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Chapter 10

THE BIOLOGY OF EMOTIONS AND OTHER FEELINGS[1]

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ABSTRACT

There is a convergence between earlier biological theories of emotion and a current, far more comprehensive view. The medieval humoral theories find a berth in current endocrine research. Lange's visceral theory is confirmed as taking part in the arousal mechanism that provides feelings of interest, novelty, familiarity, and more painful disruptions of stable states. James' emphasis on a report to the brain of bodily responses is shown to be a manifestation of the brain's representation of familiar body responses so that departures from the familiar are arousing. Cannon's thalamic theory is supported by the documentation that the diencephalon is a prime locus for receptors sensitive to the humors that determine the body's chemical response systems and thus central to the (homeostatic) maintenance of the stable representations of body states. Papez' and MacLean's extension of Cannon's brain locus to the limbic forebrain is also amply supported—and one must now add (as foreshadowed by Nina Bull's attitude theory) the basal ganglia as well—in that limbic formations and basal ganglia are shown

[1] This chapter was written in response to a request to contribute an up-to-date "biological theory of emotion" to this volume because I have attempted to formulate such a theory in the past (see Pribram, 1967, 1969, 1970, 1971; Pribram & Melges, 1969). P. T. Young (1973) has characterized these attempts as "comprehensive," a tribute to the wealth of data on the topic that had been accumulated over the past few decades by neurobiological scientists. The flow of data has not slowed since the earlier reviews were written. The current revision of the theory is therefore extensive.

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to be part of the neurochemical regulating mechanisms responsible for stable states and the sensing of departures from such stable states. Such departures were at an earlier time encompassed by Lindsley, by Duffy, and by Malmo under the banner of activation theory. Finally, as demanded by clinical evidence and social psychological experiment, the cerebral isocortex is not exempt from playing a critical role in the organization of emotional and motivational experience and expression. Laboratory evidence has shown that the intrinsic portions of the cerebral cortex, the "association" corteces of the posterior convexity and of the frontal pole, exert their influence via the basal ganglia, the forebrain focus of the arousal and readiness systems. This cortical control accounts for the specific epicritic "labeling" of feelings.

It was not so very long ago that I attended a symposium on "emotion" at an international congress in Montreal. The participants discussed factor analysis, limbic neuroanatomy, and operant conditioning. Somewhere in the agenda emotions were hidden from view, lurking in the dark alleys of our ignorance. No one even dared to use the term, and certainly no one discussed emotion as would the man in the street.

Let us therefore listen for a moment to ordinary discourse: "She's really emotional—so easily upset." "She's certainly an up and down person—so moody." "He's completely hung up on death; it would help him if he weren't so emotional about it." "Aren't they a warm emotional family?" "He makes me angry." "She is a loving person."

Common to these expressions is the theme of a cyclically recurring process—some steady state and its control. "Upsets" and "hang-ups" indicate malfunctions of control; "warmth" suggests that the regulatory mechanisms controlling emotional state are functioning flexibly and smoothly. It is these states and their regulation about which today's scientists have attained such a considerable body of evidence.

Current scientific knowledge regarding emotion has its roots in the Galenical medicine of the Middle Ages. Four "humors," sanguine, choleric, phlegmatic, and melancholic, were considered to determine temperamental differences in reactivity. The humors were thought to be bodily secretions, and modern biomedical research has supplanted these primitives with a host of endocrine hormones. The hormones must, of course, even today be seriously considered in any comprehensive treatment of the biological regulations that determine emotions.

In addition to the multiplication and specification of humors, two other major developments have occurred in the scientific study of the biology of emotions. One of these developments points to the role of nonhumoral mechanisms in the emotional process: Lange's (1887) "visceral" theory, made famous by William James (1890), and Nina Bull's (1951) "muscle"-based attitude theory are probably the most important of these.

The second major development shows brain mechanisms to be central and

critical to understanding. The realization that the brain is involved in the experience and expression of emotions began with the work of Gall and Spurzheim (1809/1969) at the beginning of the nineteenth century and achieved considerable sophistication by its end. Thus, William James (1890) could write:

> If the neural process underlying emotional consciousness be what I have now sought to prove it, the physiology of the brain becomes a simpler matter than has been hitherto supposed.
>
> Supposing the cortex to contain parts, liable to be excited by changes in each special sense-organ, in each portion of the skin, in each muscle, each joint, and each viscus, and to contain absolutely nothing else, we still have a scheme capable of representing the process of the emotions. An object falls on a sense-organ, affects a cortical part, and is perceived; or else the latter, excited inwardly, gives rise to an idea of the same object. Quick as a flash, the reflex currents pass down through their preordained channels, alter the condition of muscle, skin, and viscus; and these alterations, perceived, like the original object, in as many portions of the cortex, combine with it in consciousness and transform it from an object-simply-apprehended into an object-emotionally-felt. No new principles have to be invoked, nothing postulated beyond the ordinary reflex circuits, and the local centres admitted in one shape or another by all to exist [Vol. II, pp. 472–474].

