and postsynaptic potentials at suitable synaptic knobs). Conditioning of nonspecific arousal at these synaptic knobs takes place while specific motor patterns are learned in the $\mathscr{S}\rightarrow\mathscr{M}$ synaptic knobs. Consequently, on recall trails, the CS can activate two input channels: unconditioned specific inputs to $\mathscr{S}$ and conditioned nonspecific arousal inputs to $\mathscr{S}$. At cells in $\mathscr{S}$ where these two inputs converge, the cell potential can be driven above its spiking threshold. These cells can fire, yielding signals along $\mathscr{S}\rightarrow\mathscr{M}$ axons which activate the $\mathscr{S}\rightarrow\mathscr{M}$ synaptic knobs and reproduce at $\mathscr{M}$ the patterns encoded in these knobs. In this way, a CS can acquire UCS properties, and thus aspects of higher-order conditioning emerge as a consequence of facts VI and VII.

After a CS can activate the arousal pathway, it has UCS properties; it can serve as the UCS for a new CS in a later learning experiment. The transition from CS to UCS in these networks is effected by an alternation (not necessarily a strengthening!) of extant pathways, rather than by the creation of new pathways. Thus both CS and UCS inputs are processed in parallel pathways ("path equivalence"), except possibly the primary UCS input (for example, taste of food) on which a chain of conditioning experiments can be built. In particular, "higher-order" UCS inputs, as well as CS inputs, are delivered to $\mathscr{S}$.

The cells $\mathscr{A}$ at which conditioning of arousal takes place are neither $\mathscr{S}$ cells nor $\mathscr{M}$ cells. This is because the $\mathscr{S}$ cells must be aroused before they sample the activity of $\mathscr{M}$ cells, and $\mathscr{M}$ cell activation must await the onset of sampling—and thus prior firing—by $\mathscr{S}$ cells, or else $\theta^{(1)}$ cannot be learned. Similar arguments have been used to prove that at least two successive cell sites are needed in each sensory representation. The first site receives the CS input and thereupon sends signals to $\mathscr{A}$ and to the second site. The second site can fire to $\mathscr{M}$ only if it also receives a feedback signal from $\mathscr{A}$ (see Fig. 4). Sensory representations with more than two cell sites are also possible, but the theory restricts itself to the construction of minimal anatomies. As new requirements are imposed, the anatomy can

be expanded to include new properties. Using this strategy, we construct a hierarchy of ever more elaborate and realistic minimal anatomies, such that each anatomy parsimoniously represents the processing of which it is capable.

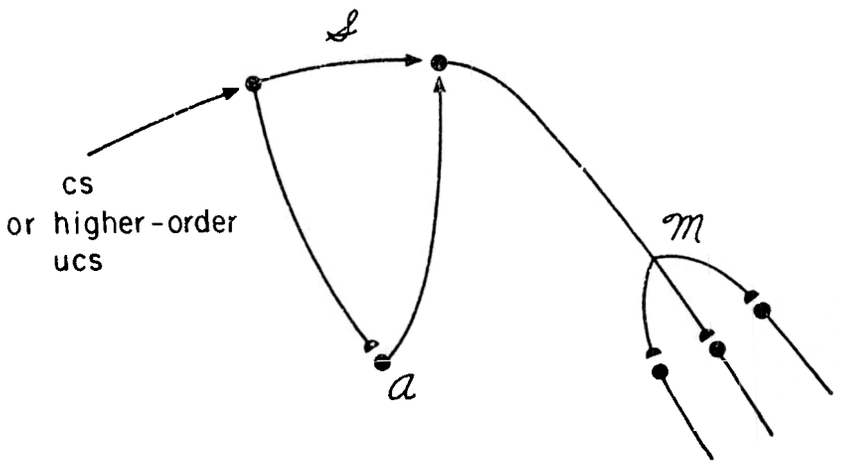

FIG. 4. Minimal representation of arousal pathway.

The $\mathscr{A}$ cells will be interpreted as network analogs of hypothalamus, reticular formation, and related brain area implicated in arousal and reinforcement tasks. Certainly $\mathscr{A}$ cells are at best rudimentary analogs of these neural regions. Nonetheless, the formal tasks which $\mathscr{A}$ cells perform are strikingly reminiscent of facts known about their neural counterparts. Moreover, the interactions between $\mathscr{A}$ cells will become increasingly complex and realistic as the derivation continues. Given this interpretation, $\mathscr{A}$ cells will include drive-activated cells. For example, when a bell (CS) is conditioned to elicit salivation (UCR), it activates the $\mathscr{A}$ cells corresponding to hunger. Now invoke Postulate VIII. Postulate VIII directs us to further expand our minimal network to include several subsets of $\mathscr{A}$ cells, such that each subset subserves a different "drive." These $\mathscr{A}$ subsets can overlap if their corresponding drives are not mutually independent: compare hunger and thirst. For convenience of representation, however, we draw them as individual points in Fig. 5. By Postulate VIII, a given sensory event can be conditioned to any of several drive contingencies. Thus, each $\mathscr{S}$ in the minimal construction will send axons to several subsets of $\mathscr{A}$ cells. Each $\mathscr{A}$ subset, in turn, sends axons nonspecifically to $\mathscr{S}$ cells; otherwise the several drives could not control nonspecific arousal signals from $\mathscr{A}$ to $\mathscr{S}$ capable of releasing signals in particular $\mathscr{S} \rightarrow \mathscr{M}$ pathways (see Fig. 5).

