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CHAPTER 21

Neural models of normal and abnormal behavior: what do schizophrenia, parkinsonism, attention deficit disorder, and depression have in common?

Stephen Grossberg

Department of Cognitive and Neural Systems and Center for Adaptive Systems, Boston University, 677 Beacon Street, Boston, MA 02215, USA

Introduction: mental disorders as emergent properties

In order to mechanistically explain how the brain gives rise to mental disorders, several problems need to be solved. First, one needs models of how the brain gives rise to the normal behaviors that are damaged or eliminated during the mental disorder. Then one needs to provide a clear description of which normal mechanisms are changed during the mental disorder. Many mental disorders may be traced to chemical or electrical imbalances of one sort or another. Knowing which chemical or electrical imbalance is involved is an important necessary condition for understanding the disorder, but it is far from sufficient. In addition, both for theoretical understanding and for informed clinical intervention, one needs to explain how each local lesion gives rise to the behavioral symptoms that characterize the disorder. Typically, such symptoms are emergent properties due to the interactions of many cells across the brain. Often these interactions involve non-linear cell properties and feedback between cells operating on multiple spatial and temporal scales. Thus, when one says that an imbalance of dopamine metabolism 'causes' schizophrenia or other mental disorders, such a statement may provide little insight into how this imbalance gives rise to the symptoms of schizophrenia, despite its great value in guiding the search for clinically effective drugs. A theoretical method is therefore needed that is strong enough to explain how local lesions give rise to behaviorally relevant emergent properties across such a complex system.

Even in cases where a malfunction may be localized within a particular neural subsystem, this subsystem's interactions with several other subsystems may be disrupted. The other subsystems may then also contribute to abnormal symptoms. For example, if two subsystems are mutually inhibitory and one subsystem becomes abnormally hyporeactive, then the other subsystem may become abnormally hyperreactive, even though its own cells would otherwise perform normally. In such a situation, it may be difficult to decide, just by looking at small subsets of biochemical or neurophysiological data, whether the syndrome is due to hyporeactivity or to hyperreactivity. Due to the complementary reactions of the two subsystems, some measures of total system performance may show no effect or conflicting effects across subjects in whom the degree of imbalance varies.

The present chapter discusses how some key symptoms of schizophrenia, Parkinson's disease, attention deficit disorder, and depression can be caused as emergent properties of neural models of

behavior when their cellular parameters are perturbed out of the 'normal' range. These examples do not purport to be complete explanations of the symptoms in question, but they do show how such a model can help to close the gap between brain and behavior. In particular, several behavioral properties of these models simultaneously covary in a manner that reflects clinical data when one of its cellular parameters is varied. Other behavioral properties serve as model predictions to further test the proposed explanation. Finally, the normal functioning of the model clarifies the transition between normal and abnormal behavioral states. Each of these model circuits will be presented in its simplest possible form. For example, some properties are more easily demonstrated using excitatory transmitter substances, whereas they may be realized in vivo using a cascade of inhibitory transmitters that exert a disinhibitory action.

The golden mean: opponent processing, inverted-*U*, and rebound

The models in question generate abnormal behavioral properties when they experience an abnormally low or high arousal level. Such a change may be due to a number of different factors. One key type of model involves an opponent processing circuit that has been called a gated dipole (Grossberg, 1972a,b, 1980). Opponent processing can influence motivated behavior. Here the opponent channels may control such opposed motivational factors as fear and relief (Estes and Skinner, 1941; Denny, 1971). Opponent processing also influences perceptual processing, where it may control opponent colors such as red and green, or perpendicular orientations such as vertical and horizontal, or opposite directions such as up or down (Helmholtz, 1866, 1962; Brown, 1965; Sekuler, 1975). Motor behaviors may also have an opponent organization, as illustrated by GO and STOP signals for gating the onset or offset of motor actions (Horak and Anderson, 1984a,b), and the opponent organization and control of flexor and extensor muscle groups.

Why is opponent processing so ubiquitous in the nervous system? It has been proposed (e.g. Grossberg, 1980, 1982c) that opponent processing helps the brain to self-organize its neural circuits in a self-stabilizing way, both during childhood development and adult learning; that is, in a way that develops and learns neural circuits that match the statistics of the environment, and that dynamically buffers these circuits against catastrophic reorganization by irrelevant environmental fluctuations.

Two key properties of these opponent processing circuits are their inverted-*U* and reset properties. The inverted-*U* property (Fig. 1) enables a gated dipole circuit to maintain a type of Golden Mean in response to the circuit's arousal level. The concept of arousal level, as here used, needs to be carefully defined, because several different mechanisms can all change the arousal level, from a functional point of view, without seeming to be arousal-specific mechanisms. This Golden Mean says that circuit sensitivity to input fluctuations is optimal at moderate arousal levels, but degrades in different ways when the circuit is either underaroused or overaroused. These properties are mathematically

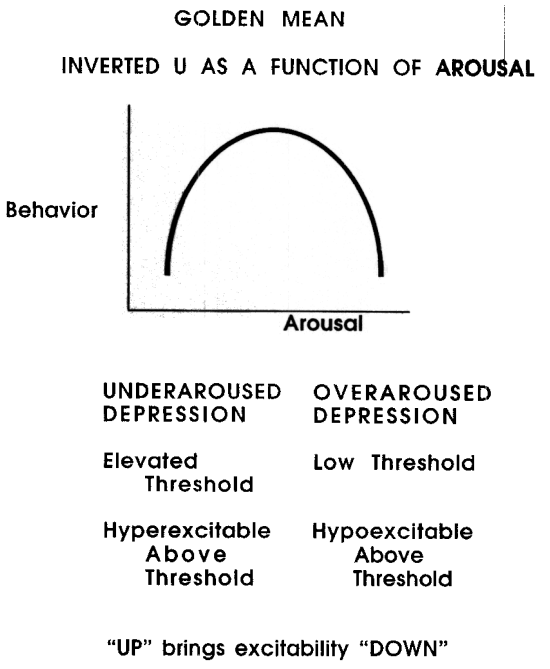

Fig. 1. Gated dipole opponent processes exhibit an Inverted-*U* as a function of arousal level, with underaroused and overaroused depressive syndromes at the two ends of the Inverted-*U*.

proved in the Appendix. The main text will describe them heuristically.

Such inverted-*U* properties are well-known to occur in behavior. For example, D-amphetamine sulfate activates feeding in an anorectic cat at the same dose that totally inhibits feeding in a normal cat (Wolgin et al., 1976). In normal cats, smaller amounts of norepinephrine can have effects opposite to those of larger amounts (Leibowitz, 1974). In like manner, amphetamine augments slow behavior and depresses fast behavior (Dews, 1958). In humans, dopamine pharmacological manipulations have shown that the relation of dopamine activity to reaction-time performance is an inverted-*U* function (Zuckerman, 1984; Netter and Rammsayer, 1991; Rammsayer, Netter, and Vogel, 1993). Subjects high on extraversion and sensation seeking scales show impaired task performance if a dopamine agonist is applied, and improved performance if an antagonist is applied. The opposite pattern was found in subjects low in these traits. inverted-*U*s have also been reported in event-related potentials, such as the contingent negative variation, or CNV (Tecce and Cole, 1974). In the analysis below of mental disorders, the assumption will be made, and supportive data cited, that various circuits are far off their optimal level of arousal, and are seriously underaroused or overaroused.

The reset property involves a process of antagonistic rebound that can be triggered in at least two ways. Both ways enable the circuit to shut off currently active cells, disconfirm their processing by transiently activating their opponent channels, and thereby restore the balance between opponent channels in order to process subsequent inputs with as little bias as possible. The two ways of causing an antagonistic rebound are a sudden decrease of a previously sustained input to one channel of the opponent circuit (see Fig. 2), and a sudden increment of arousal to both channels.

A sudden decrease of input to one channel (say, the 'fear' channel, or the 'red' channel) can lead to a transient activation, or antagonistic rebound of activity, in the opponent channel (say, the 'relief' channel, or the 'green' channel). Antagonistic rebound has many functional uses. For example, when there is a sudden decrease of fearful cues in a given situation, then both classical conditioning and instrumental conditioning mechanisms can use the relief rebound as a source of positive motivation with which to learn the sensory-motor contingencies that led to the reduction of fear (cf. Masterson, 1970; Reynierse and Rizley, 1970; Denny, 1971; McAllister and McAllister, 1971a,b; Grossberg, 1982b, 1984b, 1987b). Likewise, if hypothalamic stimulation elicits a given behavior, then its offset can transiently elicit an opposite behavior (Cox et al., 1969; Valenstein et al., 1969).

A sudden increment in arousal may be due to an unexpected event (Renault and Lesèvre, 1978; Näätänen et al., 1982; Näätänen and Gaillard, 1983; Banquet and Grossberg, 1987; Grossberg, 1984a, 1987). Such a rebound can disconfirm and reset ongoing sensory, cognitive, motivational, and motor processing in order to enable the brain to

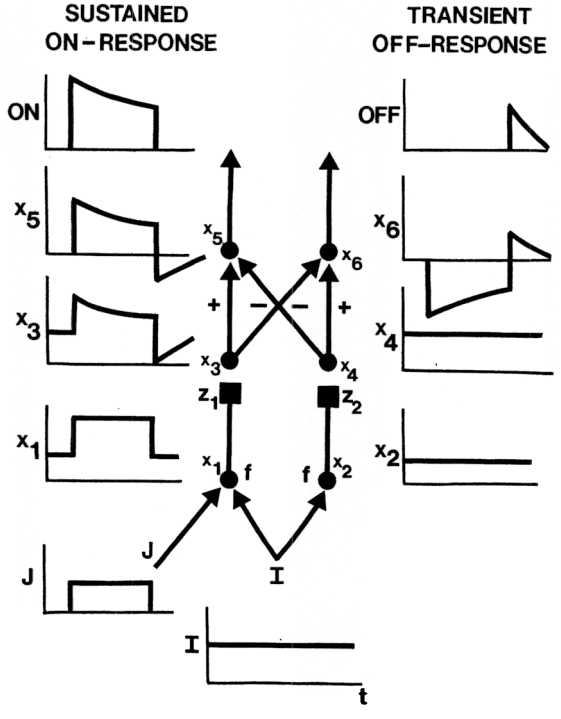

Fig. 2. The simplest feedforward gated dipole circuit. In response to a phasic input *J* and tonic arousal input *I*, the dipole generates an ON-response and a transient antagonistic OFF-rebound due to the action of the habituative transmitter gates within the square synapses. See text and Appendix for details.

better process the unexpected information and deal with it adaptively.

Underaroused and overaroused depressive syndromes

The mechanisms that enable a gated dipole to achieve these useful emergent properties at normal arousal levels also generate clinically relevant properties when the arousal level is chosen too low or too high. As noted in Fig. 1, an underaroused gated dipole generates a syndrome of Underaroused Depression. Here, due to an abnormally small arousal level, inputs must be larger than normal in order to overcome the dipole's increased response threshold. Paradoxically, once inputs are chosen large enough to overcome this threshold, then the circuit is hyperexcitable above threshold, meaning that the dipole generates abnormally large outputs in response to additional input increments. This is paradoxical because a naive view might conclude that an elevated threshold would make the circuit less, rather than more, excitable. Because such a circuit is hyperexcitable at low arousal levels, its excitability can be brought into the normal range by increasing its arousal until it reaches the peak of the Inverted-*U*. Here, the threshold is lower, but the network's excitability is also lower. These properties clarify the paradoxical fact that an arousing drug can make some patients less excitable. This fact is more completely discussed below in terms of how amphetamines help attention deficit disorder, or juvenile hyperactivity, patients (Swanson and Kinsbourne, 1976; Weiss and Hechtmann, 1979) and L-dopa helps Parkinson's patients (Riklan, 1973).