And Sigmund Freud (1895/1966) could develop a detailed neurological model of emotional development in his Project for a Scientific Psychology:

> The primary brain . . . would, to put it plainly, be a *sympathetic ganglion* [S. E., p. 303].
>
> There must [therefore] be "secretory" neurones which when they are excited, cause the generation in the interior of the body of something which operates as a stimulus upon the endogenous paths of conduction [S. E., pp. 320–321].
>
> The endogenous stimuli consists of *chemical products*, of which there may be a considerable number [S. E., p. 321].
>
> At first, the human organism is incapable of bringing about the specific action. It takes place by *extraneous help*, when the attention of an experienced person is drawn to the child's state. In this way this path of discharge acquires a secondary function of the highest importance, that of *communication*, and the initial helplessness of human beings is the *primal source* of all *moral motives* [S. E., pp. 317–318].

These early formulations capture the essence of what needs to be covered in any comprehensive theory of emotions. Understanding in psychology comes when there is a sufficiently precise delineation of the variables—both environmental and organismic—that determine the behavior which reflects the psychological category under investigation. Thus, for example, we begin to understand color vision when we can specify the stimulus dimensions, the wavelengths of the electromagnetic spectrum that are involved, and something

of their interactions. We achieve more understanding when we find in the retina three photochemicals that possess characteristics similar to those derived as "primary" from studies of the interactions among spectral components (as proposed in the Young-Helmholtz theory). And we begin to feel that we know a considerable amount when De Valois (1960) shows us that cells in the lateral geniculate nucleus reflect an opponent process which accounts for otherwise inexplicable perceptual phenomena (according to the Hering theory, 1964).

A comprehensive theory of emotion must begin by specifying the manner in which emotional behavior differs from other behavior, the stimulus dimension that elicits such behavior, and the brain mechanisms that process this dimension. We begin, therefore, by defining the limits of what we mean by "emotional behavior," just as psychophysics initially had to establish the limits of what is meant by color vision.

EMOTIONAL BEHAVIOR AS AN EXPRESSION OF THE EXPERIENCE OF INTERNAL STATES

Since Darwin's classical treatise on the expression of emotion (1965) it has been customary to separate emotional experience from emotional expression. Emotional experiences are classes of feelings, and I have elsewhere (Pribram, 1970, 1971) made the case for utilizing the category "feelings" to encompass a range of experiences that can be separated from those that allow us to perceive objects beyond our skin:

> I once had the opportunity to examine some patients in whom the medial part of the temporal lobe—including the amygdala—had been removed bilaterally. These patients, just as their monkey counterparts, typically ate considerably more than normal and gained up to a hundred pounds in weight. At last I could *ask* the subject how it felt to be so hungry. But much to my surprise, the expected answer was not forthcoming. One patient who had gained more than one hundred pounds in the year since surgery was examined at lunch time. Was she hungry? She answered, "No." Would she like a piece of rare, juicy steak? "No." Would she like a piece of chocolate candy? She answered, "Umhumm," but when no candy was offered she did not pursue the matter. A few minutes later, when the examination was completed, the doors to the common room were opened and she saw the other patients already seated at a long table eating lunch. She rushed to the table, pushed others aside, and began to stuff food into her mouth with both hands. She was immediately recalled to the examining room and the questions about food were repeated. The same negative answers were obtained again, even after they were pointedly contrasted with her recent behavior at the table. Somehow the lesion had impaired the patient's *feelings* of hunger and satiety and this impairment was accompanied by excessive eating!
>
> As yet we understand little of how this impairment comes about. Nevertheless, this example points clearly to the folly of believing that a direct match exists between observations of any particular type of behavior and introspectively derived con-

cepts. Are we to say that the patient *felt* hungry because she ate ravenously despite her verbal denial? Or are we to take her statements at face value and seek elsewhere for an explanation for her voracious eating? The paradox is resolved if, as in earlier chapters on perception, we consider the behavioral function to be composed of several processes, one of which is the feeling state reported verbally.

At the hypothalamic level a similar paradox has plagued investigators. As already noted, when lesions are made in the region of the ventromedial nucleus of the hypothalamus, rats will eat considerably more than their controls and will become obese. But this is not all. Although rats so lesioned ate a great deal when food was readily available, they worked less for food whenever some obstacle interfered (Miller, Bailey, and Stevenson, 1950).