Postulate IX imposes a new constraint on the firing of $\mathscr{A}$ cells. If an $\mathscr{A}$ cell can always fire in response to conditioned arousal inputs from $\mathscr{S}$ cells alone, then an $\mathscr{A}$ cell can always elicit (say) hunger-specific motor activity,

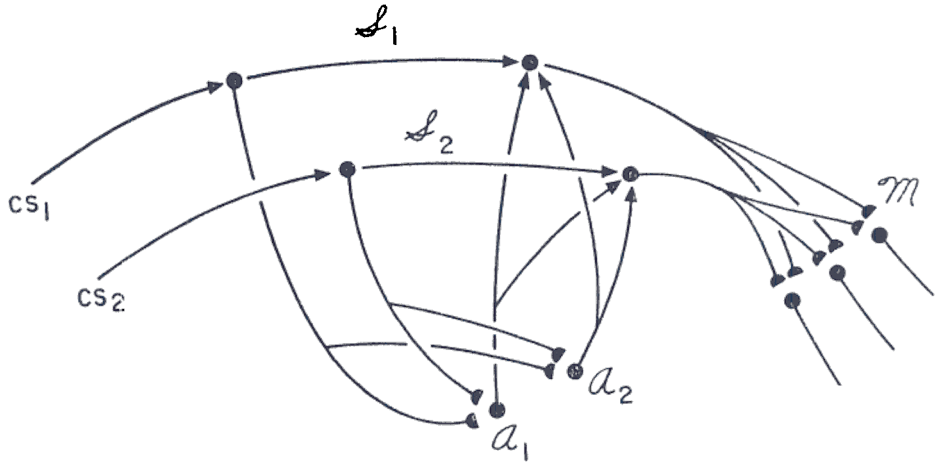

FIG. 5. Spatially distributed arousal loci.

even if $\mathscr{O}$ is not hungry, whenever food is presented. This property would kill $\mathscr{O}$. The difficulty is formally analogous to allowing an $\mathscr{S}$ cell to fire in the absence of its CS input. Maladaptive $\mathscr{A}$ cell firing of this kind can be easily prevented, just as in the $\mathscr{S}$ cell case. In the $\mathscr{S}$ cell case, an $\mathscr{S}$ cell can fire to $\mathscr{M}$ only if it simultaneously receives a nonspecific input from $\mathscr{A}$ and a specific sensory input. Require analogously that an $\mathscr{A}$ cell can fire only if it simultaneously receives a nonspecific input from $\mathscr{S}$ (for example, a conditioned input from $\mathscr{S}$ or a primary UCS input) and a specific sensory input. In the $\mathscr{A}$ cell case, the sensory input is interpreted to be a drive input whose source is within $\mathscr{O}$. The size of this input indicates the level of this drive in $\mathscr{O}$ through time. This restriction on $\mathscr{A}$ cell firing is achieved by setting the spiking threshold of $\mathscr{A} \rightarrow \mathscr{S}$ axons so high that only the sum of sufficiently large inputs from $\mathscr{S}$ and from internal drive sources can fire an $\mathscr{A}$ cell (see Fig. 6).

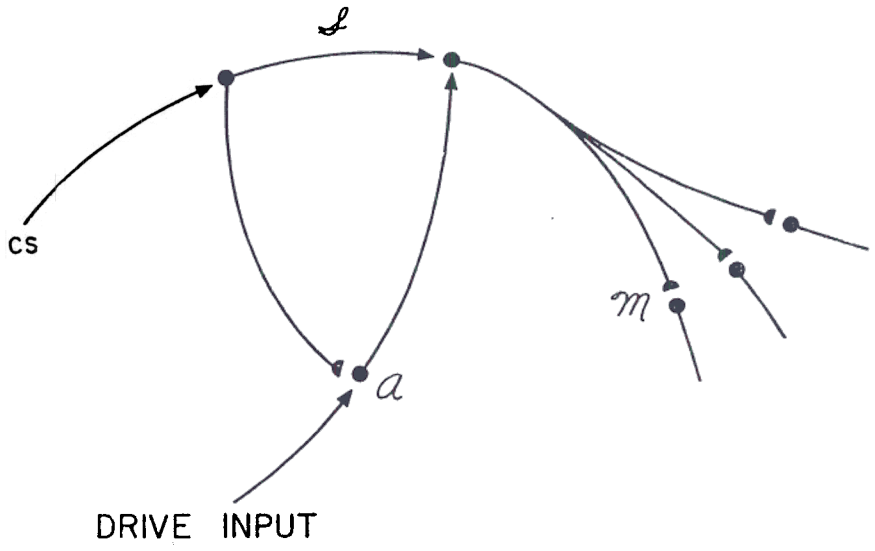

FIG. 6. The site of drive input action.

Now $\mathscr{A}$ cells are also "sensory" cells, but their sensory inputs describe the internal state of $\mathscr{O}$ rather than the external state of the world. Grossberg [38] develops these simple ideas and cites relevant data. Noteworthy is the possibility of learning to push a lever persistently to deliver electric shocks to a (consummatory) drive representation without reducing the internal drive input (no "drive reduction"), as Olds and his collaborators [61] have reported.

The foregoing construction is supported by rigorous mathematical theorems that describe the interaction of any number of cells, interconnected in prescribed anatomies, whose physiological laws can sustain perfect learning in response to network noise and inputs of complicated form [35]. For example, in Fig. 6, any number of cells in $\mathscr{S}$ can sample any number of cells in $\mathscr{A}$, where the $\mathscr{A}$ cells can receive primary UCS inputs, internal drive inputs, and/or conditioned inputs. This situation is covered by theorems in [32, 39] on completely nonrecurrent anatomies. The same theorems cover the case of $\mathscr{S} \rightarrow \mathscr{M}$ sampling. These are the only places in Fig. 6 where learning occurs. It remains only to guarantee that the thresholds and other parameters can be set to restrict the times at which $\mathscr{S} \rightarrow \mathscr{A}$, $\mathscr{A} \rightarrow \mathscr{S}$, and $\mathscr{S} \rightarrow \mathscr{M}$ signals occur. Some further network structure is needed. The main requirements will be discussed in Part II of this article.

4. COMPARISON WITH STIMULUS SAMPLING THEORY

The connection between the network of Fig. 6 and Estes's sampling theory is striking. CS inputs to $\mathscr{S}$ cells replace sampling of discriminative cues. Amplifier elements are replaced by $\mathscr{A}$ cell clusters. Sampling of amplifier elements is replaced by signals from $\mathscr{S}$ cells to $\mathscr{A}$ cells whose relative weights are subject to change by conditioning at the $\mathscr{S} \rightarrow \mathscr{A}$ synaptic knobs. These changes in synaptic weight correspond to changes in stimulus sampling probabilities. The base rate of amplifier elements appropriate to a given drive is replaced by graded internal drive ("homeostatic") inputs. The fact that the base rate of amplifier elements does not yield overt responding corresponds to the requirement that conditioned $\mathscr{S} \rightarrow \mathscr{A}$ inputs must summate with internal drive inputs to exceed the $\mathscr{A}$ cell spiking thresholds.

