Emotion. New York: The Rockefeller University Press and Russell Sage Foundation.

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Emotion: Steps Toward a Neuropsychological Theory

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DISCUSSION

Do the things external which fall upon you distract you? Give yourself time to learn something new and good, and cease to be whirled around. But then you must also avoid being carried about the other way. For those too are triflers who have wearied themselves in life by their activity, and yet have no object to which to direct every movement, and, in a word, all their thoughts.

MARCUS AURELIUS: *Meditations*

INTRODUCTION

Marcus Aurelius, Rome's philosopher-emperor, developed a formula for coping with this troubled world. He pointed out that if one tries to consider problems all-of-a-piece one is overwhelmed. His prescription was simple: segment the reach of awareness; attend only to one facet of the situation at any one time; act upon that facet and then proceed to another. Too-much-too-soon is upsetting. Segmentation reduces the demands upon awareness and thereby produces imperturbability.

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This bit of wisdom can serve as the kernel for a modern neuropsychological theory of emotion. A theory crystallized from this kernel would look considerably different from those presently in vogue, yet such a theory would have to account for the popularity of these views.

Current theories of emotion—in fact, psychological theories in general—fall into two major groups: the social–behavorial, which includes the subjective, or "intrapsychic"; and the biological, which includes the physical, chemical, and, of course, the neurological. Terms are all too often taken from one frame of reference and applied to another in haphazard and uncritical fashion. In the approach presented here, every effort will be made to keep the two universes of discourse clear. The assumption is, however, that because each of these conceptual universes denotes some body of events common to both, different aspects of this common body will be illuminated by approaching it from different points of view.

Although the initial focus of the presentation is neurological, it is not made solely in neurological terms, but interdigitates the subjective-behavioral approach with the neural, and includes some aspects of the communications sciences. In this sense, the presentation is both neurobehavioral and neurocybernetic in its conception.

PREVIEW OF THE PROPOSAL

This proposal delineates the emotions as a set of processes that 1) reflect the state of relative organization or disorganization of an ordinarily stable configuration of neural systems; and 2) reflect those mechanisms which operate to redress an imbalance, not through action, but by the regulation of input. Two such mechanisms have been identified: one achieves stability by mobilizing the already available subsystems to exclude input; the other reorganizes the system to include input. These "preparatory" and "participatory" processes thus achieve control by an "internal" and an "external" route. Much of this presentation is devoted to setting forth the experimental evidence for these mechanisms and to suggest some inferences that can be drawn.

In the past, neurological theories have emphasized the relations between viscera and emotion or have linked emotion quantitatively

with an amount of neural excitation. These relationships, although substantial, take into account neither the complexities of the emotional process nor the intricacies of the relevant neural apparatus. Characteristic of the latter is the hierarchy of self-regulating, equilibrating mechanisms, each of which controls its subunits but submits to regulation by a larger system. This set of systems provides the organism with stability, imperturbability.

Stated in another way, the organism's continuing stability depends on neural programs or plans — a set of genetic and experiential memory mechanisms — which organize the perceptions and behavior of the organism.[44] These programs consist of hierarchies of servomechanisms — feedback units that have been diagramed as nests of Test-Operate-Test-Exit units, or TOTE units (see Figure 1). The essential characteristics of the test mechanism is to sense incongruities, i.e., novelties; the essential characteristic of the operate mechanism is to effect changes that decrease the incongruity in the test mechanism. Input sufficiently incongruous can interrupt the ongoing plans; there is a temporary discontinuity, literally e-motion. (The word "emotion" comes from the Latin *emovere*, which means to be "out of" or "away from" motion.)

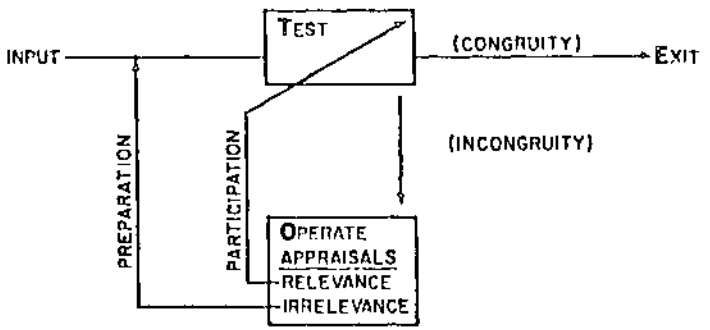

FIGURE 1 Diagram of the Test-Operate-Test-Exit (TOTE) sequence, showing the two forms (participatory and preparatory) of feedback involved in the emotional process. During participation, feedback influences the test phase to allow congruence with input; during preparation, feedback influences input to facilitate congruence with the test.

Results of experiments to be presented here suggest that the organization of emotions centers around two types of feedback which deal with the efferent control of input. They are termed *participatory* and *preparatory* processes. Participatory processes allow for the incorporation of input, and congruity is eventually achieved by alterations of the neural mechanism (or "model") against which the input is tested. On the other hand, preparatory processes alter input, thereby accomplishing congruity by manipulating the input which led to the incongruity. The system, perturbed by an incongruous input, is returned to the *status quo ante* by preparatory processes. External control is achieved by the development of new congruities, and preparation through internal control is accomplished by the return to previous congruities. Or, in terms of ongoing plans, external control develops and ramifies plans, and internal control conserves them.

This proposal differs in several respects from most currently held views on emotion. First, emphasis is on memory mechanisms, experientially derived as well as genetic, rather than on viscerally based drive processes. Second, the proposal takes as a baseline organized stability and its potential perturbation, rather than some arbitrary "level" of activation. Third, the proposal makes explicit the relation between emotion and motivation by linking both to an ongoing prebehavioral neural organization.

CURRENT NEUROLOGICAL THEORIES

There are two central themes in practically all of today's biological approaches to emotion: one deals with the relationship between visceral glandular reactions and emotion; the other deals with the quantitative relationship between neural excitation (activation) and emotion. As already noted, these relationships do not provide an adequate framework for understanding the complexities of emotional processes nor of the intricacies of the relevant neural apparatus.

The Visceral Theme

The visceral theme is reflected everywhere in our language: "he couldn't be expected to swallow that"; "she has no stomach for it"; "he broke her heart"; "the bastard has no guts"; "he sure is bilious

today." In fact, until A.D. 1800, the Galenic medical world subscribed to the notion that thoughts circulate in the ventricles of the brain, but emotions circulate in the vascular system. Gradually, medical and psychological science has become liberated from this view. But the release has been slow and guarded, partly because old theories take a long time to die and partly because the visceral view contains more than a modicum of truth. The most famous formulations that signal a retreat and liberation from the limiting aspects of the visceral concept are those of James and Lange, of Cannon and Bard, and of Papez and MacLean.

JAMES-LANGE THEORY James and Lange faced fully the knowledge accumulated in the nineteenth century about the functions of the circulatory and nervous systems. They offered the following proposition: when an organism's reaction to a situation involves visceral structures, the sensations aroused by visceral function are perceived as emotional feelings. This proposition provoked a good deal of experimentation. A summary taken from Cannon's "Critical Examination of the James-Lange Theory of Emotions" shows the theory's weaknesses.[15]

1 Total separation of the viscera from the central nervous system does not alter emotional behavior.
2 The same visceral changes occur in very different emotional states and in nonemotional states.
3 The viscera are relatively insensitive structures.
4 Visceral changes are too slow to be a source of emotional feeling.
5 Artificial induction of the visceral changes typical of strong emotions does not produce those emotions.

CANNON-BARD THEORY In place of the visceral theory, Cannon proposed a thalamic theory of emotions: emotional *feeling* results from stimulations of the dorsal thalamus; emotional *expression* results from the operation of hypothalamic structures. This theory was based on the observation that "sham" behavior could be elicited in decorticated and decerebrated cats except when thalamic structures also were ablated.[7] Further, a variety of expressive and visceral re-

sponses were obtained when the thalamus was electrically stimulated.[11] Finally, patients with unilateral lesions in the thalamic regions were described as sensing excessively what were to others ordinary cutaneous stimulations, e.g., a pinprick would elicit excruciating pain, warmth, intense delight.[27]

Probably more is known about the functions of these core portions of the brain than about any others. This is partly because the mechanisms are relatively "peripheral" in the sense that they can be shown to function as receptive mechanisms. Some of these structures contain receptive elements sensitive to a variety of physical and chemical agents that circulate in the blood stream and cerebrospinal fluid. In addition, the core mechanisms exert considerable direct control over the agent to which they are sensitive. This concept of control through feedback, which Cannon termed "homeostasis," has a great variety of biological and engineering applications.

Of equal importance is that the processes controlled are highly autonomous. Visceral and endocrinological regulation is performed via the sympathetic and parasympathetic portions of the autonomic nervous system. Experimental evidence was accumulated, especially by Hess,[28] to demonstrate the existence in the hypothalamic region of a trophotrophic, or energy-conserving, process that works primarily through parasympathetic peripheral divisions of the autonomic nervous system, and an ergotrophic, or mobilizing, system that works through the sympathetic division.

The balance between ergotrophic and trophotrophic is not static, of course. It could be tipped in one direction or the other; a temporary rebound, an "answering effect,"[23] could occur as the balance is restored. And indeed both processes can be activated simultaneously, so that in effect they work additively. Nor is this all. When such activation occurs, somatic as well as visceral musculature is involved.

An assumption that paralleled, if not actually guided, these studies was that an understanding of the organization of thalamically regulated processes would provide the key to an understanding of the organization of emotional processes. Once the thalamus and hypothalamus were identified as the neural substrate of emotions, this assumption followed logically.