It was also found that the more palatable the food, the more the lesioned subject would eat (Teitelbaum, 1955), giving rise to the notion that the lesioned animals did not show greater "drive" to eat but were actually more "finicky" than their controls. Recent experimental results obtained by Krasne (1962) and by Grossman (1966) added to the paradox: electrical stimulation of the ventromedial nucleus stops both food and water intake in deprived rats and chemical stimulation of the cholinergic mechanism produces foot stamping (in gerbils, Glickman, personal communication) and fighting if provoked (King and Hoebel, 1968).

Grossman summarizes these results with the succinct statement that medial hypothalamic manipulations change affect not appetite. But we are once again faced with our earlier dilemma. If the medial hypothalamic mechanism does not deal with motivation, how does eating, drinking, etc., come about? The data hold the answer. The ventromedial and lateral hypothalamic regions form a couplet, the lateral portion serving as a feeding, a "go" mechanism (which, when ablated, will produce rats which tend to starve), and the medial portion contains the "stop" mechanism.

The paradox is resolved by the hypothesis that processes ordinarily involved in taking the organism "out of motion" also generate affects or feelings of e-motion. Thus an important distinction between motivation and emotion becomes clarified: the term "motivation" can be restricted to the operations of appetitive "go" processes (such as those converging in the lateral hypothalamic region) that ordinarily result in behavior which carries forward an action, and the term "emotion" to the operations of affective "stop" or satiety processes of reequilibration [Pribram, 1971, pp. 192–194].

Thus, neurobehavioral data make imperative a reference to an encompassing category, feelings, with the subcategories emotion and motivation clearly distinguished. Emotion is found to be derived from processes that *stop* ongoing behavior: affective reactions accompanying the satiety mechanisms as in the quotation above, arousal as in the orienting reaction to distracting stimuli (see the following and Pribram & McGuinness, 1975), and more generally when behavior is interrupted (Mandler, 1964). By contrast, the organism is considered motivated when his readiness mechanisms are activated (see also following and Pribram & McGuinness, 1975), when he is ready to "go" and to continue "going." These responses are (as will be detailed below) critically organized by the basal ganglia (Pribram, 1977b) and have as their physiological indicators the contingent negative variation of DC brain potentials (Walter, 1967) and heart rate slowing (Lacey & Lacey, 1974).

The distinction between emotion and motivation is not a novel one. In his opening paragraph on emotions William James suggests that "emotional reaction usually terminates in the subject's own body" while motivation "is apt to go farther and enter into practical relations with the exciting object" (1890, Vol. II, p. 442). In a similar fashion, J. R. Kantor, whose interbehavioral analyses of psychological processes influenced B. F. Skinner so profoundly, distinguishes between affective and effective interactions: In affective interactions "the person is responding above all with internal body mechanisms" while effective interactions generate "implicit" (i. e., readiness) or overt responses toward the stimulus object (Kantor & Smith, 1975).

In short, for behavior, as well as for the neurophysiology of feelings, it becomes useful to distinguish emotional from motivational antecedents. Motivational antecedents imply that the organism is preparing to or actually acting on the environment, whereas emotional antecedents imply only that internal processing (internal control mechanisms) is in force. The distinction becomes manifest in the connotative differences between the meaning in English of the term "behavior" and its continental counterpart in German and French: "Verhaltung" and "comportment" both connote how one "holds oneself"—one's positive and negative attitudes—whereas the English "behavior" has the more pragmatic and active meaning of "entering into *practical* relations with the environment."

An important consideration arises at this point. If the expression of emotions is affective (rather than effective), that is, emotional expression terminates in the subject's own body, how then can we observe and work with such expressions in terms of their practical effect on the environment? Ordinarily, a behaviorally oriented experimentalist is concerned with the environmental consequences of behavior (e.g., the cumulative record in an operant situation). In these situations, according to our definition, behavior is motivated, not emotional. Thus, the behaviorist has had some difficulty in finding measures of emotional expression. Conditioned suppression of responses, bolles of rat feces, and the like have been used, but they fail to reflect the richness of (especially the pleasant and positive) emotional states that the observed organism can experience. Furthermore, ethologists working with social behavior have followed Darwin's lead and shown that organisms can "read" each other's emotional expressions and be influenced by them.