An important general difference between the two formulations can be cited. Estes provides an abstract probabilistic psychological model, whereas the present model is a concrete deterministic psychophysiological model operating in real time. The determinism of this model does not preclude a study of random factors of several types: the network equations describe the evolution of suitable averages through time; the networks can deal with noise, burst or refractory periods in spiking, suitable fluctuations in network parameters, and so on [34, 39]; even a random experimental

schedule defines a definite input sequence, and the networks can deal with suitable classes of complex inputs. The determinism of the present model means merely that a definite anatomy exists over which definite physiological laws operate. The operation of this model in real time is theoretically important, since many neural processes (and their psychological analogs) vary along several different time scales within numerous parallel channels whose interaction is hard to understand on an abstract sampling space of stimuli and responses. To represent such interacting processes conveniently, one often needs to know the "internal anatomy of the flow." The interaction between punishment and avoidance seems to be of this type.

5. SUPPRESSION BY PUNISHMENT

Our previous discussion yields a network $\mathcal{O}$ which can learn and perform consummatory responses under suitable constraints. This construction does not suffice to prevent consummatory responses if environmental contingencies change so that the response yields aversive results. The construction will now be extended to include this crucial possibility. We will consider the following situation for definiteness. Suppose that a CS (bell) which was once a cue for food is now a cue for shock. How does $\mathcal{O}$ prevent itself from inappropriately carrying out food-consummatory behavior in response to the CS and thereby getting shocked? To implement our construction we will use the following postulate, which prevents $\mathcal{O}$ from indiscriminately learning unsuccessful responses.

Postulate X. $\mathcal{O}$ does not (readily) learn escape responses that do not terminate shock.

Our construction is, of course, constrained by the network that has already been derived, since the postulates from which this network emerged still hold. In Fig. 6, consummatory behavior is modifiable by two parallel conditioning processes: Conditioning of nonspecific $\mathscr{A} \rightarrow \mathscr{S}$ arousal via the $\mathscr{S} \rightarrow \mathscr{A}$ synaptic knobs, and conditioning of specific motor patterns via the $\mathscr{S} \rightarrow \mathscr{M}$ synaptic knobs. Which of these conditioning processes must be supplemented to fulfill Postulate X? We proceed by asking for the minimal possible change: Can $\mathcal{O}$ recondition the $\mathscr{S} \rightarrow \mathscr{M}$ pathway without altering the $\mathscr{S} \rightarrow \mathscr{A}$ pathway? The answer will be "no" for the following reasons. The $\mathscr{S} \rightarrow \mathscr{M}$ pathway can be reconditioned in two ways:

1. *Passive Extinction.* Prevent firing of the $\mathscr{S} \rightarrow \mathscr{M}$ pathway for long time intervals. Then transmitter levels in $\mathscr{S} \rightarrow \mathscr{M}$ synapses can slowly decay to the level of network random noise. This process takes too long, however, to prevent $\mathcal{O}$ from violating Postulate X, and there exist workable

transmitter laws in which no passive extinction occurs [36]; for example laws such as

$$\dot{z}_{jk}(t) = \{-\delta_{jk} z_{jk}(t) + \varepsilon_{jk} x_k(t)\}[x_j(t - \tau_{jk}) - \Gamma_{jk}]^+, \tag{3}$$

in which perfect memory exists until practice or recall trials, or random bursts of presynaptic spiking, occur. Also, decay can be retarded or even reversed if recall trials intermittently occur when $\mathcal{O}$ is hungry. Then the $\mathcal{S} \to \mathcal{M}$ pathway is activated and the $\mathcal{S} \to \mathcal{M}$ synaptic levels are restored to supranoise levels by transmitter potentiation, without destroying the encoded motor pattern ("posttetanic potentiation"; Eccles [23].)

2. *Interference Theory of Forgetting* [1]. Let every occurrence of shock input generate a new UCR pattern at $\mathcal{M}$, which is incompatible with eating. If the CS also occurs at these times, and $\mathcal{O}$ is hungry, then $\mathcal{S}$ will sample the new pattern at $\mathcal{M}$ and the $\mathcal{S} \to \mathcal{M}$ synaptic knobs will encode the new UCR pattern. Thereafter, whenever the bell rings and $\mathcal{O}$ is hungry, the new motor pattern will be released, rather than eating. This mechanism has severe faults during recall trials. First, $\mathcal{O}$ cannot learn specific avoidance tasks, since the shock—and not a specific avoidance response—controls the competing UCR at $\mathcal{M}$. Second, $\mathcal{O}$ remains conditioned to the hunger $\mathcal{A}$ cells. Thus $\mathcal{O}$ will indulge in general (for example, autonomic) preparations for eating without being able to eat. Third, $\mathcal{O}$ is maladaptively fearless, since only positive consummatory drives are conditionable to the CS. Counterconditioning along a new $\mathcal{S} \to \mathcal{A}$ pathway is clearly needed. Denote the new subset of $\mathcal{A}$ cells by $\mathcal{A}_f$.

Let shock create an input at the subset $\mathcal{A}_f$. Let this input be a monotone increasing function of shock intensity. Again we are called upon to psychologically interpret a formal operation. In this case, associate activation of the cells $\mathcal{A}_f$ by shock with production within $\mathcal{O}$ of a comparable amount of fear. This interpretation introduces fear into the network using a minimum of network machinery. Given this interpretation, activating conditioned $\mathcal{S} \to \mathcal{A}_f$ synaptic knobs will yield a CER, both by eliciting fear in $\mathcal{O}$ and, perhaps, by activating autonomic expressions of fear through $\mathcal{A}_f$. Let $\mathcal{A}_h$ denote the subset of $\mathcal{A}$ cells that subserves hunger, and consider Postulate X in this context.