It is not, however, the case that unlimited increases in arousal will make a dipole behave more normally. Too much arousal generates a syndrome of Overaroused Depression. Here the extra arousal causes the response threshold to be very low. Paradoxically, however, the circuit is hypoexcitable above this low threshold, so that it generates small responses, at best, to inputs of arbitrary size. Thus 'too much of a good thing', such as amphetamine or L-dopa for the patients mentioned above, can create a new, and complementary, problem to the one for which they are being treated. For example, large doses of amphetamine and L-dopa can cause a psychosis reminiscent of schizophrenia (Riklan, 1973; Wallach, 1974), although, L-dopa has also been reported to improve negative schizophrenic symptoms (Gerlach and Luhdorf, 1975; Albert and Rush, 1983). These arousal problems are discussed below in terms of negative symptoms such as flat affect in schizophrenia, and how it may lead to other schizophrenic symptoms through its interactions with other brain processes. In the opposite direction, antipsychotic drugs that block dopamine receptors (Kuhar et al., 1978) can, in sufficient quantities, produce a catalepsy suggestive of Parkinson's disease (Hornykiewicz, 1975).

The whole is greater than the sum of its parts: arousal, transmitters, signals, competition, and thresholds are the parts

The paradoxical emergent properties of a gated dipole are due to five basic mechanisms acting together (Grossberg, 1972b, 1980, 1984a,b). Figure 2 depicts the simplest example of a circuit that realizes these mechanisms. The two dipole channels are called the ON and OFF channels in the subsequent exposition. The ON channel is turned on by a phasic input, denoted by J in Fig. 2; the OFF channel registers the antagonistic rebound that occurs when the phasic input to the ON channel shuts off. The five mechanisms are: (1) a source of nonspecific arousal, denoted by I in Fig. 2, energizes both channels of the dipole; (2) a nonlinear signal function, denoted by f in Figure 2, transduces the sum of phasic and arousal inputs to each channel; (3) a habituative transmitter substance multiplies, or gates, the nonlinear signals from both channels; (4) the gated signals compete via an on-center off-surround network; and (5) the net signal after competition is half-wave rectified, or thresholded, before generating an output from the network.

The key mechanism that governs dipole dynamics is the habituative transmitter gate. This mechanism varies on a slower time scale than the rate with which input signals fluctuate, and thereby provides the 'memory' that calibrates the size of the antagonistic rebound. This transmitter process

was originally derived as the minimal mechanism whereby data about associative learning could be explained using a chemical transmitter capable of generating an unbiased signal from one cell to another (Grossberg, 1968, 1969a). Transmitter habituation occurs when the recovery rate of the transmitter falls behind the input fluctuation rate. Similar model mechanisms have been used to explain how the relative timing of paired presynaptic and postsynaptic signals can influence the strength of the adaptive weights, or associations, that link presynaptic to postsynaptic cells (Markram et al., 1997). Other early work (e.g. Grossberg, 1972a,b, 1980, 1982b,c, 1984a,b; Grossberg and Gutowski, 1987) has explained many data about normal cognitive and emotional processing using habituative chemical transmitters operating in a gated dipole within a larger network architecture. It is because so many data about normal cognitive-emotional behaviors have been clarified by these circuits that their ability to map onto clinical properties in their underaroused and overaroused regimes takes on such potential significance.

Figure 3 illustrates how, in response to a changing input signal $S(t)$, a habituative transmitter $z(t)$ can gradually equilibrate to the signal's more rapidly changing amplitudes. In particular, higher input amplitudes lead to lower levels of transmitter. The transmitter $z(t)$ multiplies, or gates, the input $S(t)$ to generate a gated output signal $T(t) = S(t)z(t)$. Due to this gating process, monotonic changes in input amplitude $S(t)$ cause overshoots and undershoots in the gated output $T(t)$, before the transmitter gradually equilibrates, or habituates, to the new input level. In this simplified model, the transmitter accumulates to a fixed equilibrium concentration at a constant rate, and is inactivated, or habituates, at a rate proportional to $T(t)$.

Figure 2 shows what happens when such a habituative transmitter is embedded within a gated dipole, notably in the dipole's square synapses. Here the transmitter in the ON channel gates the sum of the phasic input J and the tonic arousal input I after they are transformed by the signal f (variable x_1 in Fig. 2). As a result, just as in Fig. 3, increases and decreases the total input $I+J$ are transformed by the habituative transmitter into overshoots and undershoots of the gated signal (variable x_3 in Fig. 2). When these overshoots and undershoots are processed by the on-center off-surround network and the output threshold, a habituative response in the ON channel (variable ON in Fig. 2) and a transient antagonistic rebound in the OFF channel (variable OFF in Fig. 2) are produced. The antagonistic rebound is energized by the arousal input I, which can activate the OFF channel even after the phasic input J to the ON channel shuts off. The effects of the phasic input J depend upon interactions between all the five mechanisms that define the dipole. These interactions determine the arousal levels I at which the dipole is in the mid-point of its Inverted-U or at an underaroused or overaroused extreme.

Perhaps the most recent experimental evidence for such habituative transmitters in the brain has been reported by Abbott et al. (1997), who have used the same law to explain their data about

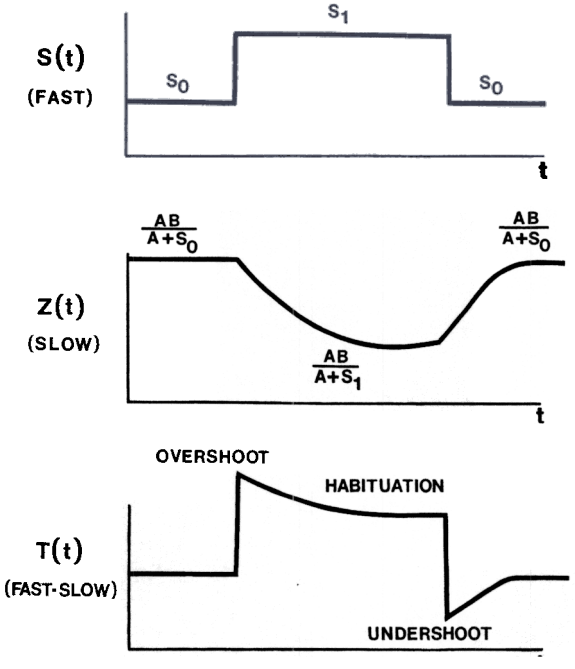

Fig. 3. In response to rapid increases and decreases in input amplitude $S(t)$, the habituative transmitter $z(t)$ decreases and increases in the reverse direction. The gated output signal $T(t) = S(t)z(t)$ is the product of these fast and slow reactions, and generates overshoots and undershoots to the changes in $S(t)$, followed by habituation to an intermediate level.

'depressing synapses' in the visual cortex. Their data exhibit many of the habituative transmitter properties that had previously been used to model other vision data (e.g. Grossberg, 1980, 1987a; Carpenter and Grossberg, 1981; Ögmen and Gagné, 1990; Ögmen, 1993; Francis et al., 1994; Francis and Grossberg, 1996a,b).

Recurrent opponent processes can actively modulate associative learning

Opponent processes often include feedback pathways within and between their ON and OFF channels, as illustrated in Fig. 4. These feedback pathways realize a type of short-term memory whereby the opponent process maintains a steady operating level against sufficiently small environmental perturbations, and switches between different pathways when a sufficiently large environmental change occurs. For example, a motivational dipole can hereby maintain a steady level of motivation during the performance of a consummatory act, and cannot be reset by insignificant environmental distractions.

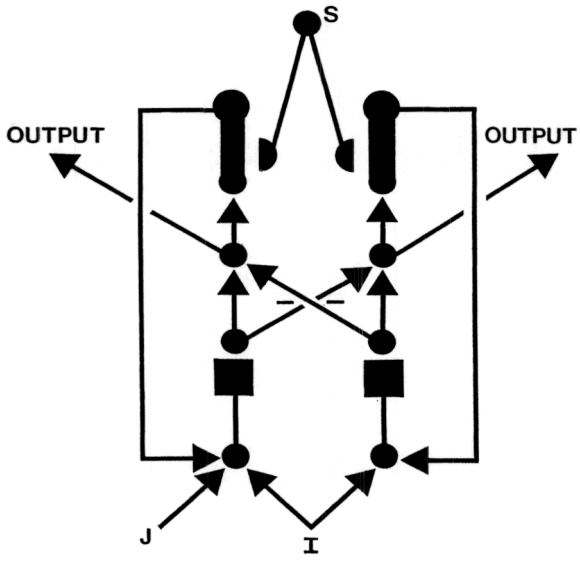

Fig. 4. A READ circuit: This circuit joins together a recurrent gated dipole with an associative learning mechanism. Learning is driven by signals from sensory representations S which activate associative weights that learn activation levels within the ON-channel and OFF-channel of the dipole.

Feedback pathways can also regulate how learning occurs by ensuring that associative synapses which input to the opponent circuit respond only to the circuit's net activity after opponent competition takes place. In this way, the circuit can dissociate the read-out of previously learned associations from the read-in of new associations. Read-in senses only the competitive 'decision' that is made by the opponent interactions. This is called the associative dissociation property.

Grossberg and Schmajuk (1987) have modeled how recurrent opponent processing circuits may operate during reinforcement learning. Here the opponent channels represent opponent drive states, such as fear and relief. The name 'READ circuit' was used to describe this circuit because it is a REcurrent Associative Dipole that combines opponent processing with associative learning (Fig. 4). The READ circuit enables the associative dissociation property to be realized in a simple way, as noted below. It hereby realizes several useful properties: learning can go on indefinitely without saturating the associative memories; these memories can persist until they are disconfirmed by unexpected events; and network learning is buffered against noise.

Dissociation is realized by placing the associative synapses on dendritic spines. Read-out of old associative values occurs at the dendritic spines and then propagates towards the cell body where they may or may not succeed in generating cell firing. Those cells which do fire and whose firing survives opponent competition may fire long enough to trigger retrograde dendritic spikes that invade the dendritic spines. Here they can drive read-in of new associative values at those synapses which are receiving concurrent presynaptic signals. This use of retrograde dendritic spikes from the cell body to dendritic spines as a way to dissociate associative read-out from read-in has been a recurrent theme during the development of the model and its extensions (e.g., Grossberg, 1975, 1987b, p. 38; Grossberg and Schmajuk, 1987). Remarkable experimental progress has recently been made towards characterizing how retrograde dendritic spikes can influence associative learning at dendritic spine sites (Johnston et al., 1996; Magee and Johnston, 1997; Markram et al., 1997; Koester and

Sakmann, 1998). It still remains to test whether or not these spikes are used to achieve associative dissociation. One type of direct test would monitor such retrograde spikes and their associative consequences during two conditions. In both conditions, the inputs to the recorded dendritic apparatus would be the same. In one condition, its cell body would 'win' the recurrent competition with a nearby stimulated cell; in the other, it would lose the competition. Learning should be greater in the former case.

Control of attention and action by cognitive-emotional interactions

READ circuits and their variants may be embedded within model representations of cognitive, emotional, and motor processes. Interactions among these representations lead to emergent properties that resemble symptoms of mental disorders when the dipoles are underaroused or overaroused. Some of these interactions are schematized in Fig. 5 in their simplest form. Circuits of this type will be called CogEM circuits henceforth in order to abbreviate their Cognitive, Emotional, and Motor interactions. CogEM models have undergone progressive development to explain ever more behavioral and neural data about normal cognitive-emotional interactions, including reinforcement learning and attention (e.g., Grossberg, 1971, 1972a,b, 1982a,b; Grossberg and Gutowski, 1987; Grossberg and Levine, 1987; Grossberg and Schmajuk, 1987; Buonomano et al., 1990; Grossberg and Merrill, 1992, 1996). The CogEM architecture may be derived from simple and broadly accepted hypotheses about how associative learning occurs (see Grossberg 1982b).