But Lashley[37] tellingly criticized the evidence upon which this identity was assumed to rest. He pointed out that the type of disturbance on which the theory is based is as often seen to follow lesions elsewhere in the nervous system. "Hyperalgesia is not a result only of lesions within the thalamus but may arise from damage anywhere along the afferent path." He also raised the question whether "emotional disturbance" in the true sense ever occurs with thalamic lesions: "In no case was the affect referred to the source of emotional stimulation . . . but always to sensations of somatic reaction to the stimulus." He does agree that "In the hierarchy of motor centers we may recognize the thalamic region, especially the hypothalamus, as the region within which the complex patterns of expressive movements are elaborated. It does not follow from this, however, that the pathological phenomenon of hyper-excitability of emotional reactions are due solely to release from cortical inhibition or that the thalamic motor center for expressive movement contributes to the emotional experience." Clearly, the dissociation between expression and emotion, which is such a common clinical and experimental observation, can be leveled against *both* the James-Lange and the Cannon-Bard theories. Unfortunately, Lashley provided no alternative to the theories he so effectively depreciates.

PAPEZ-MACLEAN THEORY Recently the James-Lange and Cannon-Bard views have been superseded by one proposed by Papez[18] and elaborated by MacLean.[41] The earlier theories had been firmly based on the evidence that the hypothalamus and dorsal thalamus were at the *apex* of the hierarchy of control of visceral or autonomic function. With the development of modern techniques for electrical brain stimulation, viscera were shown to be under the surveillance of the cerebral cortex. One part of this cortex—the limbic portion of the forebrain—came into focus for special attention.[20] Papez suggested that the anatomical interconnections among limbic structures were ideally constituted to handle the long-lasting, intense aspects of experience that are usually associated with emotion. To this idea MacLean added the facts of the relationship between the limbic area and viscera, and suggested that here at last is *the* visceral brain—the seat of emotions.

The persuasive power of this suggestion is great: Galen, James and ange, Cannon and Bard, all are saved; visceral processes are the basis f emotion; an identifiable part of the brain is responsible for emoional control and experience because of its selective relations with iscera. James and Lange were wrong only in leaving out the brain; annon and Bard were wrong only in the part of the brain they had dentified with emotion; the limbic forebrain, not the thalamus, is the esponsible agent. The path from "emotions in the vascular system" o "emotions in the forebrain" had finally been completed, and each tep along the way freed us from preconceptions popularly current hen the step was taken.

Despite its persuasiveness and continuing popularity, there are ome important criticisms of the visceral brain theory of emotions. ust as the theory gains in power from its implicit acceptance of the ames-Lange and the Cannon-Bard views, so it falls heir to the criticisms leveled against the earlier theories. It has been demonstrated exerimentally[76] that other parts of the cerebral mantle, when electrically stimulated, also elicit a visceral response. Emotional changes re observed to accompany lesions in parts of the forebrain other than he limbic areas. Further, ablation and stimulation of limbic structures influence problem-solving (cognitive) behavior in selective ways hat cannot be attributed to changes in emotions (see below). In man, n fact, specific "memory" deficiency follows limbic lesions, while hanges in "emotion" cannot be ascertained. Obviously, the Papez-Maclean theory, like its predecessors, has only a part of the problem n hand.

The Activation Theme

As one turns from the visceral to the activation theories of emotion, one can again distinguish between peripheral and central subtheories. Here, however, the argument has not been so sharp. Peripheralists have gladly accepted the diffuse, non-specific, reticular activating system as the central locus upon which and from which peripheral excitation focuses. Centralists, in turn, have been as concerned with the peripheral as with the central effects of adrenergic and cholinergic substances (e.g., Arnold[4]). Activation theory can be said, on the

whole, to be less specific, less controversial, and considerably more factually oriented than visceral theories.[40] For example, a classical visceral theorist would say that a certain amount of adreno-cortical hormone circulating in the blood stream is correlated with a specific pattern of peripheral and central neural response (in hypothalamus, or visceral brain), which, in turn, corresponds to one or another emotional experience or expression. An activation theorist states merely that a correlation exists between the amount of hormone, amount of neural excitation, and amount of emotional arousal. Considerable evidence can be marshaled in favor of activation theory.

But common observation and introspection caution that something may be missing. For example, weeping is not *just* more laughing; fear is not *just* more love—although there is some truth in the notion of quantitative continuity in these processes.

A NEW APPROACH

"Expectation" and Configuration in Neural Activation

A part of the difficulty comes from the view of activation as an elementary process opposed only by another elementary process, inhibition. True, activation can be viewed as an indicator of behavioral arousal—a temporary state of disequilibrium, a perturbation of patterns of organism-environment interactions. Also, disequilibration is often sudden, explosive, and suggests agitation. But this does not necessarily mean that neural impulse transmission is facilitated: rather, a different state of organization or disorganization may suddenly have materialized. This difference is expressed as a difference in configuration, and not necessarily as a difference in the amount of neural activity. For instance, heart rate may be slowed, cortical rhythms desynchronized, peripheral blood flow diminished, but cerebral blood flow augmented. Cerebral activation, in this context, is an indicator of a configurational incongruity between input arrival patterns and established ongoing neural events.

This view of activation as an indicator of configurational change implies that the organism is fitted with a mechanism that provides a stable baseline from which such change can take off. This baseline is established by the process of habituation of the orienting reaction. Ex-

erimental evidence has accumulated in the past several years (e.g., okolov[68]) to show that habituation of orienting is not caused by a rogressive raising of threshold to input, but to the formation of a neuronal model"—a neuronal configuration against which subsequent inputs to the organism are matched. In essence, such neuronal onfigurations form the sum of an organism's expectancies. The evilence runs like this: a person is subjected to an irregular repetition f a sound stimulus of constant intensity, frequency, and duration. nitially, the person shows a set of physiological and behavioral rections, which together form the orienting response. Among these rections is cerebral activation, i.e., a desynchronization of the electrical hythms recorded from the brain. As the repetition of the sound stimlus proceeds, less and less orienting takes place. For many years a imple rise in threshold to input was thought to cause such habituaion. But dishabituation, i.e., a recrudescence of the orienting reponses, occurs when the intensity of the sound stimulus is *decreased*, r if the duration of the sound is shortened. In this latter situation, he orienting reaction occurs as a result of the "unexpected" silence—hat is, at the *off*set of the stimulation.

Each interaction between environment and organism involves at east two components: 1) discrete interaction by way of the brain's ensory-mode, specific, classical projection systems, and its core homeostats; 2) a "non-specific," relatively diffuse, interaction by way of reticular and related formations. These "non-specific" systems act as a bias on the specific reactions: the set-point or value toward which a specific interaction tends to stabilize is set by the non-specific activity. By the nature of its receptor anatomy and diffuse afferent organization, visceral feedback constitutes a major source of input to this biasing mechanism; it is an input that can do much to determine setpoint. In addition, visceral and autonomic events are repetitiously redundant in the history of the organism. They vary recurrently, leading to stable habituations; this is in contrast to external changes that vary from occasion to occasion. Habituation to visceral and autonomic activity, therefore, makes up a large share, although by no means all, of the stable baseline from which the organism's reactions can take off.

Whenever the reaction to incongruous input is sufficient to disturb this baseline, the orienting reaction will include the dishabituation of visceral and autonomic activities. Such dishabituation may be subjectively felt as a mismatch between expected and actual heart rate, sweating, "butterflies," and so on. The sensing of such discrepancies is the basis for the visceral theories of emotion. The awareness of visceral-autonomic activities is thus often an indicator that a reaction to incongruous input has occurred, but it need not reflect the organization of the emotional process called forth. Support for this conception comes from the work of Lacey, et al.,[30] and of Barratt,[8,9] who have distinguished between two classes of variables in their studies of the visceral-autonomic system. One class is derived from measures of the variability of steady-state autonomic activity along a stabile-labile dimension; this dimension deals with the organism's *responsivity*—its predisposition to impulsive reaction to input. The other class of variables stems from measures of the response patterns of visceral-autonomic function.

If cerebral activation is conceived as a change in the state of organization of neural patterns related to the configurational incongruity between input and established neural activity, what then is its converse? As already indicated, over-all neuronal facilitation or inhibition are not involved. Rather, some indicator of congruity, of unperturbed, smoothly progressing neuronal activity must be sought. This indicator, at present, is found in the patterns of electrical activity recorded from the central nervous system. There is considerable evidence[1,88,89] that the slow graded activity of neural tissue, rather than the over-all inhibition or facilitation of nerve impulse transmission per se, is involved in the generation of such electrical patterns. The assumption is that the graded electrical activity recorded from brain reflects the relative stability of the neural system. Such stability would admit increments of change, provided these did not disrupt the system. Nor is it implied that incongruity and therefore activation are necessarily initiated by input. An input that ordinarily would be processed smoothly may perturb the system if that system is already unstable; or an internal change in the organism may initiate incongruity where a match had previously existed. The configuration of

activation of the nervous system thus can predispose the organism toward perturbability or imperturbability.

A considerable body of evidence has recently accrued about the neurophysiological and biochemical mechanisms that regulate these predispositions. As already noted, the non-specific diffuse neural systems are primarily involved in setting the bias toward which more specific organism–environment interactions tend to stabilize. These diffuse systems are largely made up of fairly short, fine fibers with many branches. Such neuronal organizations are especially sensitive to the chemical influences in which they are immersed. A potent set of such chemical influences are the catecholamines, and they have been shown to be selectively present in the diffuse systems.[80] Further, these brain amines have been shown to be the important locus of action of the various pharmacological tranquilizers and energizers that have been so successful an adjunct in altering maladaptive emotional reaction.