Why is Postulate X needed? Suppose that it does not hold. Then $\mathcal{O}$ can learn all unsuccessful escape responses. Efficient avoidance performance would therefore be unlikely, since mistakes are more likely than correct responses during a period of frantic trial and error in a complex experimental chamber. $\mathcal{O}$ would, at best, learn to execute the avoidance response as the terminal response in a long chain of previously learned incorrect responses. To prevent this from happening. $\mathcal{A}_h$ cells cannot be the only $\mathcal{A}$ cells that fire to $\mathcal{S}$ when the CS occurs and shock is on. For if they were,

not only could maladaptive consummatory responses be performed given the CS and sufficient hunger, but also all erroneous escape responses could be sampled and learned by $\mathscr{S} \rightarrow \mathscr{M}$ synaptic knobs with the $\mathscr{A}_h$ cells as the arousal source. The effect of $\mathscr{A}_h$ arousal on $\mathscr{S}$ must be inhibited while shock is on. The $\mathscr{A}_f$ cells are the minimal source of this inhibition. Hunger and fear arousal cells thus reciprocally inhibit each other, as Logan suggested in his discussion of net incentive motivation. Figure 7 displays two inhibitory mechanisms. Consider Figs. 7a and 7b when the synaptic knobs of v_1 are active. At these times, the sampling probabilities $Z(t)$ learn a weighted average of the spatial patterns $\theta(t) = (\theta_h(t), \theta_f(t))$ that reach $\mathscr{A}_h$ and $\mathscr{A}_f$. Thus the probabilities learn the net balance of hunger and fear during times when v_1 samples $\mathscr{A}$. $\mathscr{A}_h$ sends excitatory feedback signals to v_2, whereas $\mathscr{A}_f$ sends inhibitory signals to v_2. v_2 requires the sum of two excitatory inputs, one from v_1 and one from $\mathscr{A}_h$, in order to fire. As the inhibitory signal from $\mathscr{A}_f$ grows, it cancels the effect of the $\mathscr{A}_h$ input, and prevents v_2 from firing. Thus v_2 cannot sample and learn the motor patterns reaching $\mathscr{M}$ at times when $\mathscr{A}_f$ feedback is active. This is true of every sensory representation.

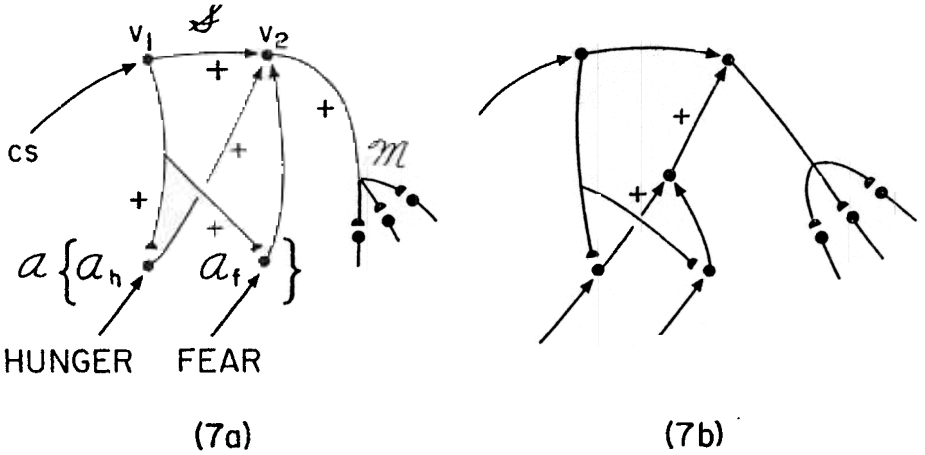

FIG. 7. Competition between antagonistic drives.

Five conclusions follow: (i) An intense shock can suppress consummatory behavior by competing with $\mathscr{A}_h \rightarrow \mathscr{S}$ arousal via the inhibitory $\mathscr{A}_f \rightarrow \mathscr{S}$ pathway. (ii) This suppression does not extinguish memory of the patterns already encoded in the $\mathscr{S} \rightarrow \mathscr{M}$ synaptic knobs. (iii) Suppression can take place faster than passive extinction. (iv) An intense shock can prevent new $\mathscr{S} \rightarrow \mathscr{M}$ associations from forming by inhibiting release of sampling signals from $\mathscr{S}$. (v) After $\mathscr{S} \rightarrow \mathscr{A}_f$ conditioning takes place, properties (i) through (iv) can be elicited on recall trials wherever the CS input activates $\mathscr{S} \rightarrow \mathscr{A}_f$ synapses.

Similar qualitative properties hold for Fig. 7b. Here, however, the $\mathscr{A}_f$ and $\mathscr{A}_h$ signals compete with each other at a second stage of processing

before a signal to $\mathscr{S}$ is emitted. It can be proved that only $\mathscr{A}_h$ can create an input (excitatory) to $\mathscr{S}$, and does so only if it emits a stronger signal than $\mathscr{A}_f$ does. The competitive mechanism is called a subtractive on-center off-surround field. Grossberg [35] discusses its mathematical properties. Figure 7b requires half as many $\mathscr{A} \rightarrow \mathscr{S}$ axons as Fig. 7a. This represents a considerable saving of axons, since each $\mathscr{A}$ subset projects nonspecifically to numerous $\mathscr{S}$ cells. On the other hand, Fig. 7a requires fewer cellular processing stations.

An important mathematical fact about competition between $\mathscr{A}_f$ and $\mathscr{A}_h$ will now be noted. For illustrative purposes, let a member of $\mathscr{S}$ send an axon only to $\mathscr{A}_h$. Whenever this $\mathscr{S}$ fires, the synaptic connection from $\mathscr{S}$ to $\mathscr{A}_h$ can be strengthened by transmitter potentiation even if $\mathscr{A}_h$ receives no UCS input. In other words, there exists a confusion between mere potentiation (use versus disuse) and learning. The situation is different when $\mathscr{S}$ projects to two or more arousal sources. Then firing of $\mathscr{S}$ without UCS presentation can potentiate the transmitter levels in $\mathscr{S} \rightarrow \mathscr{A}$ synaptic knobs without changing the pattern encoded there; no new learning occurs, except possibly some transient pattern crispening, or contour enhancement [34]. Learning occurs only if $\mathscr{S}$ firing precedes UCS presentation by a suitable interval. Potentiation and learning effects are thus factored into two distinguishable processes. Consequently, if the CS occurs regularly, $\mathscr{S}$ firing potentiates transmitter levels in $\mathscr{S} \rightarrow \mathscr{A}$ knobs and thereby achieves perfect memory of which arousal source controls behavior. This is true even if $\mathscr{A}_f$ dominates $\mathscr{A}_h$; no "overt" $\mathscr{S} \rightarrow \mathscr{M}$ firing is necessary. Perfect memory can also be achieved without potentiation if transmitter decay is multiplicatively coupled to spiking frequency, as in Eq. (3).