In these situations, emotional expression does have a practical influence beyond the emoting organism, *but only because of the communicative setting*. In such a setting the practical influence is completely dependent on the ability of other socially receptive organisms to sense the meaning of the expression. Effectiveness therefore does not depend on what the emoting organism does but on what the socially sensitive recipient is able to do. However, an intelligent self-aware organism such as *Homo sapiens* can use these emotional expressions motivationally, that is, to manipulate the social situation. Such manipulations, when deliberate and planned (see the following), characterize

the "con" artist, actor, and administrator. But often, through imitation and conditioning, the emotional expressions become automatic, leading to stereotyped interactions. Much of the social display behavior of animals (e.g., birds) is apparently of this type: Internal and/or external stimuli set in motion an emotional reaction which, when expressed, triggers another emotional reaction in a socially receptive conspecific (e.g., Hinde, 1954a,b, 1960). In these animals, behavior sequences are thus concatenated of emotional expressions (and labeled "instinctive"). Such concatenations comprising instincts can also be elicited when an organism becomes completely adapted to an ecological niche in the nonsocial environment (see Miller, Galanter, & Pribram, 1960, Chapter 5). By contrast, organized motivations ("plans") are constructed within the organism's brain and *mean* to enter into practical relations with the exciting object. The adaptive consequence of emotional expression is a function of the social matrix in which this expression occurs.

In summary, emotional behavior is defined as an expression of positive and negative emotional feelings which are inferred as reflecting certain internal neurological states of the organism. The term "feelings" is therefore not synonymous with the term "emotion" since it is possible to identify additional internal neurological states and the behaviors they determine. One such additional category encompasses motivational feelings and behavior. Emotions are distinguished from motivations in that emotional reactions ordinarily "terminate within the organism's body," whereas motivations are "apt to go farther and enter into *practical* relations with the exciting object." An exception arises in social behavior, however. When a socially sensitive organism can be influenced by the expression of emotions or when an organism is totally adapted to his ecological niche, the sequential triggering of emotional expressions can lead to automatic (instinctive) behavior that is often, though not always, highly adaptive. Note, however, that the adaptation is due not to the expression of emotion but to the forces operating in the social and physical environment. We must next explore, therefore, what constitutes these "triggering" stimuli for emotional expression. What stimulus dimension addresses a physiological state experienced as an emotion? And what neural control mechanisms determine how an emotional feeling will be experienced?

THE PROTOCRITIC DIMENSION OF STIMULI

The importance of humoral factors in determining emotional states has already been noted. Hormones are chemicals that exert their influence on the brain via receptors located in its core. In addition to this sensitivity to hormones produced by glands such as the gonads, thyroid, adrenal medulla, and cortex, the core-brain receptors monitor a host of other chemical and physical constituents of the internal environment of the organism. A respiratory con-

trol mechanism is sensitive to the partial pressure of CO_2; a temperature sensor monitors the warmth of the blood stream; sex hormones are selectively absorbed at one location and adrenal steroids at another; the difference in the concentration of sugar in the venous and arterial circulation is monitored as is the concentration of salt and, therefore, reciprocally, the concentration of water. Chemicals secreted by the walls of the gut and by the kidney and a host of other chemicals are being investigated because some experiments indicate that they too are sensed by cells in the core of the brain (see Pribram, 1971, Chapters 9 and 10, for a review).

Furthermore, this part of the brain is a veritable cauldron of chemicals locally secreted by aggregates of cells in one or another location. Catecholamines such as norepinephrine (closely related to the hormone epinephrine–adrenalin, which is secreted by the adrenal medulla) and dopamine (which metabolizes into norepinephrine), indole amines such as serotonin, and peptides such as endorphin (an endogenous morphine-like substance) abound. As might be expected, sensitivities to these neurohumors are also built into the mechanism.

Walter Cannon (1927), in his classical studies, determined that the relationship between the sensor and its chemical was such that the concentration of the chemical, though fluctuating, was maintained constant around some set point. He enunciated this relationship as the principle of homeostasis. The sensor monitors the quantity of the variable and signals by way of neural pathways or chemical secretions when the variable rises above or falls below a certain level. Such signals compose a negative feedback because their sign is opposite to that which characterizes the deviation of the quantity of the variable from baseline. Often the mechanism that counteracts the decrease of the variable, the appetitive phase, is separate from that which counteracts the increase, the satiety phase.

Individual homeostatic mechanisms are multiply interlinked into complex organizations. Thus, the thermostat regulating temperature is linked to the glucostat regulating food intake and these are linked to the osmoreceptors (the salt-water sensors) to control thirst and the thyroid-sensitive mechanism controlling activity. Through various metabolic interrelations (such as breathing) that take place in the body, these homeostatic mechanisms in turn regulate the partial pressure of CO_2, etc. (see Brobeck, 1963, for a review).