Motivation and Appetite: Finickiness and Emotion

The visceral theme can also be reappraised in the light of new data. Hypothalamic mechanisms have been related both to emotion and to motivation, but the relation between these categories has never been made explicit. Hypothalamic lesions and stimulations have resulted in massive, explosive—although often directed—muscular and endocrine discharges, which are interpreted by other organisms[16,17] and by observers[15] as expressions of emotion. Hypothalamic mechanisms for the regulation of food, water, and other appetitive behavior have also been delineated in detail.[74]

However, these experiments on motivation have produced one major paradox: when lesions are made in the region of the ventromedial nucleus of the hypothalamus, rats will eat considerably more than do their controls and will become obese. But this is not all. Although rats so lesioned will eat a great deal when food is readily available, they were found to work less for food when some obstacle interfered.[46] In addition, it was found that the more palatable the food, the more the lesioned subject would eat. This gave rise to the notion that the lesioned animals were more finicky than the controls. Further, recent

experimental results obtained by Krasne[35] and by Grossman[25] show that electrical stimulation of the ventromedial nucleus stops both food and water intake in the deprived rats. Yet, lesions of the ventromedial nucleus leave the rats "unmotivated" to work either for water or for food. Grossman resolves this paradox by making the suggestion that the neurobehavioral results occur due to *alterations in affect rather than appetite* when the ventromedial nucleus is manipulated: lesioned animals show an *exaggerated sensitivity to all sorts of stimulation.*

He notes that a discrepancy remains, however. Neurophysiological data show that the ventromedial and lateral hypothalamic regions form a couplet: the lateral portion serves as a feeding or "go" mechanism (which, when ablated, will produce rats that tend to starve); the ventromedial portion is a satiety, or "stop" mechanism. This remaining discrepancy is resolved if "stop" mechanisms are more generally conceived to reflect e-motion, processes ordinarily involved in taking the organism "out of motion" and thus relegating the term motivation to those processes which ordinarily result in behavior that carries forward a plan or program.

A NEUROLOGICAL MODEL AND SOME DATA

These are not the only data relevant to the problem. As already noted, autonomically regulated appetites and "tastes" are not the only concerns of a theory of emotion. Perhaps the simplest way in which to proceed is to present a neuropsychological model of the emotional process and then turn to the experiments that generated the model.

The model focuses on the interaction of two forms of afferent inhibition. The collateral type acts to *accentuate* the difference between active and less active sites; the activity of a cell inhibits the activity of its neighbors. Self-inhibition tends to *equalize* such differences; the activity of a cell inhibits its own activity through negative feedback. Any patterned change in the system will be enhanced by collateral inhibition; self inhibition works against change, tending to stabilize the status quo. Collateral inhibition is thus conceived to be a labile mechanism sensitive to input and concurrent activity. Self-inhibition,

on the other hand, works more slowly, countering the rapid fluctuations in the patterns of neural activity that would otherwise occur, and stabilizing more slowly occurring changes once they have taken place. Both habituation and orienting are conceived to be dependent on the occurrence of inhibitory processes in the afferent channels of the nervous system, and both types are ubiquitous. The model states that orienting is a function of collateral inhibition and habituation a function of self-inhibition.

Neurophysiological data

The model regards inhibitory neural processes—inhibition defined as a reduction in the excitation of a neural unit—and distinguishes two major types of neural inhibition (Figure 2). Lateral, or

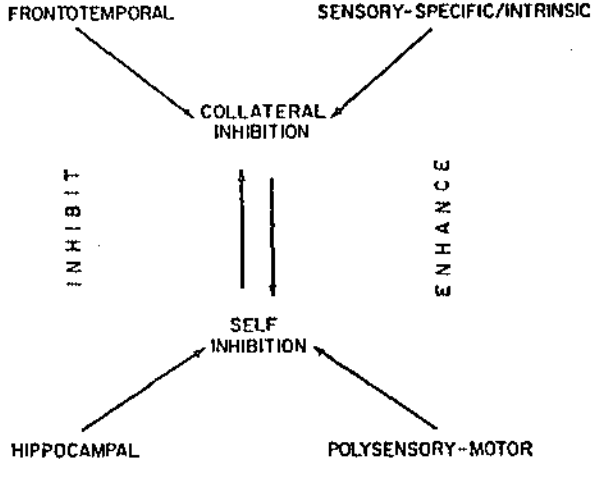

FIGURE 2 Diagram of the model of cortical control over afferent inhibitory processes.

surround, inhibition operates through collateral processes distributed among neurons or via amacrine-like cells, and is well-demonstrated in the visual,[26] auditory,[12] and somatic[47] systems, both at peripheral and central stations. This type of afferent neural interaction corresponds

to Pavlov's "external" inhibition. The second type of afferent inhibition is self inhibition.[5,13,14] It takes two forms, presynaptic and postsynaptic. Both result in the regulation of afferent activity via negative feedback. In the case of postsynaptic self-inhibition, interneurons of the Renshaw type are assumed to function, via recurrent inhibitory fibers, as dampers that control the excitability of active neurons as a consequence of their own activity.

The chief concern of the model is with efferent control exerted over this interaction. This control is primarily cerebrofugal. Mechanisms that enhance and inhibit afferent inhibition are assumed to converge upon the afferent pathways. Because of this site of operation, a fourfold mechanism of efferent or cerebrofugal control should in theory be distinguishable: 1) enhancement of collateral inhibition; 2) enhancement of self-inhibition; 3) inhibition of collateral inhibition; and 4) inhibition of self-inhibition.

There is available evidence on corticofugal control over both presynaptic and postsynaptic forms of self-inhibition. Repetitive stimulation of a variety of sensory-motor points on the lateral cortex influences presynaptic inhibition at the spinal level.[2,8,32] And the effect of hippocampal stimulation on visual evoked activity has also been recorded.[24]

The evidence for efferent control of collateral inhibition has been gathered in my own laboratory, in collaboration with Dr. D. N. Spinelli.[72] Experiments were performed on fully awake monkeys implanted with small bipolar electrodes and a device that allows chronic repetitive stimulation of one of the electrode sites. The monkeys were presented with pairs of flashes and the interflash interval was varied from 25 to 200 milliseconds. Electrical responses were recorded from the striate cortex, and the amplitude of the responses was measured. A comparison of the amplitude of the second to the first response of each pair was expressed and plotted as a function. Underlying the interpretation of this function is the assumption that when the amplitude of the second pair of responses approximates that of the first, the responding cells have fully recovered their excitability. In populations of cells such as those from which these records are made, the per cent diminution of amplitude of the second response is used as an

index of recovery of the total population of cells—the smaller the percentage, the fewer the number of recovered cells in the system.

Chronic stimulation (8 to 10 per second) of several cerebral sites alters this recovery function. When the inferotemporal cortex of monkeys is stimulated, recovery is delayed. Stimulation from control sites (precentral and parietal) has no such effect (Figures 3, 4, 5). Nor does the stimulation of inferotemporal cortex alter auditory recovery functions. These, however, can be changed by manipulations of the insular-temporal cortex, as was shown in a parallel experiment performed on cats. Here the crucial cortex was removed and recovery functions obtained on responses recorded from the cochlear nucleus.[19] Removal of insular-temporal cortex shortens recovery in the auditory system.

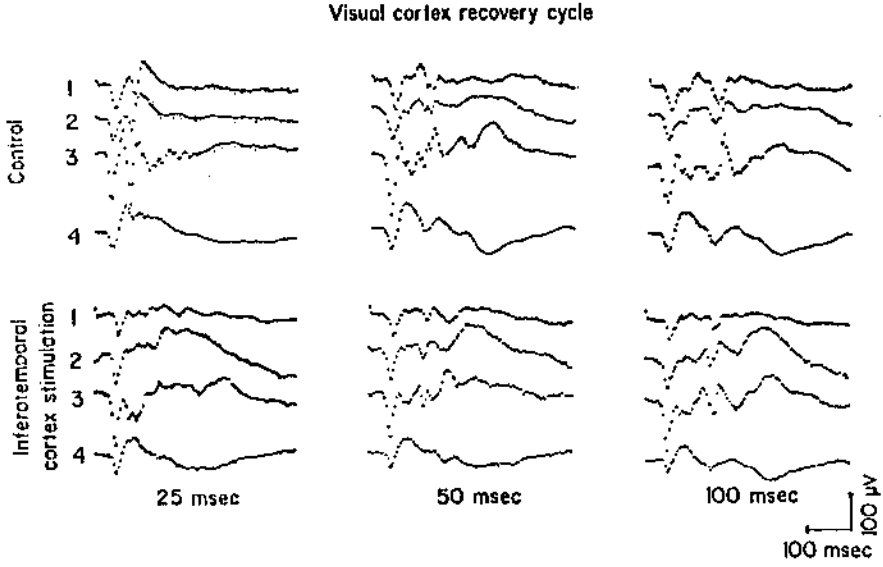

FIGURE 3 Representative record of the change produced in visual evoked responses by chronic stimulation of the inferotemporal cortex. Upper set of records was taken before stimulation; lower set, during stimulation. All traces were recorded from the visual cortex. In the first column are responses produced by a pair of flashes separated by 25 msec. Flash separation is 50 msec. in the second column and 100 msec. in the third.

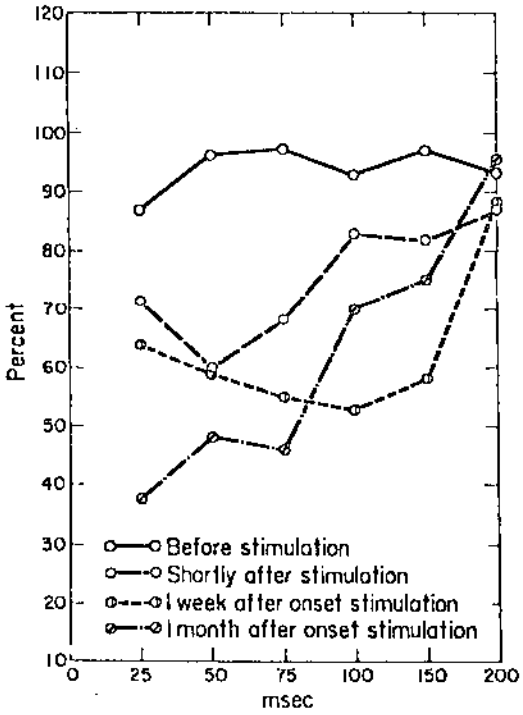

FIGURE 4 A plot of the recovery functions obtained in one monkey before and during chronic stimulation of the inferotemporal (IT) cortex.