6. AVOIDANCE: HEURISTICS

The following postulate is essentially a rewording of Postulate X.

Postulate XI. $\mathscr{O}$ learns escape responses that do terminate shock faster than escape responses that do not terminate shock.

This postulate also builds upon mechanisms that are already at our disposal. In particular, while shock is on, $\mathscr{S} \rightarrow \mathscr{M}$ sampling is prevented by $\mathscr{A}_f \rightarrow \mathscr{S}$ inhibition. Shock termination removes $\mathscr{A}_f \rightarrow \mathscr{S}$ inhibition, but $\mathscr{S} \rightarrow \mathscr{M}$ sampling remains impossible until *some* excitatory arousal source is activated. Postulate XI can thus be reduced to the question: What excitatory arousal source releases $\mathscr{S} \rightarrow \mathscr{M}$ sampling just after shock is turned off, and thereby establishes conditioned pathways from the sensory cues that are available when the avoidance response occurs to both the active arousal source and the motor controls of the avoidance response?

Speaking heuristically, this arousal source provides the "motivational support" for learning the avoidance response. We suggest that an experimental analog of exciting this new arousal source is, other things equal, an internally perceived "relief" from fear [17, 52, 66].

Denote by $\mathscr{A}_f^-$ the arousal cells that are excited by termination of shock input to the cells $\mathscr{A}_f$, which we henceforth denote by $\mathscr{A}_f^+$. Some formal requirements must be imposed on $\mathscr{A}_f^-$ and $\mathscr{A}_f^+$ to ensure that the arousals work together effectively. First, require that excitation of $\mathscr{A}_f^-$ by shock termination is transient. Transient response is needed to prevent irrelevant sensory-motor coordinations from being learned whenever shock is off. The cells $\mathscr{A}_f^+$ are *on*-cells: they are turned on by shock, and remain on until shock is shut off. The cells $\mathscr{A}_f^-$ are *off*-cells: they are turned on temporarily by shock termination. On-cells and off-cells are familiar physiological components [72, pages 253, 349]. Second, require that the outputs from $\mathscr{A}_f^+$ to $\mathscr{A}_f^-$ reciprocally inhibit each other before they send signals to $\mathscr{S}$. Thus these outputs interact to form a consensus between "fear" and "relief." A possible behavioral analog of this rebound from $\mathscr{A}_f^+$ on-cells to $\mathscr{A}_f^-$ off-cells is the rebound in behavioral effects reported to occur after electrical hypothalamic stimulation terminates [16, 29, 73]. This analogy will receive further support from a chemical and anatomical analogy which will be developed in Part II between the twofold system $\mathscr{A}_f \equiv (\mathscr{A}_f^+, \mathscr{A}_f^-)$ and sites in the twofold system of ventromedial and lateral hypothalamus.

Our network must be expanded once again to allow $\mathscr{S}$ to become conditioned to the new arousal source. Thus, let each sensory representation $\mathscr{S}$ send axons to $\mathscr{A}_f^-$ as well as to $\mathscr{A}_f^+$, $\mathscr{A}_h$, and other $\mathscr{A}$ cell clusters. At any time, the synaptic knobs of each $\mathscr{S}$ encode a spatial pattern derived from the patterns $\theta(t) = [\theta_f^+(t), \theta_f^-(t), \theta_h(t), \ldots]$. This pattern describes the net balance of excitatory and inhibitory $\mathscr{A} \rightarrow \mathscr{S}$ feedback that this representation controls. It is determined by a weighted average of the spatial patterns $\theta(t)$ that reach $\mathscr{A}$ when the given $\mathscr{S}$ is sampling.

In summary, the classical notion that instrumental reinforcement is due to "drive reduction" when shock terminates is replaced by rebound from negative-incentive motivational on-cells to positive-incentive motivational off-cells when shock terminates. The balance of excitation of on-cells and off-cells can be classically conditioned, perhaps at different times, to all $\mathscr{S}$ representations. The net $\mathscr{A} \rightarrow \mathscr{S}$ output, and thus $\mathscr{S} \rightarrow \mathscr{M}$ firing and performance on recall trials, is determined by *all* of the $\mathscr{S}$ sites that fire to $\mathscr{A}$ at such times. Even if half of $\mathscr{S}$ fires to $\mathscr{A}_f^-$, no $\mathscr{S} \rightarrow \mathscr{M}$ channel will be activated by positive $\mathscr{A} \rightarrow \mathscr{S}$ feedback if the other half fires to $\mathscr{A}_f^+$, since $\mathscr{A}_f^-$ and $\mathscr{A}_f^+$ will reciprocally inhibit each other's outputs. Similarly, shock termination yields little "relief" if it is antagonized by a switching on of

new $\mathcal{S} \rightarrow \mathcal{A}_f^+$, or "fear," channels. Shock termination per se is not necessarily "drive reducing."

Various influences of situational cues, conditioned stimuli, and primary aversive stimuli will now be qualitatively interpreted in terms of rebound from $\mathcal{A}_f^+$ to $\mathcal{A}_f^-$ and reciprocal inhibition between $\mathcal{A}_f^+$ and $\mathcal{A}_f^-$ output. In what follows, AR (CAR) denotes the (conditioned) avoidance response, and $\mathcal{S}$(AR) ($\mathcal{S}$(CAR)) denotes interchangeably the sensory representations or the sensory feedback cues that are activated by the AR (CAR). Consider the simplified situation in Fig. 8 for definiteness. Each conditioned stimulus CS_i activates a sensory representation $\mathcal{S}_i$, $i = 1, 2, 3$, that learns a spatial pattern at its synaptic knobs facing $\mathcal{A}_f^+$ and $\mathcal{A}_f^-$. The relative synaptic weights of $\mathcal{S}_i$ are determined by the times at which $\mathcal{S}_i$ samples $\mathcal{A}_f$ and the times at which shock is on. For example, suppose that $\mathcal{S}_1$ samples $\mathcal{A}_f$ only when shock is on, that $\mathcal{S}_2$ samples $\mathcal{A}_f$ in an interval when shock is both