In short, the core of the brain (mesencephalon, diencephalon, and the basal ganglia and limbic systems of the forebrain) utilizes chemical regulations to control body functions. The configuration of concentrations of these chemicals, though fluctuating around some set point, is sufficiently stable over periods of time to constitute steady "states." These states are apparently experienced—as hunger, thirst, sleepiness, elation, depression, effort, comfort, etc. (For a more complete discussion of how an experimenter infers what an observed organism might be experiencing see Douglas and Pribram [1966] and Pribram [1971, Chapter 6]. More direct evidence is obtained by

psychopharmacological experiments where the effect of drugs of known neurochemical action on psychological state is assayed.) Although the chemical characteristics of each state are as yet incompletely specified, enough is known to allow one to say that the concentration of glucose is involved in the hunger mechanism, the concentration of salt in the thirst mechanism, the concentration of the indole amine serotonin and norepinephrine (a catecholamine) in the sleep mechanism (norepinephrine in dreaming), the concentration of dopamine (another catechol) in feelings of effectiveness (i.e., of elation and depression), the concentrations of endorphins (endogenous secretions of morphine-like substances) in those of temperature, novelty, and pain, and the concentrations of the enkephalins (adrenocorticotrophic hormones of the pituitary, see below) in those of effort and comfort (for reviews, see the following and Pribram, 1971, 1977a; Stein, 1978).

Note that the control of temperature and of pain falls into the homeostatic mold. But temperature and pain are also skin senses which share a common spinal pathway, and the question thus arises whether the skin components of these sensitivities are processed separately from those involved in internal regulations. The answer to this question is that parts of the skin components of temperature and pain are processed separately and parts are processed in conjunction with the chemical homeostats of the core brain.

The part of the skin components of temperature and pain sensitivity that is processed separately (in the parietal lobes of the cortex) from the homeostatic mechanism is characterized by what is called in neurology "local sign." This means that the sensation can be located on the skin and that the duration of the sensation is limited. Henry Head (1920) labeled such sensory experiences "epicritic" to distinguish them from more diffuse experiences that are obtained during early regrowth of severed nerves.

The remainder of the skin's temperature and pain sensitivities are processed in conjunction with the chemical core homeostatic mechanisms. The spinal temperature and pain tracts end in structures (such as the substantia gelatinosa of the dorsal spinal cord, the periaqueductal gray of the midbrain, and the amygdala of the forebrain) that are loaded with endorphins. Responses to hot and cold and pain are dramatically altered by electrical stimulations of these core portions of the spinal cord, brain stem (Liebeskind, Mayer, & Akil, 1974), and forebrain and are not affected by stimulations of the parietal cortex or the tracts leading to it (Chin, Pribram, Drake, & Greene, 1976; Richardson & Akil, 1974). The assumption is that the stimulations increase the local (and perhaps general) secretions of endorphins.

What is common to the homeostatic internal mechanisms and these aspects of pain and temperature processing is that they are sensitive simply to *amounts*, the quantities, of chemical and neural excitation. Processing does not lead to identification of location in time and space (or to other qualitative aspects of the stimulus such as color). Head (1920) termed the quantitative "diffuse" aspects of sensitivity "protopathic" because, in his experiments,

they arose while the regenerating nerves were in a pathological condition. The term needs to be modified to *protocritic* in order to include current evidence that such sensitivities are part of the *normal* control of the temperature and pain (and probably other sensory) mechanisms. As noted, protocritic processes are homeostatic; that is, they control the quantitative aspects of stimuli and are thus determinants of neural *states* (for a more complete review see Chin *et al.*, 1976; Pribram, 1977a).

The protocritic dimension of experience, devoid of epicritic local sign, is therefore characteristically dependent on the quantity (the intensity) of the stimulus. Quantity (and therefore intensity) in a homeostatic system is in turn dependent on change and rate of change of the state of that system. Controlled changes of moderate amounts are apparently experienced positively, while more abrupt and overly intense changes of state lead to negative feelings (the Yerkes-Dodson law [see Hebb, 1955]). Here we are at the frontier of knowledge. As noted, the pain and temperature systems run together in the spinal cord and brain stem to terminate in and around the amygdala and frontal cortex. Do the elaborations of the temperature systems accrue to the experiencing of comfort as the elaborations of the pain systems accrue to suffering? Or is suffering experienced only when the limits of tolerable comfort are exceeded? Brain stimulations in man that protect against pain are accompanied by the feeling of cold (Richardson & Akil, 1974). In short, are there two neural systems, one for pain and one for temperature, or is there only one? And if there are two, how do they interact to produce a more or less unitary experience along a hedonic dimension?

An area of current investigation in our laboratory is aimed at establishing the individual (and situational) differences in band width (range) of tolerance of the intensive dimension of stimulation: What are the limits of comfort between changes sensed at all and those sensed to be uncomfortable? How much effort must be expended in control of the band width (i.e., how much attention must be *paid* in order to expand the range over which stimuli are sensed comfortable)? How do differences in attitudinal set and the situational setting influence the nature (positive [comfort] versus negative [effort]) of the emotional experience? These investigations are based on earlier work that discerned a distinction between a neural system that controls the comfort–effort dimension and two others upon which the comfort–effort system operates. The next section examines the evidence for this distinction.