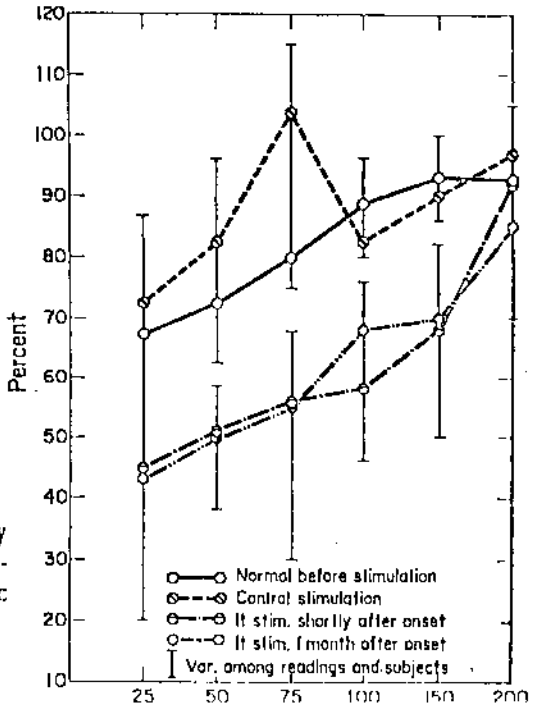

FIGURE 5 A plot of the recovery functions obtained in five monkeys before and during chronic cortical stimulation.

A great many neurobehavioral experiments have shown the importance of these isocortical temporal lobe areas (and no others) to visual and to auditory discrimination. These studies are reviewed elsewhere.[51,59] What concerns us here is that a corticofugal, efferent mechanism is demonstrated, and that this mechanism alters the rapidity with which cells in the visual and auditory afferent systems recover their excitability. Further, since stimulation delays and ablation speeds up recovery, the inference is that the normally afferent inhibitory processes which delay recovery are enhanced by the ordinary operation of these temporal lobe isocortical areas.

But the opposite effect—inhibition of afferent inhibition—can also be obtained when cerebral tissue is chronically stimulated (Figures 6, 7, 8). In these experiments the cortex of the frontal lobe and

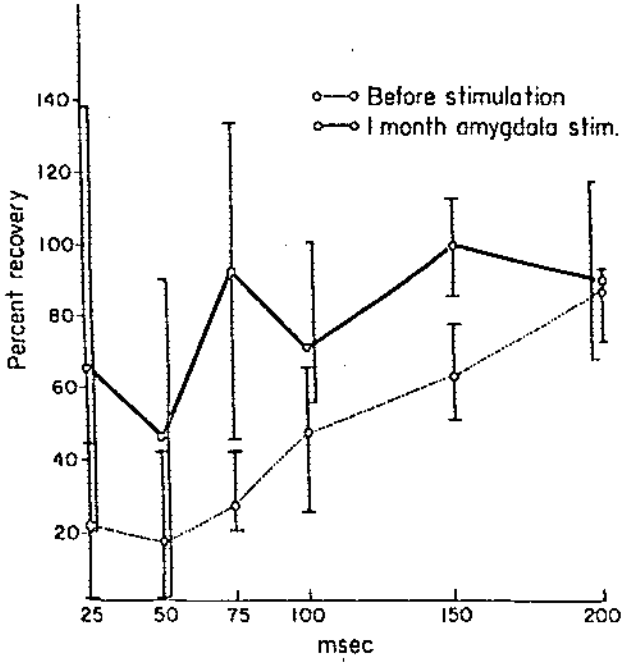

FIGURE 6 The effect on recovery function of chronic stimulation of the amygdaloid complex. The dotted line indicates the function before, the solid line after, one month of stimulation. Bars perpendicular to the curves show variability among subjects. Each curve is based on the average response of four subjects.

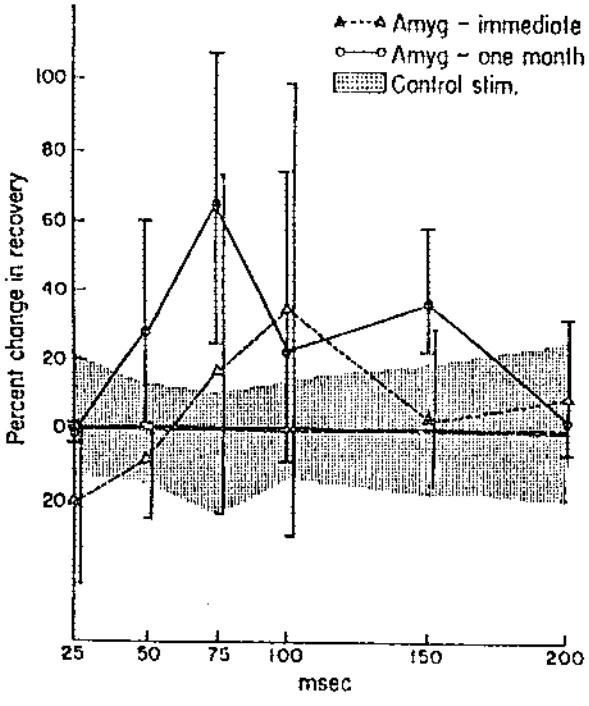

FIGURE 7 This figure represents the same data as those represented in Figure 6. However, here percentage change in recovery is plotted. Shaded area indicates range of recovery for *un*stimulated subjects.

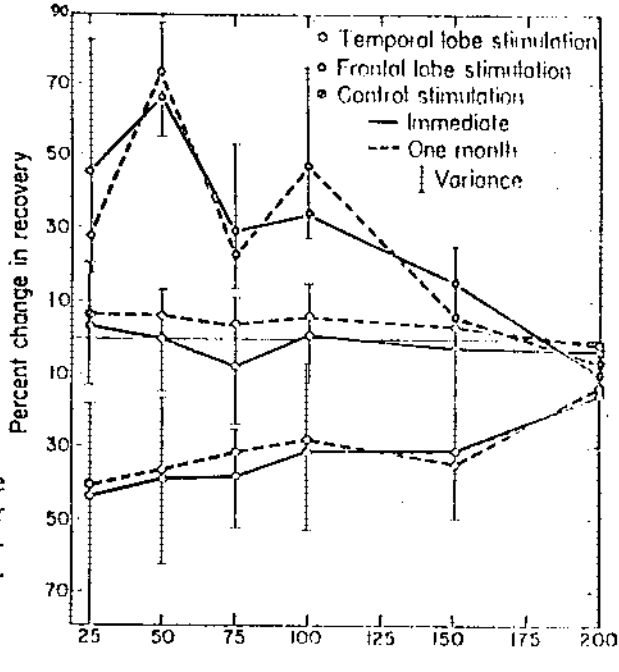

FIGURE 8 This figure plots the percentage change in recovery for all subjects in the various experiments. It is thus a summary statement of the findings.

the cortical nucleus of the amygdala were chronically stimulated, and recovery of cells in the visual system were shown to be speeded. This result has suggested that the frontal and anterior medio-basal portions of the forebrain function as efferent systems that inhibit afferent inhibitory processes.[73]

The antagonistic effect of these two efferent control systems is best illustrated by data obtained at the unit level (Figures 9, 10). Unit recordings were made from the striate cortex of flaxedilized cats to whom flashes of light were presented. Note that the silent period of a cell can be lengthened by concurrent inferotemporal stimulation. Note also that concurrent frontal stimulation can shorten this silent period. Finally, note the unit whose silent period is lengthened by inferotemporal, and shortened by frontal, stimulation.

In summary, the model is based on neurophysiological evidence of two forms of afferent inhibition: collateral and self. The reciprocal interaction of these two forms is spelled out. Data are presented indicating that afferent inhibition is under efferent corticofugal control. Further, such efferent control is shown to be balanced: both efferent enhancement and efferent inhibition of afferent inhibition were found to converge and to regulate the activity of a single system and even a single cell. The major assumption of the model is that separate forebrain systems can be found to regulate afferent neural collateral and self-inhibition.

Neurobehavioral Data

One of the consequences of this model of efferent control over afferent inhibition is a plausible neural explanation of the orienting reaction and its habituation. As already noted, a series of studies has shown: 1) that orienting can be identified by a specific pattern of behavioral and physiological indexes; and 2) that habituation of this set of indexes is not a function of a raised neural threshold to input, but to change in some neural configuration against which input is matched.[68] The reasonable suggestion can be made that habituation reflects increments of self-inhibition and that the orienting reaction manifests an override on habituation, the override taking place whenever collateral inhibition is enhanced. At least preliminary evi-

dence at the neurophysiological level is congruent with this suggestion.[69,70,71] The following data at the neurobehavioral level can also be interpreted as in accord with the model.

The data deal with functions of the limbic forebrain.[57] Thus far, these functions have been characterized either in terms of their influence on response regulation,[43,44,53] on memory,[46,49] or on emotion.[41] In and of themselves, however, these characterizations have so far failed to provide the key to the essential nature of the limbic contribution to behavior and to psychological experience.

Bilateral amygdalectomy interferes drastically with the orienting reaction as gauged by the galvanic skin response. However, the behaviorial effect of this interference is not simple (Figures 11, 12). In a variety of discrimination learning tasks, some of which amygdalectomized monkeys found more difficult than did their controls, a behavioral measure of orienting was taken (by Dr. Patrick Bateson, an ethologist, during his post-doctoral training). This measure consisted of noting the flick of monkeys' ears during the time the cues were presented. Normal monkeys show this ear flick while they are learning; once a task has been mastered the ear response does not occur. Amygdalectomized monkeys show a greater number of such ear flicks during learning, especially in those tasks in which they have difficulty.

These results[9,10,32,84] led to the idea that orienting was made up of two components—one an alerting reaction indicated by the ear flick, the other a focusing function that allowed registration of the event producing the alerting. It is this second stage that involves the amygdala and is signaled by the appearance of a galvanic skin response.