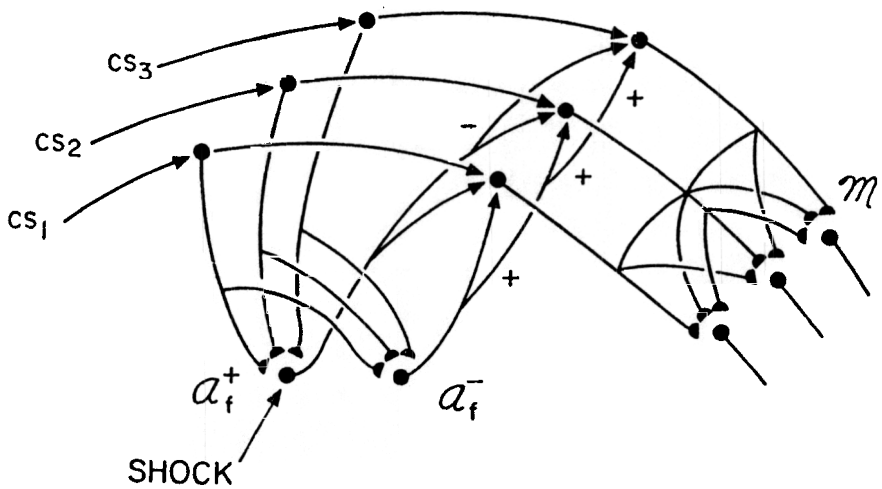

FIG. 8. Competition between fear and relief.

on and off, and that $\mathcal{S}_3$ samples $\mathcal{A}_f$ just after shock is turned off. Suppose on recall trials that one CS_i is presented at a time with a rest period between each presentation. Theorems on completely nonrecurrent networks can be applied [32, 39] to draw the following conclusions about recall trials. CS_1 will suppress consummatory responding by firing to $\mathcal{A}_f^+$, thereby generating a CER and preventing activation of $\mathcal{S} \rightarrow \mathcal{M}$ axons. CS_2 will be neutral in effect, since its signals to $\mathcal{A}_f^+$ and $\mathcal{A}_f^-$ are approximately equal and therefore cancel. CS_3 can (but need not) excite approach behavior yielding a CAR by firing to $\mathcal{A}_f^-$. CS_3 will not excite a CAR if, for example, shock is turned off by other than an AR, since then $\mathcal{S}_3 \rightarrow \mathcal{M}$ sampling on the learning trial will not encode from $\mathcal{M}$ motor controls of an AR; that is, $\mathcal{S}_3 \neq \mathcal{S}$(AR). Thus on recall trials, $\mathcal{S} \rightarrow \mathcal{M}$ firing will not reproduce motor controls of an AR. These remarks show that "relief" is possible without avoidance, since conditioning of $\mathcal{S} \rightarrow \mathcal{A}_f^-$ can occur

without simultaneous conditioning of specific motor controls of an AR in the $\mathcal{S} \rightarrow \mathcal{M}$ channel. Other factors can prevent CS_3 from activating a CAR. For example, CS_3 need not excite a CAR if on each trial AR determines different, and mutually independent, $\mathcal{S}$(AR)s. Then independent $\mathcal{S} \rightarrow \mathcal{A}$ and $\mathcal{S} \rightarrow \mathcal{M}$ channels are excited on successive learning trials. Cumulative practice in fixed $\mathcal{S}_3 \rightarrow \mathcal{A}$ and $\mathcal{S}_3 \rightarrow \mathcal{M}$ channels does not occur. Thus we are led to seek sensory filters that can identify sensory feedback cues that represent the same external event ("pattern recognition" problem).

Different effects occur if more than one CS_i is presented on recall trials. For example, let CS_1 and CS_3 be simultaneously presented on a recall trial. Then the CAR that is ordinarily released by CS_3 is suppressed, since $\mathcal{S}_1 \rightarrow \mathcal{A}_f^+$ and $\mathcal{S}_3 \rightarrow \mathcal{A}_f^-$ signals simultaneously occur, and the outputs from $\mathcal{A}_f^+$ and $\mathcal{A}_f^-$ to $\mathcal{S}$ inhibit each other.

Similar arguments yield effects that are qualitatively compatible with various data reviewed in Section 2. Response suppression without avoidance is possible [24] if only because conditioning of CS_1 to $\mathcal{A}_f^+$ can occur without conditioning of any CS_i to $\mathcal{A}_f^-$. Suppression can occur long before avoidance does [24] for several reasons: Conditioning of $\mathcal{S} \rightarrow \mathcal{A}_f^+$ pathways can occur reliably on every learning trial, since $\mathcal{A}_f^+$ is excited throughout the shocked interval and any active $\mathcal{S}$s can be conditioned to $\mathcal{A}_f^+$ during this interval. CAR conditioning requires parallel conditioning of both $\mathcal{S}$(AR)$\rightarrow \mathcal{A}_f^-$ and $\mathcal{S}$(AR)$\rightarrow \mathcal{M}$ channels. The $\mathcal{S}$(AR)$\rightarrow \mathcal{A}_f^-$ sampling can only occur during the brief interval after the AR occurs during which rebound from $\mathcal{A}_f^+$ to $\mathcal{A}_f^-$ and the $\mathcal{S}$(AR) sites are active.