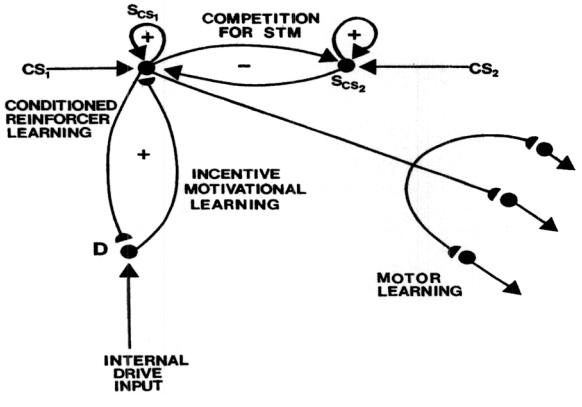

Fig. 5. The simplest CogEM circuit. At least three types of representations–sensory S, drive D, and motor M–interact to control cognitive-emotional interactions. At least three types of learning–conditioned reinforcer, incentive motivational, and motor learning–connect these representations. Conditioned stimuli (CS) activate the S representations, which compete among themselves for limited-capacity short-term memory activation and storage. The activated S representations elicit conditionable signals to drive representations and motor representations. See text for details.

Figure 5 summarizes the hypothesis that (at least) three types of internal representation interact during reinforcement learning: sensory and cognitive representations S, drive representations D, and motor representations M. The S representations are thalamocortical representations of external events, including the object recognition categories that are learned by inferotemporal and prefrontal cortical interactions (Ungerleider and Mishkin, 1982; Mishkin et al., 1983; Desimone, 1991; Gochin et al., 1991; Harries and Perrett, 1991). The D representations include hypothalamic and amygdala circuits at which homeostatic and reinforcing cues converge to generate emotional reactions and motivational decisions (Halgren et al, 1978; Bower, 1981; Gloor et al., 1982; Aggleston, 1993; LeDoux, 1993; Davis, 1994). The M representations include cortical and cerebellar circuits that control discrete adaptive responses (Evarts, 1973; Ito, 1984; Thompson, 1988; Kalaska et al., 1989). More complete models of the internal structure of these several types of representations are developed elsewhere (e.g., Grossberg, 1987b; Grossberg and Schmajuk, 1987; Carpenter and Grossberg, 1994; Fiala et al., 1996; Grossberg and Merrill, 1996; Contreras-Vidal et al., 1997; Bullock et al., 1998). Even the model in its simplest form has successfully learned to control motivated behaviors in mobile robots (e.g., Baloch and Waxman, 1991; Gaudiano et al., 1996; Gaudiano and Chang, 1997; Chang and Gaudiano, 1998).

Three types of learning take place among these representations: The $S \rightarrow D$ conditioned reinforcer learning converts a conditioned stimulus (CS) into a reinforcer by pairing activation of its sensory representation S with activation of the drive representation D, where representation D is acti-

vated by an unconditioned stimulus (US) or other previously conditioned reinforcer CSs. The $D \rightarrow S$ incentive motivational learning enables an activated drive representation D to prime, or modulate, the sensory representations S of all cues, including the CSs, that have consistently been correlated with it. Activating D hereby generates a 'motivational set' by priming all of the sensory and cognitive representations that have been associated with that drive in the past. These incentive motivational signals are a type of motivationally-biased attention. The $S \rightarrow M$ motor, or habit, learning enables the sensorimotor maps, vectors, and gains that are involved in motor control to be adaptively calibrated.

These processes control the learning, recognition, and recall of sensory and cognitive memories ('declarative memory'; Mishkin, 1982, 1983; Squire and Cohen, 1984) and the performance of learned motor skills ('procedural memory'; Gilbert and Thatch, 1977; Ito, 1984; Thompson, 1988). In particular, learned $S \rightarrow D \rightarrow S$ positive feedback quickly draws attention to motivationally salient cues by amplifying the activation of their sensory representations. The sensory representations use recurrent interactions to store these activities in short-term, or working, memory (Baddeley, 1986). This is accomplished by linking the sensory representations by a recurrent on-center off-surround network, whereby cells excite themselves and possibly their immediate neighbors, and inhibit a wider range of cells, possibly including themselves (Fig. 5). Such a network enables the sensory representations to store activities that retain their sensitivity to the relative sizes of their inputs, while also tending to conserve, or normalize, the total activity among the representations (Grossberg, 1973, 1978a,b; Bradski et al., 1994). This activity normalization property realizes the limited capacity of short-term memory, since when one sensory representation gets very active, the representations with which it competes are forced to become less active.

Two types of distractability due to overaroused drive representations

Taken together, these learning and short-term memory mechanisms help to explain data (Pavlov, 1927; Kamin, 1968, 1969) about how attention can be focused on motivationally salient cues, and 'blocked' from being allocated to less salient or irrelevant cues: When the sensory representations S that categorize conditioned reinforcers are amplified by their strong $S \rightarrow D \rightarrow S$ attentional feedback pathways, they can block activation of other S populations via $S \rightarrow S$ lateral inhibition. (Grossberg and Levine (1987) have presented model simulations of attentional blocking.) In a more elaborate version of the model, the drive representations are built up from motivational READ circuit dipoles that code such opponent drive states as fear and relief (Grossberg and Schmajuk, 1987).

If these emotional dipoles are overaroused, for any number of reasons, then they cannot be effectively activated by their sensory and cognitive inputs. Their activities remain small no matter how large these inputs become. The result is flat effect, because the dipoles cannot generate a large emotional response. As a consequence of this reduced response, the dipoles cannot generate adequate incentive motivational feedback signals with which to activate motivationally compatible sensory representations (see Fig. 5). These sensory representations thus cannot successfully compete for attention based upon their motivational salience; nor can they control the release of motivationally appropriate responses. In the absence of motivationally directed attention, motivationally irrelevant cues can attract attention and generate inappropriate responses. In summary, overaroused drive representations can lead to flat effect and distractability.

These problems may elicit additional cognitive problems as a result of the way in which the motivational circuits interact with other circuits in the brain. For example, attentional and consummatory circuits compete with orienting circuits for the control of behavior (Grossberg, 1980; Staddon, 1983), as schematized in Fig. 6. This property realizes a competition between the circuits that process expected events and those that process unexpected events. The latter circuits help to incorporate the unexpected events, notably unfamiliar events, into the corpus of expected and familiar events through learning. The competition between attentional and orienting systems enables

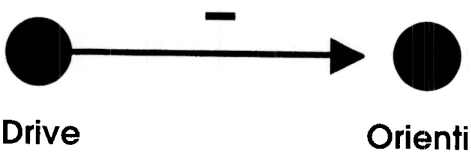

Fig. 6. Competition occurs between consummatory and orienting circuits. Here it is realized between drive representations and orienting representations.

such learning to take place without forcing unselective forgetting of previously learned knowledge (Grossberg, 1980; Carpenter and Grossberg, 1991; Grossberg and Merrill, 1996).

A simple example illustrates what this competition means: Suppose that you hear a sudden and unexpected loud noise to your right. The noise elicits a rapid orienting movement to look at the loud noise for further processing. On the other hand, suppose that you are trained to use the loud noise as a discriminative cue for pushing a button to receive a large monetary award. Then the orienting responses can be supplanted by consummatory responses such as button-pressing. When motivational dipoles in the drive representations D are overaroused, then they cannot adequately inhibit the orienting circuits with which they interact. As a result, overarousal can disinhibit orienting responses in response to any events that happen to occur, and cannot prevent these distracting responses from occurring when motivationally salient events are happening.

Overarousal of opponent drive representations can hereby have multiple effects. It can cause flat effect by desensitizing emotional dipoles to emotionally charged events. It can cause distractability in at least two ways: Overarousal can reduce the incentive motivational signals that help to focus attention upon motivationally relevant events (motivational distractability), and it can disinhibit orienting reactions whereby irrelevant events can continually disrupt attentional processing (orienting distractability). Hyperreactive orienting reactions may hereby be generated by hyporeactive emotional reactions (Ellinwood and Kilbey, 1980).

The incentive motivational pathways in Fig. 5 have been interpreted as generators of Continent Negative Variation, or CNV, event-related potentials, that covary with expectancy, decision (Walter et al., 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). The orienting reactions have been interpreted in terms of the N200 event-related potential, that has been linked to the processing of unexpected events (Renault and Lesèvre, 1978; Banquet et al., 1981; Näätänen et al., 1982). The CogEM model hereby suggests how both the CNV and the N200 can become abnormal in an overaroused emotional syndrome.

The model also suggests how the P300 event-related potential can also become abnormal in this way. In particular, CogEM interactions have been embedded within a larger framework, called Adaptive Resonance Theory, or ART, which also models how recognition categories are learned and recognized (Grossberg, 1982b, 1984b). ART suggests how the orienting subsystem may reset short-term memory in response to unexpected events, and thereby drive a memory search for a better-matching recognition category with which to represent the unexpected event. This short-term memory reset event has been interpreted in terms of the P300 event-related potential (Grossberg, 1978a, 1982b, 1984b; Banquet and Grossberg, 1987). Grossberg and Merrill (1996) have summarized how the CogEM model can be embedded into an ART recognition learning model that also includes learning of adaptively-timed motivated attention and movement. This extension clarifies how the CNV becomes adaptively timed.

Polyvalent interactions between sensory, drive, and motor representations

Where in the brain do such interactions occur? In order to make this connection, the circuit in Fig. 5 needs to be expanded. In its present form, after a reinforcing cue activates its sensory representation S, it can activate a motor representation M even as it sends conditioned reinforcer signals to a drive representation D. Thus a motor response can be initiated before the sensory representation receives incentive motivational feedback to determine whether the sensory cue should generate a response at that time. For example, eating behavior could be

initiated before the network could determine if it was hungry.

Even in the circuit of Fig. 5, each drive representation D obeys a polyvalent constraint whereby it can generate incentive motivational output signals only if it gets a sufficiently large primary or conditioned reinforcer input at the same time that it gets a sufficiently large internal drive input. The internal drive input designates whether an internal drive, such as hunger, thirst, sex, etc. is high and in need of satisfaction. Different drive representations exist to represent these distinct internal homeostatic states. Due to the polyvalent constraint, an external cue cannot activate strong incentive motivation, and with it action, to satisfy a drive that is already satisfied. On the other hand, the circuit, as it stands, could trigger such an action even if incentive motivational support is not forthcoming. A way is needed to prevent the sensory representation from triggering an action until it gets incentive feedback from a motivationally-consistent drive representation.