The two phases of orienting fit the model presented. Alerting can be explained as a consequence of initial disinhibition of collateral inhibition. In the absence of a secondary controlling mechanism, this reaction would overcome the stabilizing mechanism provided by self-inhibition. Events would continually be noticed, but *adjustment* of the stabilizing mechanism (habituation) would be precluded. This is believed to be the case after amygdalectomy. By contrast, in normal subjects collateral inhibition is, in turn, inhibited by the operation of the amygdaloid mechanism. This provides the reaction with a stop mechanism that increases the likelihood that its specific configuration

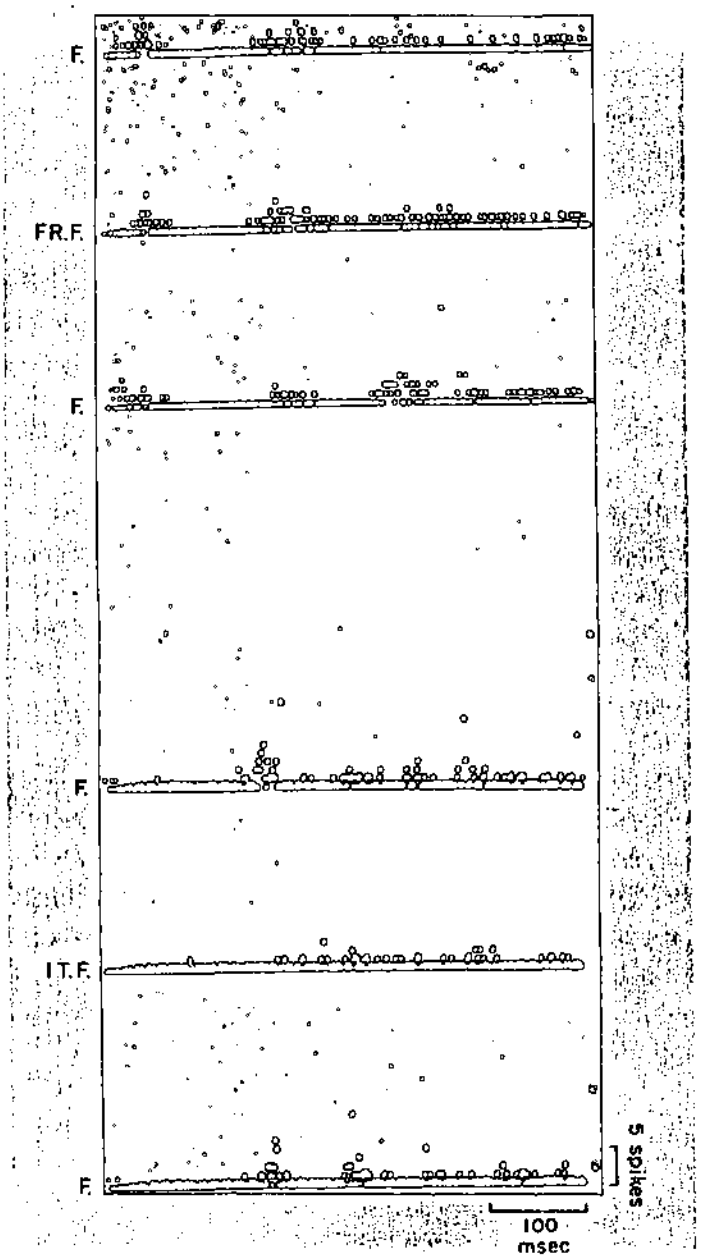

FIGURE 9 This figure is made up of photographs of a pulse histogram derived from a computer readout for average transients. Each vertical segment represents the number of impulses recorded from a neural unit during a 1.25 msec. period. The upper three traces show the effects of concurrent stimulation of the frontal cortex, the bottom three traces the effects of concurrent stimulation of the temporal cortex of cats on the unit activity evoked in the triate cortex to repeated flashes. The first and last trace in each

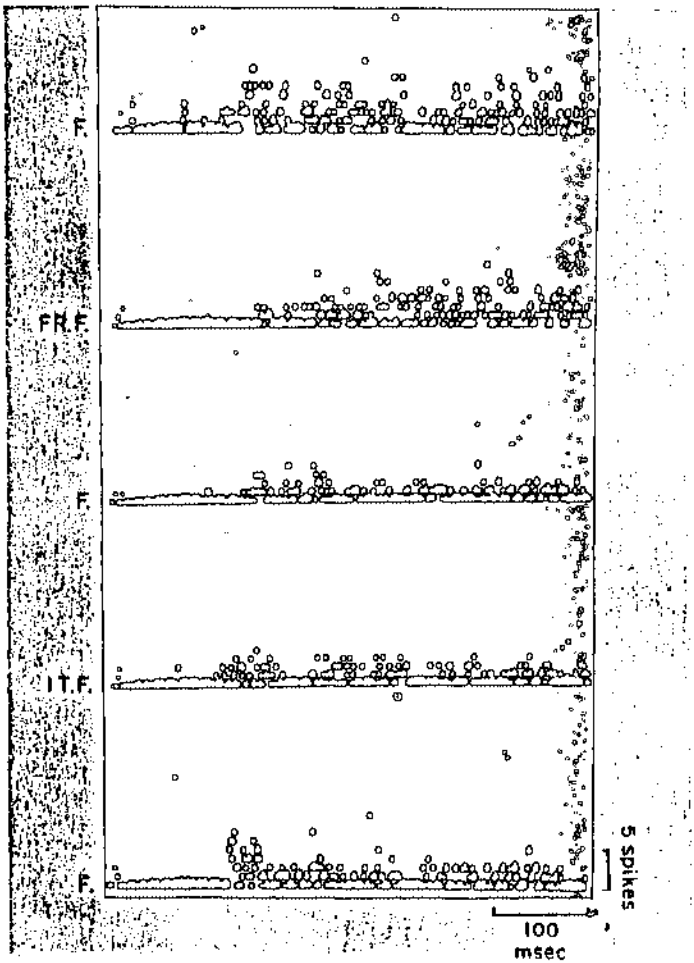

FIGURE 10 A pulse histogram obtained in the same fashion as that reproduced in Figure 9. Here the influence of concurrent frontal (second trace) and concurrent temporal (fourth trace) cortical stimulation on the flash-evoked activity of the same single unit are shown. Note that the first silent period is lengthened by concurrent frontal, and shortened by concurrent temporal, cortex stimulation.

FIGURE 9 *continued*

triad are controls; the middle traces were recorded during concurrent stimulation. Note that the first silent period is lengthened by concurrent frontal, and shortened by concurrent temporal, cortex stimulation. F, flash; FRF, frontal stimulation with flash; ITF, inferotemporal stimulation with flash.

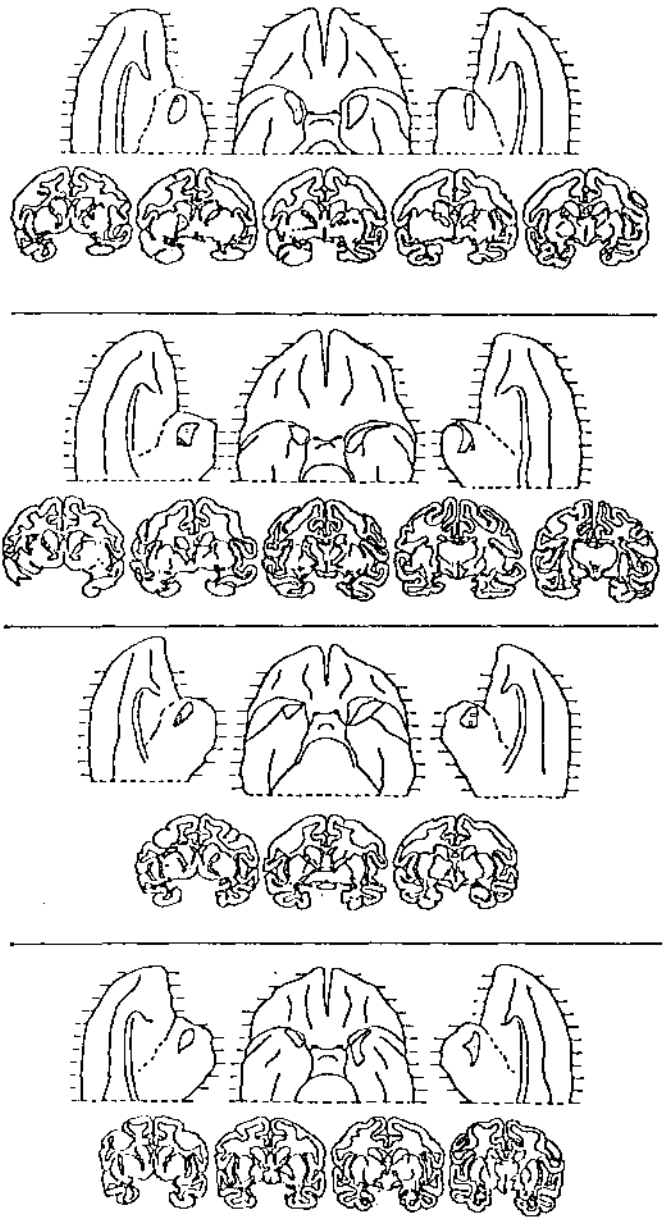

FIGURE 11 Reconstructions of the bilateral lesions of the amygdaloid complex. Black areas denote the lesion.