More elaborate input events can also be discussed. Consider the experiment in which CS_1 occurs during shock on a sequence of learning trials, and CS_3 is turned on when CS_1 is shut off on a second sequence of learning trials. During the first sequence of trials, CS_1 learns to fire $\mathcal{A}_f^+$. On the second sequence, CS_1 offset causes a rebound at $\mathcal{A}_f^-$ to which CS_3 is conditioned. Thus a CS+ (= CS_1) paired with shock can excite fear, and a CS− (= CS_3) paired either with shock offset *or* offset of a secondary fear source can inhibit fear [51]. CS+ acts as a negative reinforcer in our network in the following formal sense. It suppresses $\mathcal{A}_f^- \rightarrow \mathcal{S}$ feedback and inhibits $\mathcal{S} \rightarrow \mathcal{M}$ sampling. CS− is a positive reinforcer in the following formal sense. It excites $\mathcal{A}_f^- \rightarrow \mathcal{S}$ feedback and elicits $\mathcal{S} \rightarrow \mathcal{M}$ sampling [21, 22, 42, 64, 65, 74, 75]. In a similar fashion, a feedback stimulus (FS) that occurs right after the AR can serve as a positive reinforcer in the following formal sense. It can be conditioned to $\mathcal{A}_f^-$ since the AR shuts off $\mathcal{A}_f^+$ and causes a rebound at $\mathcal{A}_f^-$. FS presentation thereafter activates $\mathcal{A}_f^-$ and can drive $\mathcal{S} \rightarrow \mathcal{M}$ sampling. The effects of a CS+ and an FS can be independent if the two stimuli activate separate $\mathcal{S}$ channels. Nonetheless, prolonging CS+ presentation after the AR can weaken conditioning of FS

to $\mathscr{A}_f^-$ by reducing the drop in total $\mathscr{A}_f^+$ input and thus the rebound at $\mathscr{A}_f^-$, as will be proved in Part II [10, 11, 48, 68]. More generally, the amount of *effective* reinforcement is determined by the increase in $\mathscr{A}_f^-$ input relative to the fixed sizes of previous $\mathscr{A}_f^+$ and $\mathscr{A}_f^-$ inputs, as will be proved in Part II [54, 55]. If in particular only the input to $\mathscr{A}_f^+$ changes, say by decreasing rapidly, then the size of $\mathscr{A}_f^-$ rebound is determined by the *relative* drop in $\mathscr{A}_f^+$ input; namely, by the decrease in input as compared to the initial input size. Some pain analgesic effects can also be interpreted in terms of a reduction in the emotional effects of one input due to the simultaneous occurrence of other inputs that reduce the relative size of input to $\mathscr{A}_f^+$ as compared to $\mathscr{A}_f^-$ [14, 27, 60].

Termination of proprioceptive cues from nonavoidance responses can be positively reinforcing in our networks, as punishment theory suggests [20, 56, 67]. Nonavoidance responses, denoted non ARs, occur while $\mathscr{A}_f^+$ is active, and their sensory feedback cues, denoted $\mathscr{S}$(non AR), can be conditioned to $\mathscr{A}_f^+$. This occurs even if $\mathscr{S}$(non AR)$\rightarrow\mathscr{M}$ conditioning is suppressed by $\mathscr{A}_f^+\rightarrow\mathscr{S}$ inhibition. Termination of $\mathscr{S}$(non AR) cues when the AR occurs can drive a rebound at $\mathscr{A}_f^-$ to which $\mathscr{S}$(AR) cues can be conditioned. The $\mathscr{S}$(AR) cues, supplemented by the $\mathscr{A}_f^-$ rebound, can also drive $\mathscr{S}$(AR)$\rightarrow\mathscr{M}$ conditioning of the AR motor controls at $\mathscr{M}$. Of course, if the $\mathscr{S}$(non AR) and $\mathscr{S}$(AR) cues overlap significantly, then prior $\mathscr{S}$(non AR)$\rightarrow\mathscr{A}_f^+$ conditioning can reduce the rebound at $\mathscr{A}_f^-$, since some $\mathscr{S}$(non AR) cues will be reinstated when the $\mathscr{S}$(AR) cues appear. A reduction in $\mathscr{S}$(AR)$\rightarrow\mathscr{A}_f^-$ conditioning will result.

Transfer of CS+ and CS− effects from classical to instrumental situations [4, 18, 65, 74, 75] has the following interpretation in our networks. *All* conditioning is classical in the sense that it involves cross-correlations of pre- and postsynaptic activity at prescribed synaptic knobs. The instrumental contingency determines when and at which knobs classical conditioning will occur. If a CS− is classically conditioned to $\mathscr{A}_f^-$ on a sequence of learning trials, it is automatically a positive reinforcer on a later sequence of trials because it enhances the same rebound from $\mathscr{A}_f^+$ to $\mathscr{A}_f^-$ that is driven by shock termination.

Forced extinction of a CAR without fear extinction [15] can occur by forcing the CAR to occur while $\mathscr{A}_f^+$ is active and thereby counterconditioning the $\mathscr{S}$(CAR) cues from $\mathscr{A}_f^-$ to $\mathscr{A}_f^+$. This mechanism allows some savings to occur on later avoidance trials, since the CAR can be suppressed by $\mathscr{A}_f^+\rightarrow\mathscr{S}$ feedback without counterconditioning $\mathscr{S}$(CAR)$\rightarrow\mathscr{M}$ channels.

Contingent versus noncontingent shock can affect fear and suppression of a given response R in opposite ways [63, 77]. In the contingent case, the $\mathscr{S}$(R)$\rightarrow\mathscr{M}$ channels are suppressed by conditioning the $\mathscr{S}$(R)$\rightarrow\mathscr{A}_f^+$ channels. In both contingent and noncontingent cases, the net fear will be

determined by *all* the $\mathscr{S}$s that fire to $\mathscr{A}_f^+$ at any time. If the frequency and intensity of noncontingent shock is increased on learning trials, the relative input on recall trials to $\mathscr{A}_f^+$ rather than $\mathscr{A}_f^-$ from *all* $\mathscr{S}$s will increase, even though each $\mathscr{S}$—including the $\mathscr{S}$(R)s—might have a small suppressive effect; that is, small relative preference for $\mathscr{A}_f^+$. In the contingent case, the $\mathscr{S}$(R)s control most of the suppressive effect; that is, these cues control a large relative preference for $\mathscr{A}_f^+$. Thus one can increase fear without suppressing R in the noncontingent case by "spreading the fear around" $\mathscr{S}$.

Rapid switching from $\mathscr{A}_f^+$ activation to $\mathscr{A}_f^-$ activation can be effected by a rapid scanning from cues which fire $\mathscr{A}_f^+$ to cues which fire $\mathscr{A}_f^-$. Nonchalant avoidance on asymptotic avoidance trials is a possible consequence [70], as are an asymptotically reduced CER to the CS+ [47], absence of autonomic arousal to the CS+ [6], and the existence of an avoidance latency too short to allow autonomic arousal [71]. To establish the CAR, fear elicitation on learning trials is needed only to drive the rebound at $\mathscr{A}_f^-$. Once $\mathscr{S}$(AR) cues are conditioned to $\mathscr{A}_f^-$, they no longer require $\mathscr{A}_f^+$ as a motivational source. A scanning mechanism that focuses on $\mathscr{A}_f^-$-conditioned cues, rather than $\mathscr{A}_f^+$-conditioned cues, can therefore minimize the role of the CER during asymptotic conditioned avoidance trials.