Figure 7 describes the minimal network in which this property can be achieved (Grossberg, 1971; Grossberg and Levine, 1987). In it, the sensory representation corresponding to a given cue is broken into two stages, or populations, rather than the single stage in Fig. 5. Presentation of a given cue, or CS, activates the first stage of its sensory representation. This activation is stored in short-term memory using the positive feedback pathways within the sensory representation. This stored

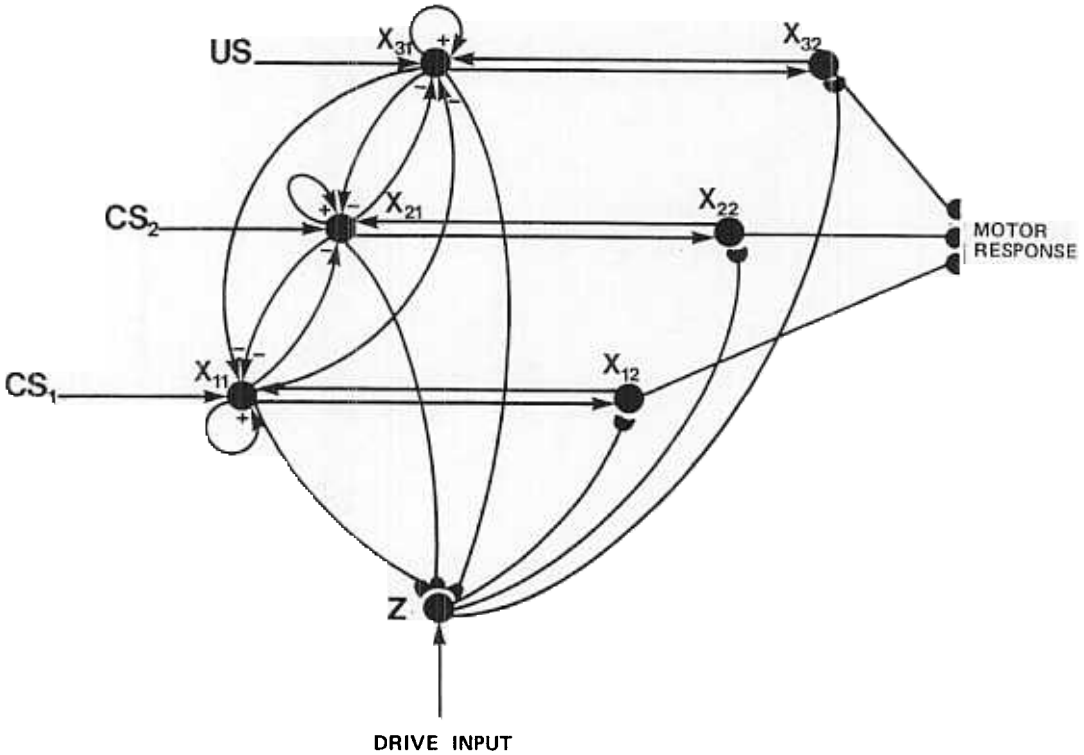

Fig. 7. Each sensory representation possesses (at least) two stages with STM activities x_{il} and x_{i2}. A CS or US input activates its corresponding x_{i1}. Activation of x_{i1} generates signals to x_{i2} and conditioned reinforcer signals to D. In response to a conditioned reinforcer CS, conditioned incentive motivational signals from D activate the second stages x_{i2}, which deliver feedback signals to the corresponding first stages x_{i1}. Some first stages x_{i1} are hereby amplified by conditionable feedback and block activation of other, less favored, sensory representations. Motor learning is elicited by sensory-motor signals from the winning x_{i2} to the motor representations.

activity gives rise to output signals to all the drive representations with which the sensory representation is linked, as well as to the second stage of its sensory representation. The second stage of its sensory representation obeys a polyvalent constraint; it cannot fire unless it receives converging signals from the first stage and from a drive representation.

To see how this works, suppose that the first stage of the sensory representation has a strong connection to one or more drive representations, whether the prewired connection of a primary reinforcer or the learned connection of a conditioner reinforcer. If the sensory representation strongly activates a drive representation when the drive representation is receiving a sufficiently large drive input, then the polyvalent constraint of the drive representation is satisfied and the drive representation can fire. All the drive representations that are active at that time compete among themselves to allow the most active one – the one that represents the best combination of sensory and drive information at that moment – to fire. If the winning drive representation has a strong prewired or learned incentive motivational pathway to the second stage of the cue's sensory representation, then the polyvalent constraint of the second stage is overcome, and the sensory representation can fire. Such firing can control the release of motivationally compatible actions. In summary, by making the final stages of both the sensory and the drive representations polyvalent, then the $S \rightarrow M$ motor pathways are activated only if the $S \rightarrow D \rightarrow S$ feedback pathway can get sufficiently activated.

Motivational amplification and blocking of sensory and cognitive processing

Figure 7 indicates how the second stage of sensory representation may be gated by motivational signals. How does the first stage of sensory representation benefit from motivational modulation? The model proposes that excitatory feedback pathways exist from the second stage to the first stage. Keep in mind that the second stage receives motivational input only if a drive representation with which it is associated is prepotent in the present cognitive-emotional context. Only those second stage representations that receive these motivational signals can fire. As a result, positive feedback from the second stages to the first stages amplifies those active first-stage sensory representations that are motivationally prepotent in the present context. This provides the motivational amplification of activity that enables these sensory representations to attentionally block less salient representations via $S \rightarrow S$ lateral inhibition.

Figure 7 illustrates why feedback from higher to lower stages of sensory and cognitive processing is needed to simultaneously achieve motivationally-appropriate sensory attention and responding. In particular, the top-down feedback enables motivationally-relevant selection of distributed information on the lower level of processing. Such top-down feedback is also needed to prevent sensory and cognitive learning from being destabilized and eroded by the 'blooming buzzing confusion' of irrelevant events (Grossberg, 1980, 1995).

Interactions between sensory cortices, amygdala, and orbital prefrontal cortex

The circuit in Fig. 7 may, in principle, be replicated at multiple stages in the thalamocortical and corticocortical elaboration of environmental cues. One such brain circuit is depicted in Fig. 8. This figure is taken from Barbas (1995), who noted that many different types of sensory cortex, including visual, somatosensory, auditory, gustatory, and olfactory cortex, are connected to both the amygdala and to the prefrontal cortex. In this interpretation of Fig. 7, the various sensory cortices play the role of the first stages of the sensory representations, the prefrontal cortex plays the role of the second stages of the sensory representations, and the amygdala plays the role of the drive representations. The amygdala has been identified in both animals and humans to be a brain region that is involved in learning and eliciting memories of experiences with strong emotional significance (Halgren et al, 1978; Gloor et al., 1982; Aggleston, 1993; LeDoux, 1993; Davis, 1994). The feedback between the second and first sensory stages may be interpreted as an example of the ubiquitous positive feedback that occurs between cortical regions

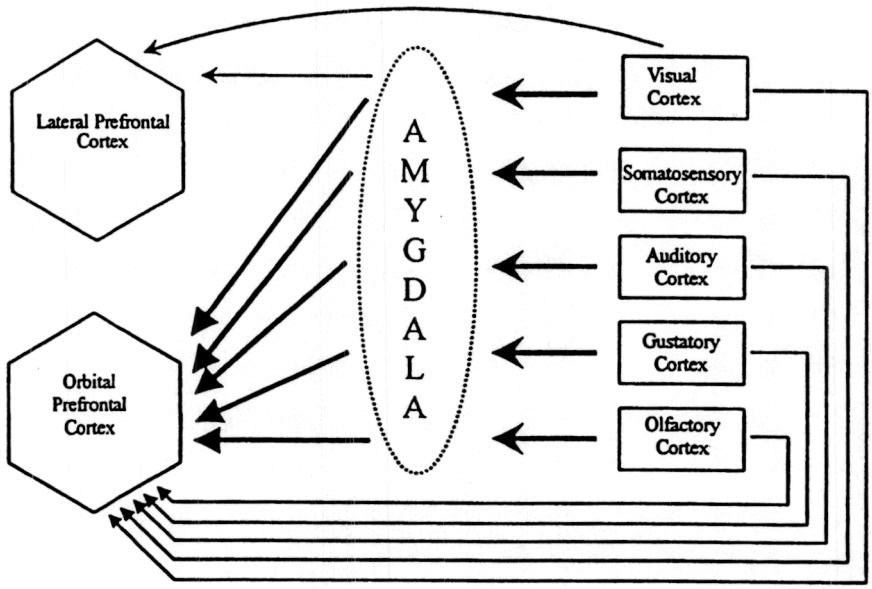

Fig. 8. Caudal orbitofrontal areas receive projections from sensory cortex (visual, somatosensory, auditory, gustatory, and olfactory) and from the amygdala, which also receives inputs from the same sensory modalities. (Reprinted with permission from Barbas (1995).)

(Macchi and Rinvik, 1976; Tsumoto et al., 1978; van Essen and Maunsell, 1983; Felleman and Van Essen, 1991; Sillito et al., 1994).

Schizophrenia and arousal: cognitive-emotional interactions

The formal symptoms of the CogEM model when its drive representations are overaroused (or underaroused) are strikingly reminiscent of schizophrenic symptoms. This linkage was made in Grossberg (1972b, 1984a,b) in an attempt to connect neural mechanisms of normal cognitive-emotional behavior with properties of schizophrenic behavior. Some of the main symptoms are now reviewed. Because the model is still under development, the proposed linkage is necessarily incomplete. In addition, other model mechanisms that are reviewed below may also contribute to schizophrenic behaviors; not all of these symptoms are attributed to direct and indirect effects of improperly aroused emotional dipoles.

Some types of schizophrenia have been ascribed to dopamine hyperactivity of various parts of the limbic system, including increased dopaminergic input to the amygdala (Lloyd, 1978; Reynolds, 1983, 1987). This type of effect may be interpreted as an overaroused condition. This hypothesis is consistent with data showing that dopaminergic agonists, such as L-dopa and amphetamine, can produce a behavioral syndrome that has been compared to schizophrenia (Riklan, 1973; Torrey and Peterson, 1974; Wallach, 1974; Stevens, 1993), although L-dopa has been reported to improve negative schizophrenic symptoms (Gerlach and Luhdorf, 1975; Albert and Rush, 1983). In the opposite direction, various antipsychotic drugs block dopamine receptors (Kuhar et al., 1978) and in sufficient quantities can produce a catalepsy that resembles Parkinson's disease (Hornykiewicz, 1975). This latter result, which suggests that some schizophrenics and Parkinson's patients are at opposite ends of a dopamine continuum, is consistent with model properties in the underaroused state that resemble Parkinson's disease (see below). More generally, the facts that an underaroused syndrome can be transmuted into an overaroused syndrome using a given drug, and that the reverse

transformation can be caused by an oppositely acting drug, suggest that the two syndromes may be extremal points on an inverted-*U* of a common mechanistic substrate, albeit one that may exist in multiple brain regions for different behavioral purposes. Because opponent processes like gated dipoles are assumed to exist in many brain regions, too much of a drug that is aimed at correcting a dopaminergic imbalance in one brain region may create an opposite dopaminergic imbalance in that and other brain regions. Multiple secondary effects, including lateralized effects that are different in different brain hemispheres, may also occur due to these dopaminergic abnormalities (Early et al., 1994), but these are beyond the scope of the present chapter.

When the drive representations of Fig. 7 are overaroused, the interpretation of this circuit using Fig. 8 is consistent with data suggesting a possible involvement of prefrontal cortices in schizophrenia (Weinberger, 1988). In support of the CogEM model hypothesis that the prefrontal sensory representation gates the release of properly motivated actions, Fuster (1989) has concluded from studies of monkeys that the orbital (ventral) prefrontal cortex helps to suppress inappropriate responses. These monkey data are consistent with clinical evidence that patients with injury to orbital prefrontal cortex tend to behave in an inappropriate manner (Blumer and Benson, 1975; Liddle, 1994). Other research has suggested that schizophrenia may involve a chronic deficiency in striatal glutamate transmission due to decreased activity in those regions of the prefrontal cortex that project to the striatum (Carlsson, 1988; Andreasen, 1990; Grace, 1991; Lynch, 1992). One possible cause of decreased prefrontal activity may be a reduction in incentive motivational signals from overaroused (or underaroused) amygdala circuits that project to the prefrontal cortex.

Other symptoms of schizophrenia are also similar to model properties. Since the time of Kraepelin (1913/1919), it has been noted that schizophrenics have difficulties with attentional control, motivation defects, and disorganization of behavior. Kraepelin wrote: 'This behavior is without doubt clearly related to the disorder of attention which we very frequently find conspicuously developed in our patients. It is quite common for them to lose both inclination and ability on their own initiative to keep their attention fixed for any length of time' (pp. 5–6). Attentional deficits in schizophrenia have also been emphasized by a number of other workers; e.g. Bleuler (1911/1950), Mirsky (1969) and Braff (1985).