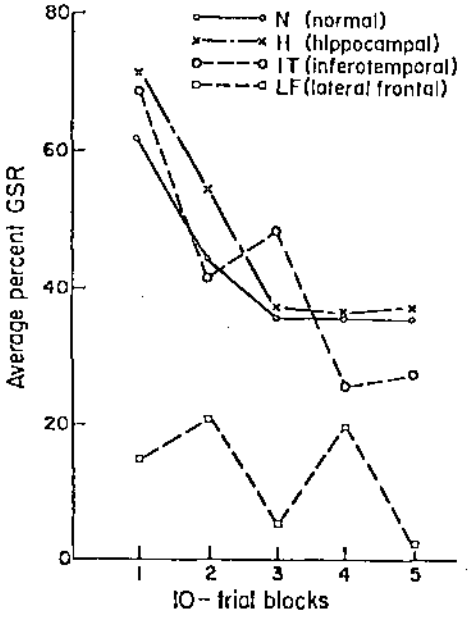

FIGURE 12 Curves of the percentage of galvanic skin response (GSR) to the first fifty presentations of the original stimulus for the normal and three experimental groups.

will be stabilized, i.e., registered. A difficulty in registering events could show up behaviorally in a variety of ways. One of them certainly would be in direct measures of habituation. Short-term measures should show an increased speed of habituation. On the other hand, longer-term measures should show that such habituation had failed to incorporate the orienting experience. This is exactly what has been found.[67] Another consequence of difficulty in registration would be the relative inefficiency of reinforcement. And, indeed, a series of experiments has shown that changing the deprivation conditions,[64] or the size of the reward,[65] or the distribution of its occurrence,[66] has considerably less effect on amygdalectomized monkeys than on their controls (Figures 13, 14, 15).

Dr. Robert Douglas, another postdoctoral student in my laboratory, first formulated in precise behavioral terms a proposal that I have taken the liberty of incorporating into my model. He suggested

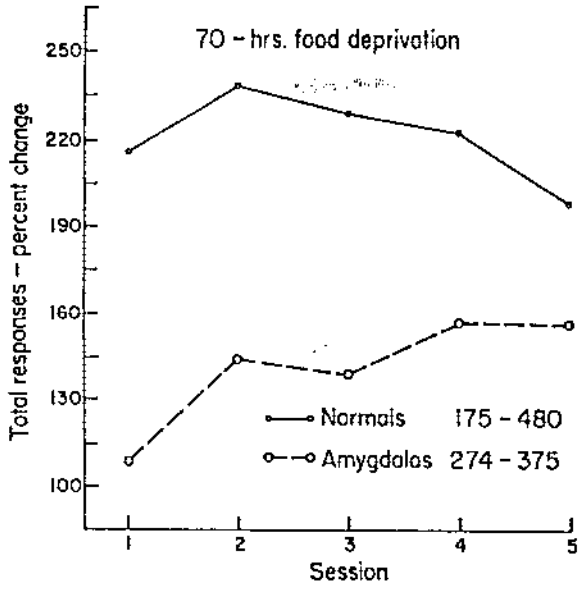

FIGURE 13 Mean percentage changes in total responses of test sessions that followed prolonged deprivation of food. The values in the legend refer to the range of total responses for the three preceding control sessions on which the percentage changes are based.

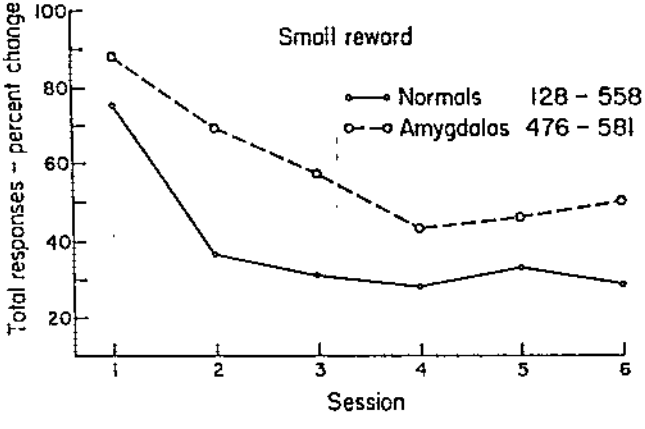

FIGURE 14 Mean percentage changes in total responses in test sessions with the small reward. (The values in this legend and in that of Figure 15 refer to the range of total responses for the three preceding large-reward sessions on which the percentage changes are based; group differences are not significant statistically.)

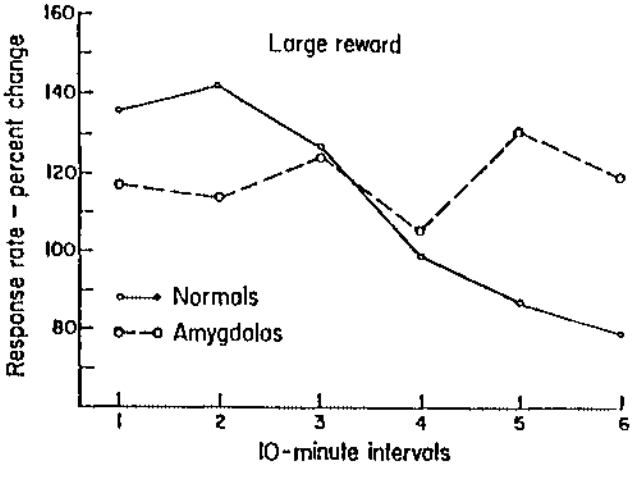

FIGURE 15 Mean percentage changes in total responses within test sessions with the large reward.

that while the amygdaloid system operates as a reinforce-register mechanism, the hippocampal formation serves as a mechanism necessary to evaluate error. He devised several ingenious experiments to test hypotheses derived from the suggestion. I shall present three of these. (For a full report, see Douglas and Pribram.[21]) All were performed in an automated discrimination apparatus, which allowed programming of tasks by a special-purpose computer that could also be used for data reduction and analysis.[58]

Dr. Douglas modified a standard behavioral testing procedure to his purpose (Figures 16, 17, 18). Ordinarily, one cue is rewarded 100 per cent of the time and the other is never rewarded. However, in the modification, called probability matching, subjects are trained to discriminate between two cues. One cue is rewarded some percentage less than 100 per cent — say 70 per cent — while the other is rewarded on the remaining occasions — in this instance, 30 per cent of the time. This task is, of course, more difficult than ordinary discrimination, and is more interesting, because different organisms demonstrate different strategies in solving the problem. Douglas trained monkeys (bilaterally amygdalectomized, hippocampectomized, and sham-operated controls) in such a probability matching situation and then paired a

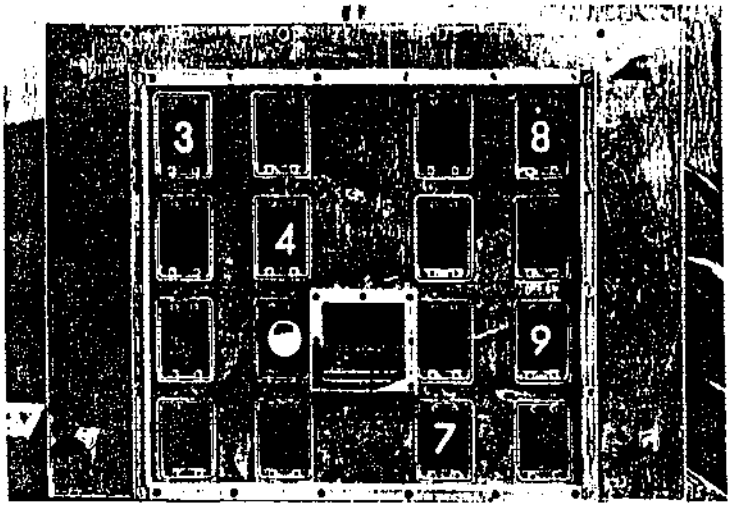

FIGURE 16 Display panel of the automated discrimination apparatus. Note 16 clear, hinged windows through which patterns can be displayed, and the central tray attached to feeder mechanism.

novel cue with either the most- or least-rewarded of the familiar cues. His results were striking.

First, monkeys with hippocampal lesions learned the probability task more slowly than did the other groups (Figure 19). This slower learning is interpreted as consonant with an impaired error-evaluate system in the hippocampectomized monkeys.

Second, monkeys with hippocampectomies, when compared with the other groups, chose the familiar cue more often when it was paired with a novel cue, irrespective of whether the familiar cue had been reinforced in 70 per cent or in 30 per cent of the trials (Figure 20). The choice of the familiar is also consonant with an intact reinforce-register function and an impaired error-evaluate mechanism.

Third, the cues used in the probability matching task were again presented, this time without reinforcement. As could be predicted, control subjects quickly shifted their responses away from the previously rewarded cues, because these responses were now erroneous. And, again, hippocampally ablated monkeys came to the support of the theory by failing to shift their responses on the basis of error (Figure 21).

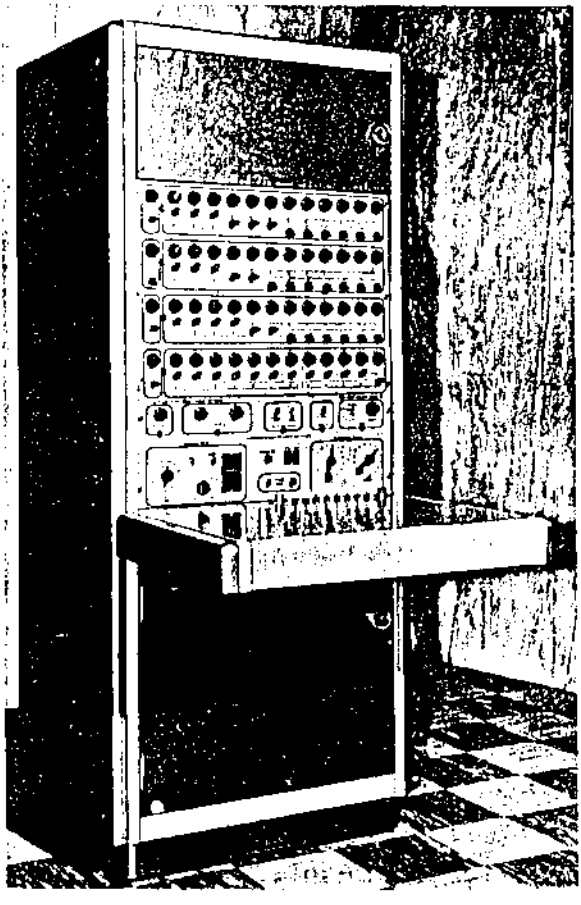

FIGURE 17 Control console and special-purpose computer for the automated discrimination apparatus. This allows programing of tasks as well as data reduction and analysis.