Fear is not useless on asymptotic avoidance trials, however, at least in a formal sense. A CAR can extinguish if its $\mathscr{S}$(CAR)$\rightarrow\mathscr{A}_f^-$ conditioning is not bolstered from time to time by rebound from $\mathscr{A}_f^+$ to $\mathscr{A}_f^-$. Extinction can, for example, be driven by irrelevant $\mathscr{S}$ cues which fire equally to $\mathscr{A}_f^+$ and $\mathscr{A}_f^-$ while $\mathscr{S}$(CAR) cues are active. The ratio of input to $\mathscr{A}_f^-$ and input to $\mathscr{A}_f^+$ is equalized by the input from irrelevant cues. The relative strength of $\mathscr{S}$(CAR)$\rightarrow\mathscr{A}_f^-$ channels is thus gradually weakened by countercconditioning to $\mathscr{S}$(CAR)$\rightarrow\mathscr{A}_f^+$ channels. Such extinction can be prevented if $\mathscr{O}$ can focus on only $\mathscr{S}$(CAR) cues during avoidance trials. The topography of the experimental chamber, among other factors, will influence $\mathscr{O}$'s success in doing this.

Noncontingent punishment of the CAR during extinction trials can delay the extinction process [5, 28]. Such punishment can strengthen $\mathscr{S}$(CAR)$\rightarrow\mathscr{A}_f^-$ conditioning by first strengthening $\mathscr{S}$(non CAR)$\rightarrow\mathscr{A}_f^+$ conditioning. Termination of $\mathscr{S}$(non CAR) cues when the CAR occurs then drives $\mathscr{A}_f^-$ rebound, which is sampled by $\mathscr{S}$(CAR). This mechanism also "spreads the fear around" $\mathscr{S}$. In this example, however, one studies a response whose cues are present after shock rather than, as with suppression due to contingent shock, a response whose cues are present during shock. Similar effects of "spreading the fear around" $\mathscr{S}$ lead to formal analogs of differences between one-way [4] and two-way [76] avoidance training. The importance of knowing which cues are conditioned to $\mathscr{A}_f^+$

or $\mathscr{A}_f^-$ is emphasized by studies [44] in which the main experimental variable is frequency of shock, which can be reduced by prescribed instrumental behavior. Changes in this frequency influence the pattern $\theta(t) = [\theta_f^+(t), \theta_f^-(t), \theta_h(t), \ldots]$ that each $\mathscr{S}$ samples. It will be clear from Herrnstein's article that the rebound mechanism is related to, but not identical with, classical two-factor theories.

Counterconditioning by irrelevant cues is also a possible formal mechanism for extinguishing the CER and thus for spontaneous recovery of a suppressed consummatory response R. Suppose that the cues $\mathscr{S}(R)$ are partitioned into two subsets $\mathscr{S}_1(R)$ and $\mathscr{S}_2(R)$. Let $\mathscr{S}_1(R)$ be conditioned to $\mathscr{A}_f^+$ and $\mathscr{S}_2(R)$ be conditioned to $\mathscr{A}_f^-$. Let $\mathscr{S}_1(R) \rightarrow \mathscr{A}_f^+$ channels suppress responding with R when $\mathscr{S}(R)$ presentation and hunger coincide. Let $\mathscr{S}(R)$ be presented on extinction trials along with irrelevant cues that send equal signals to $\mathscr{A}_f^+$ and $\mathscr{A}_f^-$. Then the $\mathscr{S}_1(R) \rightarrow \mathscr{A}_f^+$ channels will become gradually weaker. If $\mathscr{S}_2(R) \rightarrow \mathscr{A}_f^-$ conditioning is more resistant to the effects of irrelevant cues, then R responding will spontaneously recover. Even if $\mathscr{S}_2(R) \rightarrow \mathscr{A}_f^-$ channels weaken, $\mathscr{S}(R) \rightarrow \mathscr{M}$ conditioning will remain, thus permitting rapid reacquisition of the response R. Can a greater resistance to extinction of $\mathscr{S}_2(R) \rightarrow \mathscr{A}_f^-$ than of $\mathscr{S}_1(R) \rightarrow \mathscr{A}_f^+$ channels be expected? Yes, if $\mathscr{S}_2(R)$ is elicited selectively by the response manipulandum, whereas $\mathscr{S}_1(R)$ is elicited by unspecific situational cues; this is especially true if $\mathscr{S}_2(R)$ releases an AR that removes $\mathscr{O}$ from $\mathscr{S}_2(R)$ input sources. No, if forced extinction of R in the presence of $\mathscr{S}_2(R)$ cues is coupled with fear conditioning.

Once it is explicitly constructed, the rebound mechanism will reveal another source of "irrelevant" cues; namely, a *tonic* arousal sources that simultaneously drives both $\mathscr{A}_f^+$ and $\mathscr{A}_f^-$ in order to supply energy for the rebound. This tonic source will also influence fear and avoidance thresholds. The explicit mechanism will, in fact, clarify and extend all of the foregoing conclusions. Various other data also require further structure in our network; for example, the Blanchard and Blanchard [8, 9] and Brener and Goesling [12] data. We here need to know how asymmetries in the spatial distribution of fearful cues and of painful stimuli at $\mathscr{O}$'s receptors drive unconditioned responding that is controlled by other channels than the suppressed $\mathscr{S} \rightarrow \mathscr{M}$ channels. These motor events can then be sampled at $\mathscr{M}$ by appropriate $\mathscr{S}$ channels if the net $\mathscr{A} \rightarrow \mathscr{S}$ feedback becomes sufficiently positive, say due to the termination of shock by an AR that excites $\mathscr{S}(AR)$ sampling cells and drives the rebound from $\mathscr{A}_f^+$ to $\mathscr{A}_f^-$.

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