Liddle (1994) has refined this analysis by segregating schizophrenic symptoms into 'three distinguishable syndromes: (1) psychomotor poverty (poverty of speech, flat affect, decreased spontaneous movement); (2) disorganization (disorders of the form of thought, inappropriate affect); and (3) reality distortion (delusions and hallucinations)' (p. 43), which have been supported by several studies (Arndt et al., 1991; Pantelis et al., 1991; Sauer et al., 1991). Liddle suggested that two of these syndromes 'reflect volitional disorders: psychomotor poverty reflects a difficulty initiating activity and disorganization reflects a difficulty in the selection of appropriate activity' (p, 43). Both of these problems are, moreover, associated with impairment in neuropsychological tests of frontal lobe function (Liddle and Morris, 1991).

The CogEM model suggests that one possible source of flat affect may be in overaroused emotional centers, such as the amygdala and its projections, and that this flat affect can lead to multiple deficits in behaviors that require the ability to sustain motivated attention on a consummatory task. Modeling work has not yet explicitly characterized how brain mechanisms of speech and movement control react to overarousal, although Grossberg et al. (1997) and Bullock et al. (1998) have modeled speech and movement control mechanisms that include volitional gain control mechanisms that may malfunction during certain mental disorders. One can nonetheless already discern how symptoms of poverty of speech, decreased spontaneous movement, disorders of the form of thought, and inappropriate affect might all be influenced by how flat affect reduces the incentive motivational signals that normally energize these behaviors. Reality distortions are ascribed below to overarousal of a different type of brain circuit.

How do schizophrenics lose a 'theory of mind'?

Frith (1992, 1994) has interpreted schizophrenic symptoms as impairments in the processes that underlie a 'theory of mind', including the ability to represent beliefs and intentions. For example, when asked to describe photographs of people, schizophrenics described their physical appearance, rather than their mental states (Pilowsky and Bassett, 1980). Frith noted, however, that the theory of mind approach 'does not explain the other major feature of negative schizophrenia: their impoverishment of will (Frith, 1994, p. 150). He also wrote that 'mental states include not only affects and emotions, but also goals and intentions. A person who was unaware of their goals could, on the one hand, be a slave to every environmental influence or, on the other hand, be prone to perseverative or stereotyped behavior, because they would not have the insight to recognize that certain goals were unobtainable or inappropriate' (Frith, 1994, p. 151).

The present model provides an intuitive framework that can begin to explain both types of behavior. Concerning the impoverishment of will: This loss may be linked to the flattening of affect and the consequence collapse of incentive motivational signals. Without these emotional/motivational resources, all mental activities that depend upon interpreting one's own emotional state, as well as the emotional states of others, will be diminished. Concerning goals and intentions: Without adequate incentive motivational signals, the prefrontal representations, such as those schematized in Figs 7 and 8, will not be adequately activated. Without adequately activated prefrontal representations, their top-down signals to earlier sensory and cognitive processing stages will be eliminated. As a result, these earlier representations will not be able to organize information according to its emotional meaning or to the individual's motivational goals. In addition, motivationally irrelevant information will not be adequately blocked from attention, thereby making it difficult to maintain attention upon motivationally relevant events. Or, in Kraepelin's words, schizophrenics 'lose both inclination and ability on their own initiative to keep their attention fixed for any length of time.' This summary illustrates how a problem that is localized within one type of brain circuit can seriously disturb cognitive and emotional processing throughout the entire network with which that circuit interacts.

The model summarized in Fig. 7 has been extended in various ways. One extension suggests how the hippocampal system may interact with cortical and amygdala circuits to learn new recognition categories and to adaptively time motivated attention to match situational constraints. Grossberg and Merrill (1996) have reviewed this extension and suggested why the cerebellum also contains adaptively timed circuits for the control of movement. Fiala et al. (1996) have modeled adaptive timing in terms of the metabotropic glutamate receptor system.

Contingent negative variation vs. readiness potential

The CogEM model helps to clarify the functional difference between the Contingent Negative Variation, or CNV, event-related potential (Walter et al., 1964; Brunia et al., 1985; Bribaumer et al., 1990) and the Bereitschaftspotential, or BP, or readiness potential (Kornhuber and Deecke, 1965). The BP is a DC potential that precedes motor action by 1 to 2 seconds, and appears to originate in the Supplementary Motor Cortex. The CNV is a slow negative potential of prefrontal origin that occurs even earlier than the BP, and has been associated with an animal's expectancy, decision, motivation, volition, preparatory set, and arousal (Fuster, 1995). Figure 9 summarizes the model hypothesis (Grossberg, 1975, Fig. 10; Grossberg, 1987b, p. 67) of how these two events may be related.

The functional need for this anatomical distinction may be understood from the following example. Consider the incentive motivational feedback that is generated by positive and negative drive representations. Both types of drive representation carry positive incentive motivational signals, because it is just as important to pay attention to a source of fear as it is to pay attention to a source of pleasure. These positive incentive signals can amplify the sensory representations corresponding to fearful or pleasurable events, and

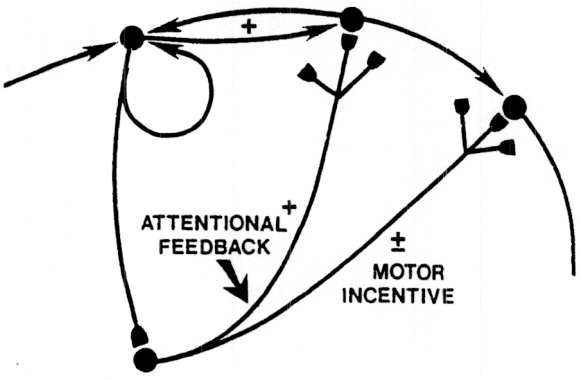

Fig. 9. Distinct incentive pathways: The positive attentional feedback pathway from both positive and negative drive representations is distinguished from the positive or negative feedback pathways by which positive and negative reinforcers can activate or suppress movements, or control approach and avoidance movements.

thereby rapidly focus attention on them. For example, in his experiments on the effects of mood on memory, Bower (1981) found that sad-congruent lists are learned no worse than happy-congruent lists. He also found that incongruent moods can interfere with recall, which can be explained by the competitive interactions between drive representations and cue representations. On the other hand, many conditioning data (e.g. Estes and Skinner, 1941; Reynolds, 1968; Estes, 1969; Maier et al., 1969; Grossberg, 1972a) describe how fearful and other negative drive sources can suppress responding. How can attention be drawn to fearful cues at the same time that these cues can suppress responding?

The CogEM model proposes that the incentive motivational inputs to the prefrontal cortex have positive sign, but that subsequent processing stages in the elaboration of motor actions may be modulated by both positive and negative motor arousal sources, which may also be used for the control of approach and avoidance behaviors. The former signals are linked to the CNV event-related potential, the latter to the BP event-related potential. Although this distinction would have to be carried much further to understand the details of how such responses are planned and executed, its very existence illustrates that decreased activity in prefrontal cortex undergoes processing at multiple stages before it influences observable actions.

Schizophrenia as an overaroused syndrome: working memory and learned serial order

The inverted-*U* that occurs in an opponent process is not the only type of inverted-*U* that can influence abnormal behaviors. Another inverted-*U* has been proposed to occur during the processing of serially ordered events, such as a sentence or a planned series of actions. Such a sequence of events is temporarily stored in working memory (Grossberg, 1978a,b; Baddeley, 1986) before relationships between the events are encoded in long-term memory by associative learning. A model of this process has been developed (Grossberg, 1969b, 1978a,b; Grossberg and Pepe, 1970, 1971) in which sequences of events cause working memory activations that decay due to interference by subsequent events and the passage of time. These active short-term memory traces generate learning signals which sample the distribution of activity across all the other event representations. Lateral inhibition among the representations enables the strongest associations to suppress weaker ones. In more complex versions of the model, as sequences of events are encoded in working memory, they trigger learning of cognitive planning chunks, or categories, that are selectively activated by particular event sequences. These chunks, in turn, learn to predict which subsequent events will occur.

Grossberg and Pepe (1970, 1971) discovered an inverted-*U* that occurs when such networks learn and perform sequentially ordered series of events at different levels of arousal. The underaroused end of this inverted-*U* is easily understood in terms of an insufficient amount of arousal with which to energize the learning and encoding of short-term memory patterns into long-term memory. The overaroused end of the inverted-*U* is more difficult to understand because an ample amount of arousal is available with which to energize learning and performance. However, the patterning of learning and performance through time is seriously impaired by overarousal. It has been mathematically proved (see Grossberg, 1974 and 1982a, for reviews) that when all of the representations in such an associa-

tive network are overaroused, there is a reduction of associative span, contextual collapse, and noisy network activations. In other words, the network loses its ability to represent plans and other higher-order contextual representations that depend upon sequential information. The resulting contextual collapse, fuzzy response categories, and punning based on low-order associations are also characteristics of schizophrenia (Maher, 1977).

These properties may clarify how the positive schizophrenic symptom of thought derailment may arise. Andreason (1979) defines derailment as 'A pattern of spontaneous speech in which the ideas slip off the track onto another that is clearly but obliquely related, or onto one that is completely unrelated.' This happens in overaroused serial learning networks because they cannot represent the higher-order temporal contexts that can keep thoughts 'on track'. Other positive symptoms, such as auditory hallucinations and thought insertions may also be, at least in part, due to the collapse of the ability to maintain a sequential context long enough for its meaning to be elaborated, and the decrease in the network's signal-to-noise ratio. Several types of evidence point to regions of the prefrontal cortex, such as the dorsolateral prefrontal cortex and the regions with which it interacts, as a substrate of working memory and its associative consequences (Fuster, 1973, 1989; Milner, 1982; Goldman-Rakic, 1987).

A synthesis of these proposed cognitive and emotional sources of arousal lead to the (greatly oversimplified) schematic shown in Fig. 10 of how several positive and negative schizophrenic symptoms may be generated in patients who may be generally overaroused. As noted below, however, because there is an inverted-U as a function of arousal in a gated dipole, some properties of overarousal can also be caused by underarousal. To the extent that these properties are due to opponent processes like gated dipoles, they can be differentiated by parametric properties of underarousal such as the following.

The underaroused depressive syndrome

A number of paradoxical properties are generated together in an underaroused gated dipole. These properties, which are listed below, are mathematically proved in the Appendix, and interpreted below in terms of symptoms of attention deficit disorder and Parkinson's disease.

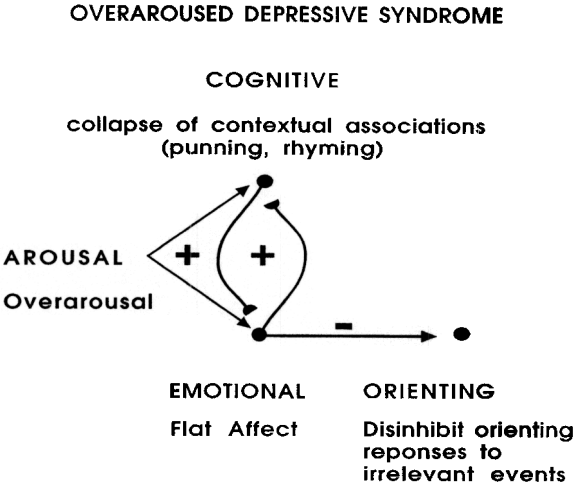

Fig. 10. Schizophrenia as an overaroused depressive syndrome: Overarousal of emotional opponent processes and of cognitive circuits for short-term and long-term memory of sequentially organized events (e.g. language, motor plans) can yield a combination of positive and negative symptoms.