As already noted, the behavioral process invoked to explain these results is an error-evaluate mechanism. On the basis of the model and data presented, the hippocampus is suggested as providing this mechanism. By inhibiting self-inhibition, the erroneous experience is allowed to register. In the absence of the hippocampus, the stabilizing effect of self-inhibiton is assumed to be sufficiently strong to overcome the registration of nuances: the system of afferent inhibitory processes tends to revert to the *status quo ante*. This hyperstability

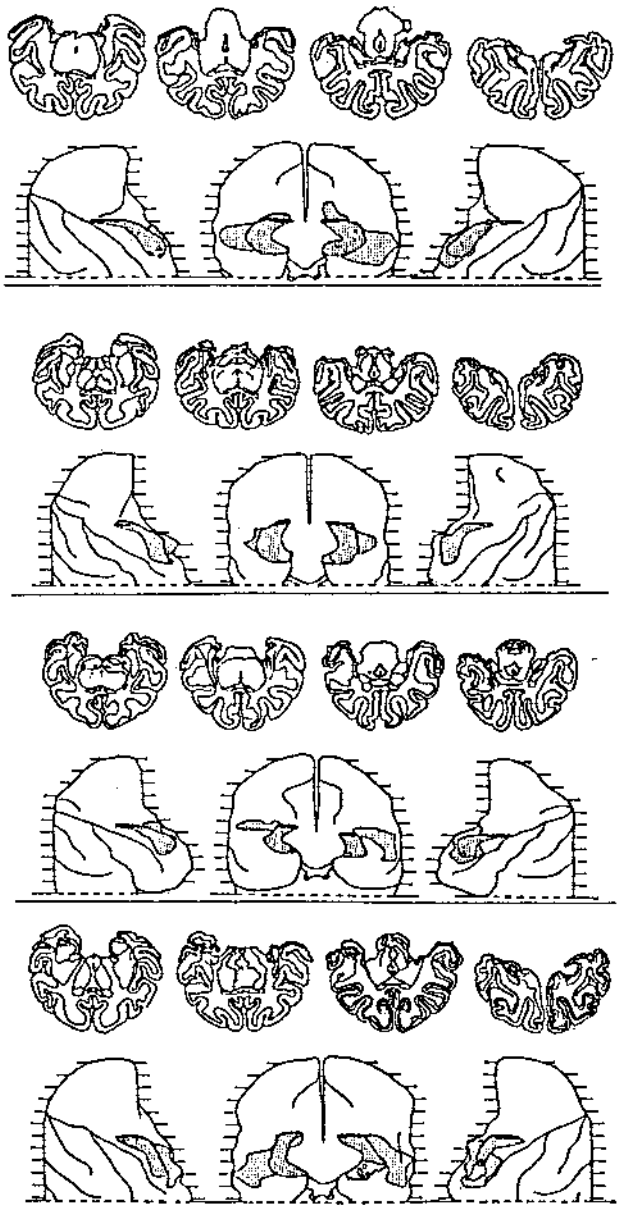

FIGURE 18 Reconstructions of the bilateral lesions of the hippocampus. Dashed areas denote the lesion, black areas denote sparing. Dotted areas show the overlying cortex removed in the approach. Heavy lines on the cross-sections show the extent of the lesion on the ventral surface.

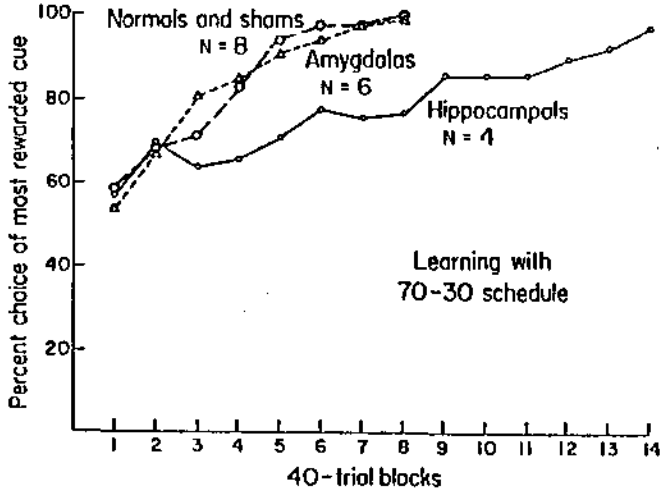

FIGURE 19 Per cent choice of most-rewarded cue in probability task involving learning with a 70 to 30 per cent reward schedule.

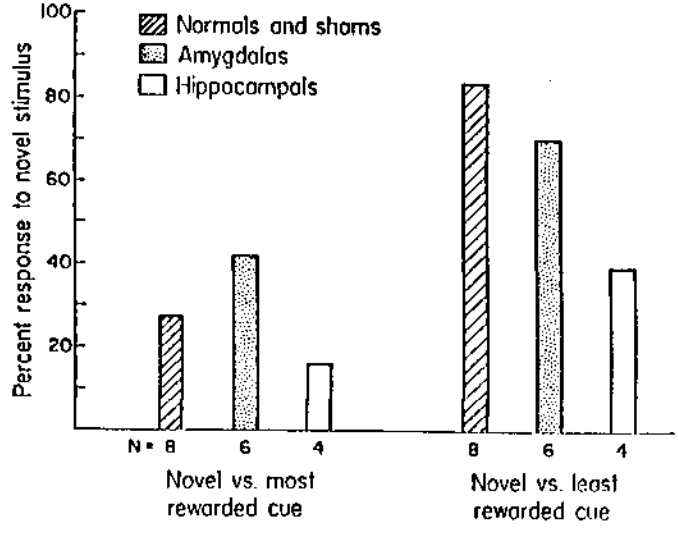

FIGURE 20 Per cent response to novel stimulus in groups of novel versus most-rewarded cue, compared with groups of novel versus least-rewarded cue.

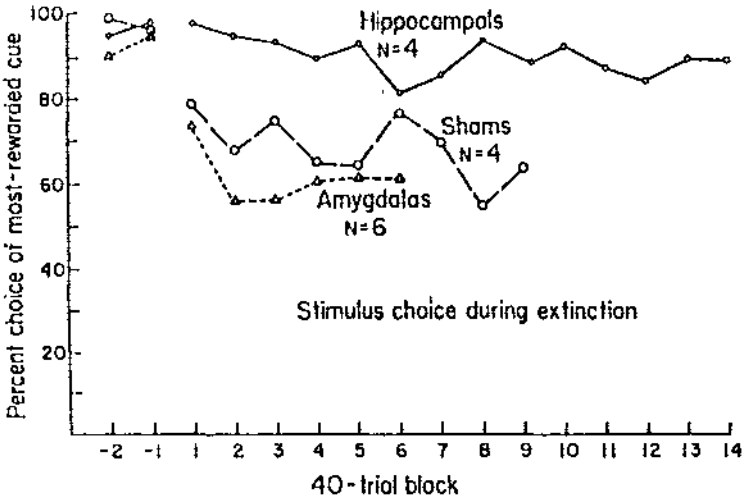

FIGURE 21 Per cent choice of most-rewarded cue in 50-trial block, where cues used in the probability matching task were again presented, this time without reinforcement.

is overcome only if the orienting events are overwhelming or if they recur regularly. Probabilistic occurrences, such as errors, fail to "get through." According to this view, short-term habituation should be slowed by hippocampectomy and registration limited to regularly recurring events. There is evidence in support of both these statements.[20,31,61]

In summary, collateral and self-afferent inhibition are bucked against one another, forming a primary couplet of neural inhibition within afferent channels. Four forebrain mechanisms are assumed to provide efferent control on this primary couplet (see Figure 2). Two of these — frontolimbic and sensory-specific-intrinsic (which includes the inferotemporal cortex) — work their influence by regulating collateral inhibition; two others — hippocampal and polysensory-motor — regulate self-inhibition. The sensory-specific-intrinsic and polysensory-motor "association" cortical systems exert their control by enhancing, while the frontotemporal and hippocampal systems exert control by inhibiting afferent neural inhibition.

Perturbation and the Control of Input

The significance of this demonstration of cerebral control over its own input is manifold. That this control shows two opposing tendencies is of direct relevance to the problem of emotion. One tendency accentuates orienting reactions and thus the perturbations of the system initiated by input to it. The other tendency reinforces the habituated baseline, i.e., the prior neural activity, by de-emphasizing these perturbations. In searching for adjectives for these two tendencies, the following, mentioned earlier, were deemed appropriate: *preparatory*, or better, *pre-repairatory*, and *participatory*. A preparatory process is one that prepares the organism for further interaction by repairing its perturbed system to its previous stability. A participatory process utilizes perturbation to adapt the system to the current input. Both processes are effected through feedbacks, as indicated in Figure 1. Preparatory operations are conservative and even defensive; they serve to deal with input by de-emphasis and elimination; participatory operations enhance the effect of input and so serve to increase the likelihood that the system itself will be changed.

Preparatory and participatory processes lead to different types of stability. Preparation tends to lead the organism toward relatively lasting (prospective) stability by recourse to an earlier (retrospective) organization; this type of stability is achieved through what can be termed *internal* control. Participation tolerates the temporary instabilities produced by incongruities by achieving reciprocal constancies with aspects of the environment, thus "realizing" the here-and-now (*external* control). Preparatory processes re-establish continuity at the cost of simplification. Participatory processes tolerate transience to gain flexibility through a more complex organization.

In terms of information measurement theory, these emotional processes effect a subtle balance between maximum redundancy (through preparation) and maximum information density (through participation).[62,76]

As discussed elsewhere,[53,54] the achievement of external control is

conceived, through the accommodation of past experience to current input, to lead to what is subjectively felt as satisfaction. The latter occurs when similarities are identified, when congruities develop between past experience and concurrent input. On the other hand, the achievement of internal control, through the fulfillment of intentions or the restoration of ongoing plans, is conceived to result in what is experienced subjectively as gratification. The organism is gratified when there is congruity between present outcomes and past plans—when it can do things pretty much as it intended to do them. This formulation, derived from neurobehavioral data, fits the neurophysiological facts that, whereas the process labeled participation is accomplished largely through the posterior intrinsic or classical sensory "association" mechanism, the process labeled preparation is effected through the functions of the frontolimbic system.