PROTOCRITIC PROCESSING BY CORE-BRAIN CONTROL MECHANISMS

Historically, the humoral theory of emotions gave way to the visceral theory of Carl Lange, which was promulgated by William James. As already noted, however, James emphasized the visceral (and somatic) components of

stimulation to the brain rather than the visceral phenomena per se (as is ordinarily suggested). Cannon (1929) performed a series of experiments designed to show that visceral stimulation per se did not account for emotional experience and expression. Cannon's experiments pointed to the diencephalon as the locus involved in organizing the states responsible for emotion. The evidence has already been reviewed that confirms the essence of Cannon's conclusions but extends the locus posteriorly to include the mid- and hindbrains, and even the spinal cord, and anteriorly to include the limbic formations and basal ganglia of the forebrain.

However, additional evidence, much of it from my laboratory, has shown that input from the body, including the viscera, through the autonomic nervous system is, after all, specifically involved in the organization of the neural states basic to emotional and motivational feeling. This work has shown that three classes of influence can be discerned and that this influence is necessary to stabilize the states rather than acting as a cue to emotional feeling. Some years ago Lindsley (1951) proposed an activation theory of emotions based on the fact that during emotional upset the electrical activity of the brain becomes desynchronized. Our evidence (reviewed in Pribram & McGuinness, 1975) showed that three separate systems could be discerned to influence electrocortical desynchronization. One system regulates phasic desynchronization i.e., brief, lasting at most several seconds), another regulates tonic desynchronization, and a third coordinates the other two (over a longer period of time—the duration of an attention span).

We called phasic desynchronization arousal. The system responsible for arousal centers in the forebrain on the amygdala, a basal ganglion of the limbic forebrain. Removal of the amygdala eliminates the visceral and autonomic responses that ordinarily accompany orienting and alerting to a change in stimulus conditions (Kimble, Bagshaw, & Pribram, 1965; Bagshaw, Kimble, & Pribram, 1965; Bagshaw & Benzies, 1968; Pribram, Reitz, McNeil, & Spevack, 1974; reviewed by Pribram & McGuinness, 1975). Furthermore, this elimination of the visceroautonomic responses apparently leads to a failure of behavioral habituation, which normally occurs rapidly when the novel stimulus is repeated: The visceroautonomic reaction speeds familiarization. *Thus, contrary to Lange and James, the visceral input appears not to be experienced directly as an emotion but leads to rapid habituation of the input. As shown by Sokolov (1960), habituation forms a stable neural representation. Such a stable state is necessary for appreciating subsequent change—the novelty which then arouses (emotional) interest and, when the novelty exceeds certain limits, the experiencing of (emotional) upset. James and Lange were correct in suggesting that visceral input is important to emotion but erroneous in the specific role they assigned it in the emotional process.*

The second system involved in the desynchronization of cortical electrical activity [in this instance a tonic (minute long) activation] is centered on the nonlimbic basal ganglia of the forebrain: the caudate nucleus and putamen (reviewed by Pribram, 1977b). These structures are concerned with maintain-

ing the (motivational) readiness of the organism: postural readiness, motor readiness, and the readiness produced by the establishing of sensory (i.e., attentional) sets (Lassonde, Ptito, & Pribram, 1975; Reitz & Pribram, 1969; Spinelli & Pribram, 1966, 1967). It is this second system that forms the neural basis for "attitudes," much as suggested by Nina Bull (1969) except that, in the context of the proposals made here, her book would be entitled "The Attitudinal Theory of Feelings" rather than "of Emotion."

A third system centers on the hippocampus and coordinates arousal and readiness (see Pribram and McGuinness [1975] for a review of the evidence on which this statement is based). Arousal phasically interrupts ongoing tonic readiness. The balance between interruption and continuation must be coordinated, and neurobehavioral and neurophysiological evidence points to the hippocampal system as serving such a function. Coordination has been shown to involve neural work, that is, to take effort (see book edited by Ingvar & Lassen, 1975).

Neurochemically the three systems also differ (reviewed by Pribram 1977a). As already noted, the amygdala is rich in endorphins and the caudate and putamen are characterized by dopamine. The hippocampal system is involved in the pituitary-adrenal hormonal controls, selectively absorbing adrenocortical hormone (see, e.g., Bohus, 1976; McEwen, Gerlach, & Micco 1976) and being acted upon by ACTH (adrenocorticotrophic hormone) and related enkephalins (van Riezen, Rigter, & Greven, 1977).

The humoral, visceral, and activation theories of emotion (and motivation are thus converging into a more comprehensive view that subsumes the earlier ones. The momentary arousal produced by novelty appears to be related to endorphin homeostasis, the activation of motivational readiness is based on a dopaminergic system, and coordinating effort (or its inverse, comfort) is experienced as a result of operations of the brain representation of the pituitary-adrenal hormonal stress mechanism.