A. Elevated response threshold to phasic inputs. The output threshold is elevated in response to a phasic input J (see Fig. 2). In other words, a larger intensity J is needed to elicit a positive ON-output from an underaroused dipole.

B. Suprathreshold hypersensitivity. The ON-reactions are hypersensitive to increments in input intensity J that exceed the elevated threshold. In other words, larger than normal ON-outputs are produced by suprathreshold input increments in an underaroused dipole than in a normally aroused dipole.

C. Sensitivity is brought down by a drug that brings arousal 'up'. These hypersensitive reactions are reduced by a drug that acts like an arousal 'upper.' In other words, a drug that causes a parametric increase in arousal level I (see Fig. 2), or leads to an equivalent effect through an action taking place at previous or subsequent processing stages, decreases dipole sensitivity to increments in J.

D. Too much 'up' causes an overaroused syndrome. Too much of an 'upper' drug can depress output size by carrying the dipole over its Inverted-*U* into the overaroused (large *I*) range.

E. Hyposensitive OFF-reactions occur to phasic input decrements. This is true despite the fact that hypersensitive ON-reactions occur in response to phasic input increments. In particular, no OFF-rebound may occur in response to cutting the phasic input *J* in half to *J*/2, and cutting an input *J*/2 to 0 may cause an abnormally small OFF-rebound. Since OFF-rebounds act to reset the dipole, underaroused dipoles may substitute paradoxical ON-reactions for the OFF-reactions that would have occurred in the normally aroused case.

F. Paradoxical dishabituation by unexpected events. Sudden increments ΔI in arousal level that cause an OFF-rebound in a normally aroused dipole can cause a paradoxical enhancement, or dishabituation, of the previous ON-response in an underaroused dipole. As a result, event representations that may have been attentionally blocked, or ignored, as being irrelevant, may attract attention when an unexpected event occurs.

Attention deficit disorder as an underaroused depressive syndrome

How these properties reflect themselves in the brain depends upon where the affected dipoles may be found. For example, in a case where sensory and/or motivational dipoles are affected, symptoms relevant to attention deficit disorder may be created, whereas when motor dipoles are affected – as in the basal ganglia systems that control how motor actions, among other processes, are gated ON and OFF (e.g. Horak and Anderson, 1984a,b) – symptoms familiar from Parkinson's disease emerge.

Consider, for example, the case of sensory dipoles and its interpretation in terms of attention deficit disorder. Here, the elevated response threshold to phasic cues may clarify why thresholds during an electroencephalic audiometry test are reduced by medications such as amphetamine (Weber and Sulzbacher, 1975). The suprathreshold hypersensitivity defines the behavioral syndrome. The reduction of sensitivity by an arousal 'upper' may be compared with the fact that children exhibiting this syndrome often suffer from catecholamine deficiencies (Shaywitz et al., 1977; Shekim et al., 1977) and amphetamine-type drugs are used as a treatment (Swanson and Kinsbourne, 1976; Weiss and Hechtmann, 1979).

The property that too much 'upper' causes an overaroused syndrome can be compared to data showing that an amphetamine psychosis can occur in response to large drug doses (Ellinwood and Kilbey, 1980; MacLennan and Maier, 1983). The property that hyposensitivity of OFF-reactions may occur to halving of a sensory cue ($J \rightarrow J/2$) is unknown. For example, does cutting a reward or punishment in half cause an abnormally small affective reaction of opposite sign? Does halving the intensity of a previously sustained visual cue cause an abnormally small negative after effect?

Finally, the property of paradoxical dishabituation by unexpected events predicts that irrelevant sensory cues can attract attention after an unexpected event occurs. To the extent that a reduced reset event maps into a reduced P300, such a reduction of the P300 may be expected to correlate with enhanced attention to irrelevant events.

Parkinson's disease as an underaroused depressive syndrome

In the case of Parkinson's disease, the elevated response threshold to phasic inputs is translated into the difficulty which Parkinson's patients have in initiating movements (Briley and Marien, 1994). The suprathreshold hypersensitivity is translated into their difficulty in terminating movements after they begin. The fact that they can be treated by an arousal 'upper' translates into the fact that, for example, in Parkinson's disease, dopamine-rich cells of the substantia nigra show marked degeneration (Weiner and Klawans, 1978) and L-dopa, a dopaminergic agonist, is used as a treatment.

The fact that too much of an 'upper' can cause an overaroused syndrome is interpreted in terms of the fact that too much L-dopa can elicit schizophrenic symptoms (Riklan, 1973; Wallach, 1974). The fact that these extremes are part of the same Inverted-*U* is illustrated by the fact that antipsychotic drugs that block dopamine receptors (Kuhar et al., 1978) can, in sufficient quantity, produce a catalepsy akin to Parkinson's disease (Hornykiewicz, 1975).

The predicted hyposensitivity of OFF-reactions to decrements in phasic inputs ($J \rightarrow J/2$) seems to be unknown. The paradoxical dishabituation by unexpected events has a natural analog in the fact that Parkinson bracing occurs in response to an unexpected push; that is, 'if suddenly pushed forward or backward while standing, many people with Parkinson's brace rigidly without stepping, or with short shuffling steps which are unable to counteract their fall' (Schallert et al., 1979). Why do these patients not right themselves as normal people do, or just fall over? In the model, these bracing reactions may be at least partly caused by the enhanced ON-reactions of the motor commands that were active before the push. An enhanced ON-reaction would strengthen the current motor pattern, rather than rebounding it to an antagonistic pattern that could facilitate a righting reaction. The hypothesis that such reactions are due to under-aroused circuits are consistent with data showing that intraventricular application of 6-OHDA severely depletes brain catecholamines and thereby produces symptoms such as catalepsy, akinesia, and Parkinson bracing (Levitt and Teitelbaum, 1975; Schallert et al., 1978a,b, 1979). This interpretation suggests the utility of studying how novelty-mediated motor potentials may vary with the amount of bracing.

Identification of a key opponent processing circuit and how it may be afflicted during a disease like Parkinson's is only one step in developing a more complete neural theory of the disease. Contreras-Vidal and colleagues (e.g. Contreras-Vidal and Stelmach, 1995; Teulings et al., 1997; Contreras-Vidal et al., 1998; Van Gemmert et al., 1998) have suggested how a gated dipole circuit, suitably specialized, may be embedded within a larger theory of sensory-motor control (e.g. Bullock and Grossberg, 1988; Bullock et al., 1993a,b; Bullock et al., 1998) to provide a more complete explanation of Parkinsonian symptoms.

Weber law models of mental disorders: similar symptoms with opposite causes?

By contrast with the hyperractive orienting reactions that may be indirectly released by a hyposensitive overaroused gated dipole, the hyporeactive reactions that can occur in an underaroused gated dipole are direct properties of this circuit. Although these two types of reactions may look similar to casual behavioral analysis, they may be differentiated in terms of their triggering events and parametric properties. Likewise, although both underaroused and overaroused depressive syndromes may both cause a reduction in output from an afflicted brain region, whether amygdala, basal ganglia, or prefrontal cortex, these reductions may be due to different, indeed, opposite causes that lie a polar ends of an inverted-U. Some authors view schizophrenic and Parkinsonian symptoms as having a similar cause (e.g. Ingvar, 1996), whereas others suggest that 'although the pattern of impairment [of schizophrenics] was similar to that seen in Parkinson's disease, different underlying processes may be involved in the two conditions' (Pantelis and Nelson, 1994, p. 223). The existence of opponent processes that exhibit similar properties at opposite ends of their Inverted-U make the interpretation of these data more difficult.

Neural models of normal cognitive and emotional behaviors may provide an additional tool by which the brain mechanisms underlying these abnormal behaviors may be unified, classified, and explained. The habituative dynamics of arousal-modulated opponent processing circuits have been particularly rich in data implications. In addition to the types of properties that have been summarized herein, gated dipoles have been used to explain data about decision making under risk, gambling, memory repression, self-punitive behaviors, eating disorders, analgesic effects, and sleep rhythms, among others (Grossberg, 1972a,b, 1982b, 1984b; Carpenter and Grossberg, 1983, 1984, 1985; Grossberg and Gutowski, 1987).

That such a simple combination of neural mechanisms can begin to rationalize a wide range of normal and abnormal behavioral and neural properties provides converging evidence that mechanisms of this type are used by the brain. A key component in these explanations is the way in which tonic arousal sets the sensitivity to phasic inputs when they both activate habituative transmitters and opponent competition. Taken together, these properties realize a Weber Law explanation of various mental disorders. Such a Weber Law, as in

visual psychophysics, suggests how the size of a baseline or tonic input can influence the sensitivity to a phasic input that is superimposed upon it.

Grace (1991) has also described a Weber Law model of schizophrenia in which tonic baseline signals play a key role in determining the brain's sensitivity to phasic inputs. As in the present theory, Grace notes that low arousal can cause hyperreactive responses to phasic inputs, whereas high arousal can cause hyporeactive responses, and uses this hypothesis to interpret data about dopamine metabolism in more detail than was attempted here. Grace suggests that low arousal due to abnormally low prefrontal activity is the basis of schizophrenia. He does not focus on the possible causative role of limbic overarousal or underarousal in causing flat affect and, with it, low levels of prefrontal activity. The model of Grace also does not incorporate the possible role of opponent interactions, and does not make significant contact with behavioral data.

Thus, although the Grace (1991) model and the Grossberg (1972b, 1984a,b) model both emphasize Weber Law processing as key in these mental disorders, there are significant differences in other model hypotheses. Deciding definitively between them may require more complete models of how the prefrontal cortex, basal ganglia, amygdala, and their interactions with other brain regions generate behavioral properties. Such models are currently being developed in a number of laboratories worldwide. A better mechanistic understanding of the neural substrates of schizophrenia and other arousal-modulated mental disorders may thus soon be available.

Appendix: gated dipoles

Transmitters as gates

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

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

$$T = SB, \tag{1}$$

where $B > 0$, on the next nerve cell v_2. Suppose, in addition, that the signal from v_1 to v_2 is due to the release of a chemical $z(t)$ from v_1 that activates v_2. If such a chemical transmitter is persistently released when S is large, what keeps the net signal, T, from getting smaller and smaller as v_1 runs out of transmitter? Some means of replenishing or accumulating the transmitter must exist to counterbalance its depletion due to release from v_1. To accommodate this interpretation, we can rewrite Eqn (1) in the form

$$T = Sz \tag{2}$$

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

$$z(t) \cong B \tag{3}$$

at all times t? This is a question about the sensitivity of v_2 to signals from v_1, since if z could decrease to small values, then even large signals S would have only a small effect on T. Equation (2) has the following interpretation. The signal, S, causes the transmitter, z, to be released at a rate $T = Sz$. Whenever two processes, such as S and z, are multiplied, they are said to interact by mass action, or that z gates S. Thus, (2) says that z gates S to release a net signal T, and Eqn (3) says that the cell tries to replenish z to maintain the system's sensitivity to S. The simplest law that joins together both (2) and (3) is the following differential equation for the net rate of change, dz/dt, of z:

$$\frac{dz}{dt} = A(B - z) - Sz \tag{4}$$

Equation (4) describes the following four processes going on simultaneously.