PREPARATORY PROCESSES The clinical and experimental literature is replete with examples of preparatory processes. Concepts such as "repression," "suppression," and "perceptual defense mechanisms" can be interpreted as preparatory processes, for they are forms of defensive "gating out," that is, ignoring or repudiating those aspects of the situation that initiated the emotional state. Facets of sleep also have this "shutting out" characteristic, especially the syndromes of cataplexy and narcolepsy, which are often accompanied by affective changes.[18,34] (The above examples refer to the efferent control of afferent input at the neural level; other states represent the preparation for control of input through motivated action. Of the latter, Cannon's fight-flight reactions are probably the best known.)

By definition, preparatory processes do not accommodate the organism to the input; rather, they are internal stabilizing responses for the eradication of perturbation. The system is prepared to make itself independent of input—in effect, to make itself temporarily autonomous of concurrent input. Re-equilibration is directed toward some *status quo ante*. As it is detailed elsewhere,[80] concentration, fear, anger, and apprehension have in common the intent, implicit or explicit, to change the situation so that the organism can *repair* to the previous equilibrated state out of which it was so rudely jarred. In

this way, the ongoing plans of action are conserved, provided the preparatory processes are successful in eliminating input.

But attempts to eliminate input are often not successful. The source of the disturbing input remains and the perturbation may become incessant. For when preparations are directed to the input processing channels, they have the disadvantage of not disposing of the source of the inputs responsible for disequilibration. Under such circumstances, preparations may become chronic, for the incongruities arise again and again. Repeated preparations progressively lead to the hyperstability of complete internal control; the organism becomes divorced from reality; the plans of action become inflexible. Thus, more and more, novel inputs become appraised as irrelevant, or not feasible to the ongoing plans. When this hyperstable, inflexible state is finally disrupted by an input that cannot be eliminated, the entire system becomes perturbed. Then, as the saying goes, "all hell breaks loose."

PARTICIPATORY PROCESSES By contrast, participatory processes deal with incongruity by searching and sampling the input and accommodating the system to it. In this case, re-equilibration does not take the form of achieving the *status quo ante*; rather, the experience becomes part of the organism and the plans of action are appropriately modified. Re-equilibration, by incorporating input, proceeds to alter and restructure the organization so that it can again function gracefully, with a minimum of disequilibrium. Interest, affection, compassion, admiration, awe, wonder—all partake of this participatory quality. Such examples of participatory processes have in common some kind of involvement, engagement, or commitment to environmental events or plans that extend beyond the organism.

In the extreme, participatory reactions can lead to overwhelming external control or regulation of behavior. This makes the system highly dependent on environmental vicissitudes, with little recourse to a core organization, so the organism's ongoing plans are likely to become fragmented and the continuity of the psychological process and of behavior are sacrificed. The system becomes unstable, hyperreactive, and the organism overly distractible.

Emotion and Motivation

That an ongoing pre-perceptual and pre-behavioral organization—some dispositional context or plan—is so fundamentally related to the emotional processes of preparation and participation clarifies the relationship between motivation and emotion. Just why these two psychological categories are so often juxtaposed is seldom mentioned in the literature. When psychologists are asked to make the relationship explicit, the explanations are often muddied: "Both are related to physiological drives" (How?); "Sometimes an emotion is motivating" (In what way?); "There really isn't any difference" (Then why use both words?). However, in light of the present proposal, once it is clear that emotions are not just viscerally derived, that they stem primarily from dispositional contexts—from ongoing plans—the enigma is resolved. Motive implies action; to *e-mote* implies to be *out of* or *away from* action. In terms of the TOTE unit (Figure 1), the emotions are concerned with the regulation of input, which is to say with the feedbacks, the preparatory and participatory processes effecting efferent control over input. In essence, then, emotions are the result of neural dispositions or attitudes that regulate input when action is temporarily interrupted—literally e-motion. Motive, on the other hand, involves the organism in action, in the execution of its plans. Emotion and motivation, passion and action: these are the two poles of the Plan.

Paradoxically, whereas an organism has a good deal of control over input, it has much less control over the outcomes of behavior, except in very restricted situations. Input can be ignored, if necessary, but action always begets risk: one cannot be sure what will happen in the environment as a consequence of the action. Risk is countered only by experience.

The suggestion is that those terms we call "emotions" can also serve as names for "motives": love as an emotion has its counterpart in love as a motive. Fear the emotion has its mirror image as fear the motive. Being moved by music can be apposed to being moved to make music. And so on. Emotions and motives can, of course, be gracefully interdigitated; that is, when either the passive or active mode of the Plan

becomes prepotent, maladaptation is likely to occur. Too much emotion leads either to disruption through participation or to rigidity through preparation. Furthermore, the emotion may become a disequilibrating input in itself, for it begets further incongruities which cannot be acted upon. Too much planned action, on the other hand, leads to a narrowness of purpose and a poverty in values.

In the well-constructed individual, the process of motivation and emotion go hand-in-hand. Experience is segmented, action is monitored by passion, and passion is molded into timely action. Having come full circle, this is the theory of emotion that emerges from today's neurological knowledge: the realization of Marcus Aurelius' dictum.

SUMMARY AND CONCLUSION

In conclusion, then, this proposal differs saliently in several respects from most currently held views on emotion. First, the proposal is memory-based rather than drive-based or viscerally based. Second, it takes as a baseline organized stability and its potential perturbation, rather than some level of activation. Third, it makes explicit the relation between motivation and emotion by linking both to an ongoing, prebehavioral organization, a disposition, a program, or a plan. Fourth, the proposal defines emotion as e-motion, a process which, by taking the organism out of motion, effects control not through action but by the regulation of input. Fifth, the proposal identifies, on the basis of data presented, two forms of input regulation: one reduces, the other enhances, redundancy.

Thus, two forms of such control over input are recognized. One constitutes a preparatory, protective mechanism that conserves the current configuration by simplifying the input channels and thus limiting the effective input. The other, a participatory operation, influences the input channel in the direction of complexity, thus opening the current configuration to revision by nuances.

Both preparatory and participatory processes momentarily preclude action. Control is achieved either through stabilization of the neural configuration per se or through meshing this configuration with cur-

rent input. Emotion so conceived is therefore an essential mechanism for increasing the strength and flexibility of the organism's repertoire of internal alternatives with which novel situations are met.

Although different from most of the popular neurological views of emotion, this proposal does not stand completely alone. Peters[60] has made the case for emotions as appraisals—states that are related to the passive frame of reference. Melges and I have elsewhere[40] amplified and extended this relation between the proposed mechanism and subjective phenomena. On the behavioral side, Mandler has described a series of experiments in which the interruption of action is manipulated.[42] In a social context, Schachter[63] has detailed the differences between the effects of internal and external control over behavior. Although different in detail, all of these share with the present proposal the inclusion of other than visceral or activational aspects of emotion. Emotion is thus enriched; and this enrichment stems in each instance from the belated recognition by behavioral scientists that "nonaction" can be as complex and interesting a topic for psychological study as behavioral action.

The Neural Basis of Aggression in Cats

JOHN P. FLYNN

First I will criticize certain aspects of Dr. Pribram's theory of emotions, and second will present some data relevant to the neurophysiological basis of attack behavior in cats.

Dr. Pribram says that emotion means putting an organism out of action. I find this in disagreement with certain obvious forms of emotional behavior. For example, in classical "sham rage" animals, relatively trivial stimuli can release a flurry of activity—that is, a highly

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emotional display. Similarly, one can stimulate the hypothalamus of a cat resting quietly in a cage and cause it to attack another cat or a rat furiously. The cat is set *in* motion, not taken *out* of motion.

Dr. Pribram also says that emotions are associated with disruption of plans. To follow his usage, I think of them as sometimes *establishing* plans. Directed behavior has been a primary criterion in distinguishing "real rage" from "sham rage." If a cat chases a rat around a cage, I assume it has a plan. Certainly the rat being chased behaves as though it thought so, too.

Dr. Pribram also speaks of emotion as being associated with regulation of sensory inputs by afferent inhibition. I agree that sensory inputs are regulated in emotional behavior, but the emergence of a sensory field during stimulation of the brain is not inhibition, or even inhibition of inhibition. Later I will discuss an example of a change in the sensory field that occurs in cats stimulated to attack.

Furthermore, the changes that take place in emotional states are not restricted to variation of sensory input. Preparation for fight or flight takes place via the sympathetic nervous system, a motor system. Our own data also suggest that there are analogous preparations at the level of the nerve cells controlling skeletal musculature.

In view of this volume's intention to deal with the neurophysiological basis of emotions, I would like to state what I mean by emotion, and how evidence can be gained about its neurophysiological basis. Then I will summarize our findings with respect to the cat's attack upon a rat, and point out some of the various neurophysiological mechanisms involved in this form of behavior.

The term emotion has at least three meanings. First, it is regarded as a purely subjective feeling, recognized only through our own introspections or, secondarily, through those of other human beings—if they choose to tell us about them. The basic mechanisms within the central nervous system that mediate emotion in this sense can be investigated only in conscious human beings, and there is a relative paucity of such information. Second, emotion may mean an expression or display, which may or may not be accompanied by a subjective feeling, such as the sham rage of a decorticate dog, or the facial expression of an actor. The parts of the neural axis needed for one

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ACKNOWLEDGMENT

This research was supported by NIMH Career Award MH–15,214, NIMH Grant MH–03732, and Dept. of the Army Contract MD–2328. I am especially indebted to Dr. Fred Melges who collaborated with me on an extension of this proposal, and to Mr. Walter Tubbs and Mrs. Phyllis Ellis, who retyped and edited the many revisions.

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