The model of feelings (including emotional feelings) that emerges from these data centers on a set of core-brain neurochemical states that comprise the experience of a feeling of "familiarity." Such a feeling implies equilibration, a feeling of a reasonable amount of stability and a smooth transition from one state to another. This set of stable states can be altered by novel or pain-producing events, and what is perceived as novel, or painful, is dependent on the configuration of the states that determine what is familiar. The distinction between a feeling of novelty and pain is one of intensity *only* (e.g., electrical stimulations of the amygdala in animals and man produce orienting [interest], avoidance [fear], attack, and escape [pain] as a function of ascending stimulus intensity [Gastaut, 1954]. In contrast to the arousing disequilibrations produced by the novelty-pain mechanism, the maintenance of states is effected by tonic operations of the readiness system. This system may have evolved from, or in close coordination with, the temperature system.

There is considerable evidence that the maintenance of a stable basal temperature involves the food appetitive, water balance, and tonic muscular readiness systems, among others (see Brobeck, 1963, for a review). When the demands of arousal are pitted against those of continuing readiness, the feelings of stress and effort are experienced. These experiences are allayed by a coordinating mechanism that adjudicates the smooth transition from state to state within some comfortable band width of tolerance.

THE CORTICAL CONTRIBUTION TO A LABELING OF FEELINGS

The biological contribution to an understanding of feelings in general and emotional feelings in particular cannot rest here. A basic problem set out at the beginning of this chapter in the quotations from William James and Freud has to be faced. Freud proposed that the critical neurological mechanisms involved in emotion are neurochemical and derive from body stimulation (the endogenous paths) that affects a certain portion of the brain. The work reviewed here has given substance to Freud's proposal and enlarged upon it: A protocritic dimension of stimulation was identified, a dimension describing much of the input through visceroautonomic (endogenous) paths but also receiving a contribution from exteroceptors (exogenous paths), especially those of the pain and temperature senses. Furthermore, the processing of this protocritic dimension was found to take place in limited portions of the brain: the core-brain systems of the brain stem and the limbic forebrain.

William James (1890), in the passage from which the earlier quotation was taken, faced the possibility that such separate neural processing of emotion occurs:

> And yet it is even now certain that of two things concerning the emotions, one must be true. Either separate and special centres, affected to them alone, are their brainseat, or else they correspond to processes occurring in the motor and sensory centres already assigned, or in others like them, not yet known. If the former be the case, we must deny the view that is current, and hold the cortex to be something more than the surface of "projection" for every sensitive spot and every muscle of the body. If the latter be the case, we must ask whether the emotional *process* in the sensory or motor centre be an altogether peculiar one, or whether it resembles the ordinary perceptive processes of which those centres are already recognized to be the seat. Now if the theory I have defended be true, the latter alternative is all that it demands [Vol. II, pp. 472-474].

James opted for the cortex, as we saw in the remainder of this quotation in the introduction to this chapter. Was he wrong?

I do not believe so. There is more to feeling than the protocritic dimension.

Schachter (e.g., Schachter & Singer, 1962), in a classical set of experiments, has delineated two aspects to feeling: one that devolves on its intensity (which has been discussed here as the protocritic dimension) and the other that "labels" the feeling. Labels are specific: They identify the feeling with respect to a spatial and temporal or other qualitative context. In short, labeling is epicritic, and we should turn, as James proposed, to the cerebral convexity in the search for the neural mechanisms that are involved.

According to James (1890), what needs to be demonstrated is that "the reflex currents pass down through their preordained channels, alter the condition of muscle, skin and viscus; and these alterations, perceived, like the original object, in as many portions of the cortex, combine with it in consciousness and transform it from an object-simply-apprehended into an object-emotionally felt" (p. 253).

The work reviewed above has demonstrated that "the condition of muscle, skin and viscus" need not, in fact, be altered. A stable representation, a neural representation of bodily function including its quantitative hormonal composition, is interposed between "muscle, skin and viscus" and the cortex. All that needs to be established is that the representation (and its potential or actual perturbation) be addressed. The pathways whereby this can occur have now been thoroughly established both anatomically and physiologically (Goldman & Nauta, 1977; Kemp & Powell, 1970; Lassonde & Ptito, in preparation; Nauta, 1964; Reitz & Pribram, 1969).

The cortical contribution to the regulation of more primitive functions is, as might be expected, complex. Sense can be made of this complexity, however, by relating the myriad of observations on the effect of cortical lesions and excitations to the two simpler dimensions that have been delineated thus far. The cerebral isocortex is directly connected both to brain stem (core and shell portion) and to the remainder of the forebrain (basal ganglia and limbic formations). These connections can therefore modulate the epicritic-protocritic and the affective-effective dimensions of experience and behavior which are regulated by the more primitive structures.