Accumulation or Production and Feedback Inhibition: The term $A(B - z)$ enjoys two possible interpretations, depending on whether it represents

a passive accumulation process or an active production process. In the former interpretation, there exist B sites to which transmitter can be bound, z sites are bound at time t, and $B-z$ sites are unbound. Then term $A(B-z)$ says that transmitter is bound at a rate proportional to the number of unbound sites. In the latter interpretation, two processes go on simultaneously. Term AB on the right-hand side of Eqn (4) says that z is produced at a constant rate AB. Term $-Az$ says that once z is produced, it inhibits the production rate by an amount proportional to the concentration of z. In biochemistry, such an inhibitory effect is called feedback inhibition by the end product of a reaction. Without feedback inhibition, the constant rate of production, AB, would eventually cause the cell to burst. With feedback inhibition, the net production rate is $A(B-z)$, which causes $z(t)$ to approach the finite amount B, as we desire by Eqn (3). The term $A(B-z)$ thus enables the cell to accumulate a target level B of transmitter.

Gating and Release: Term $-Sz$ in Eqn (4) says that z is inactivated or released at a rate Sz. As in Eqn (2), inactivation or release of z is due to a mass action interaction, or gating, of S by z.

Equations (2) and (4) describe the simplest dynamic law that corresponds to constraints (2) and (3). These equations reconcile the two constraints of unbiased signal transmission and maintenance of sensitivity when the signals are due to release of transmitter.

Weber-law adaptation and habituation

To determine how the net signal, $T=Sz$, reacts to a sudden change in S, as in Fig. 3, suppose that $z(t)$ reacts slowly compared to the rate with which $S(t)$ can change. For definiteness, suppose that $S(t)=S_0$ for all times $\tau \leq t_0$ and that, at time $t=t_0$, $S(t)$ suddenly increases to S_1. By Eqn (4), $z(t)$ reacts to the constant value $S(t)=S_0$ by approaching an equilibrium value $z(t_0)$. This equilibrium value is found by setting $dz/dt=0$ in Eqn (4) and solving for

$$z(t_0)=\frac{AB}{A+S_0} \tag{5}$$

By Eqn (5), a larger value of S_0 causes more transmitter to be inactivated or released. In other words, $z(t_0)$ is a decreasing function of S_0. By contrast, Eqn (2) implies that the net signal to v_2 at time t_0 is

$$S_0 z(t_0)=\frac{ABS_0}{A+S_0} \tag{6}$$

By Eqn (6), the rate of transmitter release is an increasing function of S_0. Now let $S(t)$ switch to the value $S_1>S_0$. Because $z(t)$ is slowly varying, $z(t)$ approximately equals $z(t_0)$ for awhile after $t=t_0$. Thus, the net signal to v_2 during these times is approximately equal to

$$S_1 z(t_0)=\frac{ABS_1}{A+S_0} \tag{7}$$

Equation (7) has the same form as a Weber law, $J(A+I)^{-1}$. The signal S_1 is evaluated relative to the baseline, S_0, just as J is evaluated relative to I. This Weber law is due to slow intracellular adaptation of the transmitter gate to the input level through time. It is not due to fast intercellular lateral inhibition across space (Grossberg, 1980, Appendix C and D), which also obeys a Weber law. Many of the properties derived below are due to this intracellular Weber law.

As $z(t)$ in Eqn (4) begins to respond to the new transmitter level, $S=S_1$, $z(t)$ gradually approaches the new equilibrium point that is determined by $S=S_1$, namely

$$z(\infty)=\frac{AB}{A+S_1}. \tag{8}$$

The net signal consequently decays to the asymptote,

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

Thus, after $S(t)$ switches from S_0 to S_1, the net signal Sz jumps from (6) to (7) and then gradually decays to Eqn (9). The exact course of this decay is described by the equation

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

for $t \geq t_0$, which shows that the rate, or gain, $A+S_1$ of the response increases with the signal S_1, just as in the case of shunting lateral inhibition (Grossberg, 1980). The sudden increment followed by slow decay can be intuitively described as an overshoot followed by habituation to the new sustained signal level, S_1 (see Fig. 3). Both intracellular adaptation and habituation occur whenever a transmitter fluctuates more slowly than the signals that it gates.

The size of the overshoot can be found by subtracting Eqn (9) from Eqn (7). For definiteness, let $S_0=f(I)$ and $S_1=f(I+J)$, where $f(w)$ is a function that transmutes the inputs I and $I+J$ that exist before and after the increment J into net signals S_0 and S_1, respectively. Then the overshoot size is approximately

$$S_1 z(t_1) - S_1 z(\infty) = \frac{ABf(I+J)[f(I+J)-f(I)]}{[A+f(I)][A+f(I+J)]}. \quad (11)$$

It is shown below that the rebound size in response to specific cue offset is related to (11) in a way that allows both $f(w)$ and the arousal level, I, to be estimated.

Intracellular habituation due to a slow transmitter gate is not the only type of habituation in the brain. An intercellular variety of habituation can also occur. After a feedback expectancy is learned, a mismatch of the feedback expectancy with feedforward data can trigger an orienting reaction by dishabituating the network's orienting subsystem (Grossberg, 1980; Grossberg and Merrill, 1996). Feedback expectancies and slow gates are both needed to regulate perceptual and motivational events, but they are quite distinct mechanistically.

A gated dipole

It is shown below how, if transmitters gate signals before the gated signals compete, as in Fig. 2, then antagonistic rebound can be elicited by offset of a specific cue, as in light-ON vs. light-OFF, or fear vs. relief. It is also shown how unexpected events can cause an antagonistic rebound. They do this by triggering an increase in the level of nonspecific arousal that is gated by all the transmitter pathways.

Figure 11 depicts the simplest network in which two channels receive inputs that are gated by slowly varying transmitters before the channels compete to elicit a net output response. In such a feedforward gated dipole, specific phasic inputs are turned on and off by internal or external cues and nonspecific arousal inputs are on all the time, or tonic, even though their size can vary through time. Each channel can have its own sum of specific inputs, K_1 or K_2, such as hunger or satiety drive inputs, respectively, that are added to positive or negative conditioned reinforcer signals. Both channels also receive the same arousal input, L. The total signals to the two channels are, therefore, $S_1=f(K_1+L)$, where the signal function, $f(w)$, is monotone increasing.

The relative sizes of S_1 and S_2 and their rates of change through time relative to the transmitter fluctuation rate determine whether an antagonistic rebound will occur. To emphasize this fact, let

$$I=\min(K_1+L, K_2+L) \quad (12)$$

and

$$J=|K_1-K_2| \quad (13)$$

The quantity I determines the network's net arousal level and J determines how asymmetric the inputs are to the two channels (cf. Fig. 2). Suppose, for definiteness, that $K_1>K_2$. Then $S_1=f(I+J)$ and $S_2=f(I)$. The notational shift from $S_1=f(K_1+L)$ and $S_2=f(K_2+L)$ to $S_1=f(I+J)$ and $S_2=f(I)$ in Eqns (12) and (13) is motivated by more than formal convenience. The notation I and J emphasizes that the dipole does not know how many input sources are perturbing it through time. All it can compute is the net arousal level, I, and the degree of asymmetry, J, above I, whether one or a million input sources are active. If a million cues equally perturb the ON-channel (positive reinforcers) and another million cues equally perturb the OFF-channel (negative reinforcers), the net effect of all the cues will be to increase I, not J. Thus, after dipole competition takes place, all these cues need not generate any incentive motivation. On the other hand, by increasing I, these cues can alter the sensitivity of the dipole to other asymmetrically distributed inputs due to the dipole's inverted-U

properties. This is the kind of simple but subtle distinction that the I and J notation emphasizes.

Rebound due to phasic cue offset

A rebound can be caused if, after the network equilibrates to the input J, the input is suddenly shut off (see J in Fig. 2). This effect is analogous to the reaction that occurs when a previously sustained light is shut off or a previously sustained aversive cue is shut off. To see how this rebound is generated, suppose that the arousal level is I and that the cue input is J. Let the total signal in the ON-channel be $S_1 = f(I+J)$ and that in the OFF-channel be $S_2 = f(I)$. Let the transmitter in the ON-channel, z_1, satisfy the equation

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

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

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

After z_1 and z_2 equilibrate to S_1 and S_2, $(d/dt)z_1 = (d/dt)z_2 = 0$. Thus, by Eqns (14) and (15),

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

and

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

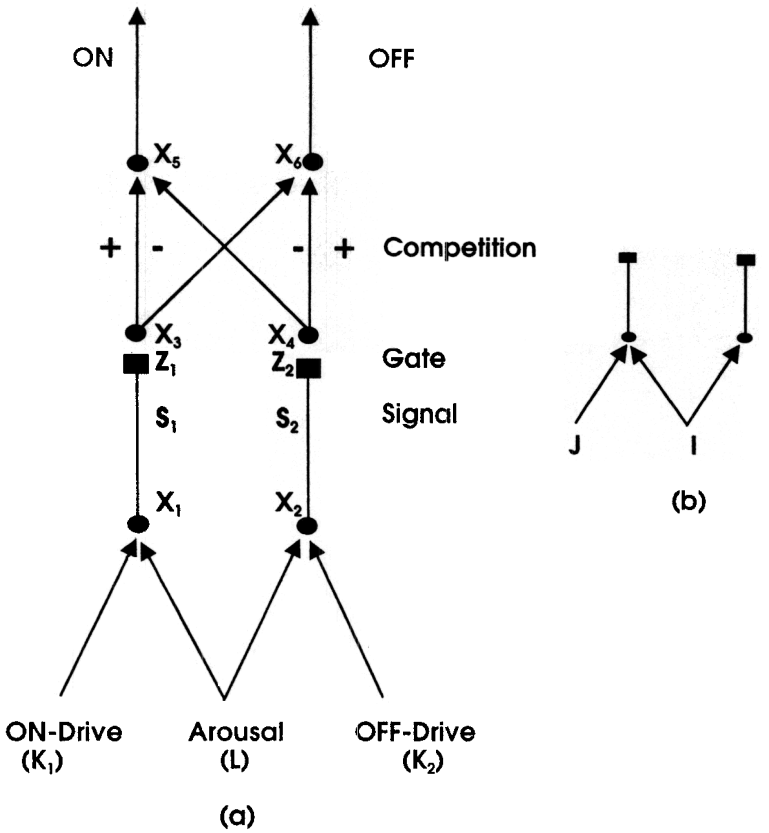

Fig. 11. (a) Specific inputs (K_1 and K_2) and a nonspecific input (L) have the same effect on a gated dipole as (b) a specific input J and a net arousal level I if $K_1 > K_2$.

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

$$S_1 z_1 = \frac{ABS_1}{A + S_1} \tag{18}$$

and

$$S_2 z_2 = \frac{ABS_1}{A + S_2}, \tag{19}$$

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

$$S_1 z_1 - S_2 z_2 = \frac{A^2 B[f(I+J) - f(I)]}{[A + f(I)][A + f(I+J)]}. \tag{20}$$

Division of the overshoot amplitude Eqn (11) by the sustained ON-output amplitude Eqn (20) yields an interesting relationship between the size of the overshoot in the ON-channel and the size of the steady-state ON-output; namely,

$$\frac{\text{on-overshoot}}{\text{steady on-output}} = \frac{f(I+J)}{A}. \tag{21}$$

which provides an estimate of $f(w)$ if J is parametrically varied. In particular, if $f(w)$ is a linear signal, $f(w) = w$, then Eqn (20) becomes

$$S_1 z_1 - S_2 z_2 = \frac{A^2 BJ}{(A+I)(A+I+J)}, \tag{22}$$

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

Now shut J off to see how an antagonistic rebound (relief) is generated. The cell potentials rapidly adjust until new signal values, $S_1^* = f(I)$ and $S_2^* = f(I)$, obtain. However, the transmitters z_1 and z_2 change much more slowly, so that (16) and (17) are approximately valid in a time interval that follows J offset. Thus, the gated signals in this time interval approximately equal

$$S_1^* z_1 \cong \frac{ABf(I)}{A + f(I+J)} \tag{23}$$

and

$$S_2^* z_2 \cong \frac{ABf(I)}{A + f(I)}. \tag{24}$$

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

$$S_2^* z_2 - S_1^* z_1 = \frac{ABf(I)[f(I+J) - f(I)]}{[A + f(I)][A + f(I+J)]}. \tag{25}$$

Division of the rebound amplitude Eqn (25) by the steady-state ON-output Eqn (20) yields an interesting relationship between the maximal OFF-rebound-output and the steady ON-output; namely,

$$\frac{\text{off-rebound}}{\text{on-output}} = \frac{f(I)}{A}, \tag{26}$$

which provides an estimate of $f(w)$ as I is parametrically varied. A comparison of Eqn (21) with (26) shows that, as I is parametrically varied, Eqn (21) should have the same graph as Eqn (26), shifted by J. This comparison provides an estimate of J (that is, of how the behavioral input is transformed into neural units) and also a strong test of the model. Once $f(w)$ is estimated, Eqns (20) and (25) can be verified. If $f(w) = w$ in Eqn (25), then

$$S_2^* z_2 = S_1^* z_1 = \frac{ABIJ}{(A+I)(A+I+J)}. \tag{27}$$

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

The rebound is transient (see OFF in Fig. 2) because the equal signals, $S_1 = S_2 = f(I)$ gradually equalize the z_1 and z_2 levels until they both approach $AB(A + f(I))^{-1}$. Then $S_1 z_1 - S_2 z_2$ approaches zero, so the competition between channels shuts off both of their outputs.

Rebound due to arousal onset

A surprising property of gated dipoles is their reaction to sudden increments in the arousal level, I. Such increments may, for example, occur in response to unexpected events. Suppose that the ON-channel and the OFF-channel have equilibrated

to the input levels I and J. Now suddenly increase I to I^*, thereby changing the signals to $S_1^*=f(I^*+J)$ and $S_2^*=f(I^*)$. The transmitters z_1 and z_2 continue to obey Eqns (16) and (17) for a while, with $S_1=f(I+J)$ and $S_2=f(I)$. A rebound occurs if $S_2^* z_2 > S_1^* z_1$. In general,

$$S_2^* z_2 - S_1^* z_1 = \frac{\begin{array}{c} AB[f(I^*)-f(I^*+J)] \\ +B[f(I^*)f(I+J)-f(I)f(I^*-J)] \end{array}}{[A+f(I)][A+f(I+J)]}$$

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

$$I^* > I+A, \tag{29}$$

since then

$$S_2^* z_2 - S_1^* z_1 = \frac{ABJ(I^*-I-A)}{(A+I+J)(A+I)}. \tag{30}$$

Thus, given a linear signal function, a rebound will occur if I^* exceeds $I+A$ no matter how J is chosen. If the event is so unexpected that it increments the arousal level by more than amount A, then all dipoles in the network will simultaneously rebound. Moreover, the size of the OFF-cell rebound increases as a function of the size of the ON-cell input, J, as Eqn (30) shows. In particular, no rebound occurs if the ON-cell was inactive before the unexpected event occurs. Thus, the rebound mechanism is selective. It rebounds most vigorously those cells which are most active ($J \gg 0$) and spares inactive cells ($J \cong 0$).

Inverted-U in dipole output

The inverted-U effect holds if $f(w)$ is a sigmoid, or S-shaped, function; that is, if $f(0)=df/dw(0)=0$, $df/dw(w)>0$ if $w>0$, $f(\infty)<\infty$, and $d^2f/dw^2(w)$ changes sign once from positive to negative as w increases. Sigmoid signal functions are found in many neural systems if only because of their noise suppression and contrast-enhancement properties (cf. Grossberg, 1980). In particular, if $f(w)$ is sigmoid, an inverted-U occurs in the sustained ON-output Eqn (20) as I is parametrically increased, despite the fact that an inverted-U does not obtain in (22) when $f(w)$ is linear. The results are simplified by using the signum function

$$\text{sgn}\{w\} = +1 \text{ if } w>0,\ 0 \text{ if } w=0, \text{ and } -1 \text{ if } w<0. \tag{31}$$

First consider the ON-reaction in Eqn (20), which is denoted by x_5 (Fig. 11). Writing the derivative of a function $g(I)$ as $g'(I)$, then, by Eqn (20), for each fixed J,

$$\begin{aligned} \text{sgn}\{x_5'(I) = \text{sgn}\{&A^2[f'(I+J)-f'(I)] \\ &+2A[f(I)f'(I+J)-f(I+J)f'(I)] \\ &+[f^2(I)f'(I+J)-f^2(I+J)f'(I)]\}. \end{aligned} \tag{32}$$

Since $f(w)$ is sigmoid,

$$f(0)=f'(0)=0. \tag{33}$$

Thus, by Eqns (32) and (33),

$$\text{sgn}\{x_5'(0)\} = \text{sgn}\{A^2 f'(J)\} > 0. \tag{34}$$

At large values of I,

$$f(I+J) > f(I), \tag{35}$$

whereas

$$f'(I+J) < f'(I). \tag{36}$$

Consequently, each term in brackets on the right-hand side of Eqn (32) is negative. Thus, at large I values,

$$\text{sgn}\{x_5'(I)\} < 0 \tag{37}$$

The inequalities in Eqns (34) and (37) show that, for fixed J, $x_5(I)$ increases and then decreases as a function of I. This is the inverted-U for the ON-reaction. In fact, since $f(\infty)<\infty$, Eqn (20) implies that $\lim_{I\to\infty} x_5(I)=0$. A similar proof holds for the OFF-reaction.

Hypersensitive underaroused reaction to phasic increments

To illustrate why the underaroused syndrome is hypersensitive to phasic increments, suppose that I is chosen abnormally small and, consequently, that $f(I)$ is very small because of f's S-shaped graph. Let J represent the intensity of a fearful cue (e.g., a shock level) and let the dipole ON-output Eqn (20) be correlated with the amount of fear. Since I is so small, the 'fear threshold is raised' in the sense that a larger value of J is needed to create a large net

ON-output than when I is chosen in the 'normal' range. Although the fear threshold is high, once J is chosen sufficiently large to elicit a detectable net ON-reaction, additional increments in J create larger than normal increments in fear. This is because the terms $f(I)$ in the numerator and denominator of Eqn (20) are abnormally small. More precisely, differentiating Eqn (20) with respect to J, we find the rate at which the ON-output increases to unit increases in J. This rate is

$$\frac{\partial}{\partial J}(S_1 z_1 - S_2 z_2) = \frac{A^2 B f'(I+J)}{[A+f(I+J)]^2}. \qquad (38)$$

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

Paradoxical on-reaction to unexpected events and differential enhancement of unattended cues

Two other properties of underaroused dipoles are related to Parkinsonian bracing. These properties, like underaroused hyperexcitability, are due to the faster-than-linear, or threshold, behavior of the S-shaped signal function, $f(w)$, at small activity values, w. Neither property holds if the signal function is linear, say $f(w)=w$. In particular, by Eqn (30), when $f(w)=w$, an arousal increment ΔI in response to an unexpected event causes a rebound whenever $\Delta I > A$. The minimal ΔI capable of causing a rebound is independent of the ambient arousal level, I. This property does not hold when $f(w)$ grows faster than linearly, say $f(w)=w^2$, which approximates the sigmoid shape of $f(w)$ at low arousal levels. By (28), a rebound occurs when $f(w)=w^2$ only if

$$\Delta I > g(I, J), \qquad (39)$$

where the function

$$g(I, J) = \frac{A - I(I+J) + (A+I^2)^{1/2}[A+(I+J)^2]^{1/2}}{2I+J} \qquad (40)$$

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

$$\frac{\partial}{\partial \Delta I}\left[\frac{(I+\Delta I+J)^2}{} \quad \frac{(I+\Delta I)^2}{}\right]$$

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

Use of a sigmoid function also helps explain how, in response to an arousal burst, previously unattended sensory representations can be enhanced even while very active sensory representations are inhibited. This is because the function $g(I, J)$ is a decreasing function of J, as well as of I. This means that it is easier to rebound a more active sensory representation than a less active sensory representation.

Paradoxical lack of rebound to phasic decrement: ordering of reinforcement magnitude

This section illustrates how several behavioral indices should all covary as arousal level is parametrically increased. The first index says that reducing J units of shock (or other negative reinforcer) to $J/2$ units is less rewarding (i.e. produces a smaller rebound) than reducing $J/2$ units of shock to 0 units, despite the fact that both operations reduce shock by $J/2$ units. This result is based on the fact that Eqns (20) and (25) include Weber law ratios of I and J terms as well as differences of I and J terms. A formula has been derived that predicts when reducing J_1 units of shock to K_1 units at arousal level I_1 is more reinforcing than reducing J_2 units of shock to K_2 units at arousal level I_2 (Grossberg, 1972b). To make these assertions, assume that the size of the relief rebound caused by reducing the shock level is proportional to the rewarding effect of the manipulation, other things being equal.

To simplify the computations, it is convenient to use a signal function

$$f(w) = \max(w - C, 0). \qquad (42)$$

Such a signal function has a threshold C, below which it equals 0 and above which it grows linearly.

This threshold function approximates a sigmoid function in the activity range before saturation occurs. Denote the steady-state ON-reaction that occurs after a specific input of intensity J is kept on for S time units by $x_5(S, J \rightarrow K)$ and the OFF-rebound that occurs when intensity J is switched to K at time $t=S$ by $x_6(S^+, J \rightarrow K)$. To compute $x_6(S^+, J \rightarrow K)$, the transmitters z are approximated by their steady-state values at $t=S$ and the potentials x by their new steady-state values in response to input K.

Given an arousal level I that exceeds the threshold, C, then

$$, J \rightarrow \frac{J}{2}\Big) = \frac{AB\frac{J}{2}(I-A-C)}{(D+I)(D+I+J)}, \tag{43}$$

where $D=A-C$. By comparison, Eqns (20) and (25) imply that

$$x_5(S, J \rightarrow 0) = \frac{A^2BJ}{(D+I)(D+I+J)} \tag{44}$$

and

$$J \rightarrow 0) = \frac{ABJ(I-C)}{(D+I)(D+I+J)} \tag{45}$$

from which it also follows that

$$\frac{J}{2} \rightarrow 0\Big) = \frac{AB\frac{J}{2}(I-C)}{(D+I)\left(D+I+\frac{J}{2}\right)} \tag{46}$$

and

$$\frac{x_6(S^+, K \rightarrow 0)}{x_5(S, K \rightarrow 0)} = A^{-1}(I-C) \tag{47}$$

for any $K>0$. Comparing Eqns (43) and (46) shows that the relative rebound sizes satisfy

$$x_6\left(S^+, \frac{J}{2} \rightarrow 0\right) > x_6\left(S^+, J \rightarrow \frac{J}{2}\right), \tag{48}$$

or that cutting J units in half is less rewarding than shutting off $J/2$ units. In addition, the ratio (47) increases with I, as in the more general Eqn (26). Substituting Eqn (47) into Eqn (43) shows that

$$x_6\left(S^+, J \rightarrow \frac{J}{2}\right) = \frac{A^2B\frac{J}{2}\left[x_5^{-1}(S, K \rightarrow 0)x_6(S^+, K \rightarrow 0) - 1\right]}{(D+I)(D+I+J)} \tag{49}$$

By Eqn (49), an arousal level that favors the possibility of learned avoidance in the presence of fearful cues (i.e. the OFF-rebound is much bigger than the ON-response so that the right hand side of (49) is positive) also favors a large rewarding effect when the shock level is halved. If I is chosen to be small (underarousal), then x_6 in Eqn (43) can be negative (no rebound occurs) even if x_6 in Eqn (46) is positive (a rebound occurs).

Such dipole properties are linked to the membrane equations that define cell dynamics in Grossberg (1984b).

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