The protocritic-epicritic dimension is reflected in the cortex by a front-back distinction in function. The anterior frontal cortex is so intimately related to the limbic systems that it can be conceived as the "association area" for these systems (Pribram, 1954, 1958a,b). Anatomically the anterior frontal (frontal intrinsic) cortex receives projections from the n. medialis dorsalis of the thalamus (an "intrinsic" nucleus because it is only indirectly connected with extracerebral inputs) which lies embedded within nuclei that project to limbic cortex. Behaviorally, resections of frontal intrinsic cortex result in deficits in delayed alternation performance, deficits also obtained when lesions are made of limbic structures but not when the posterior cortical convexity is damaged. By contrast, damage to the posterior cortical convexity (the posterior intrinsic cortex which receives its input from the pulvinar, another intrinsic thalamic nucleus) produces deficits in discrimination learning and

performance which remain unaffected by frontal and limbic lesions. The difference between alternation and discrimination has been conceptualized to reflect the difference between context-sensitive, episode-specific reactions on the one hand and context-free, automatic information-processing on the other (Pribram, 1978). More on this follows.

Recent evidence from the human neurological and neuropsychological clinic and from the recording of electrical brain activity in man has suggested that the effective-affective dimension receives a cortical contribution which is to some extent lateralized, i.e., the left and right hemispheres of the cerebral cortex contribute unequally to the regulation of behavior and the monitoring of feelings (Galin, 1977; Gazzaniga, 1970; Schwartz, 1975; Sperry, 1974). The fact of right-hand dominance (dexterity) and that linguistic expression is regulated by the functioning of the left cerebral hemisphere in most right-handed persons has been well known for a long time. What is new is evidence that the right hemisphere may also be specialized in the direction of a more holistic, parallel processing, experiential mode of operation.

The front-back and right-left distinctions of cortical regulation converge to produce a new dimension which I have labeled esthetic-ethical (Pribram, 1968). This dimension is based on the distinction between the processing of "external space" and the processing of a "body image" or "self." Processing which results in the effective use of local sign (the epicritic dimension) is a function of a band of cortex surrounding the three major cerebral fissures: Sylvian, Rolandic (central), and Calcarine. (The continuity between peri Rolandic and pericalcarine cortex is established at the apex of the cortical convexity: In the monkey brain this is at the confluence of the intraparietal, superior temporal, and lunate sulci. The continuity between peri Sylvian and peri Rolandic cortex lies at the foot of the central fissure).

In primates including man, the growth of the cortex surrounding these major fissures has split the remaining cortex into two subdivisions: (1) a posterior focused on the inferior parietal lobule on the lateral surface and the precunens on the medial (connected via the medial extension of the confluence between intraparietal and lunate sulci); and (2) the cortex covering the poles of the frontal and temporal lobes (interconnected by the fibers of the uncinate faciculus and adjacent to the orbitofrontal—anterior insular—periamygdaloid cortex which is a part of the limbic systems). The functional connectivities of these divisions and subdivisions of the cortical mantle have been most clearly demonstrated by strychnine neuronography (Bonin and Bailey, 1947; Pribram and MacLean, 1953) and have been confirmed histologically by the use of silver staining techniques (Jones, 1973; Nauta, 1964).

The behavioral evidence showing that the perifissural cortex processes "external space" while the remaining cortex processes "self" is so extensive that only the highlights can be listed here: (1) Beginning with the precentral (preRolandic) cortex, Pribram, Kruger, Robinson, and Berman (1955) showed that the environmental consequences of movement, not movements or muscle

contractions per se are encoded in this "motor" cortex (see review by Pribram, 1971). (2) The postcentral and superior parietal cortex deals with the somatosensory (haptic) discrimination of objects in external space (Brody and Pribram, 1978; Kruger and Michel, 1962; Mountcastle, Lynch, Georgopoulos, Sakata, and Acuna, 1975; Pribram and Barry, 1956). (3) The pericalcarine cortex deals with visual processing (see Weiskrantz, 1974, for review) and its extension into the inferior temporal gyrus, with making visual discriminations (see Pribram, 1974, for review). (4) The posterior perisylvian cortex is involved in auditory processing (see D. Neff, 1961, for review) and its extension into the superior temporal gyrus with auditory discriminations (Dewson, 1977; Dewson and Cowey, 1969; Dewson, Pribram, and Lynch, 1969). (5) The anterior perisylvian cortex in the depths of the fissure and extending forward to the temporal pole and orbital surface of the frontal lobe processes gustatory information (Bagshaw and Pribram, 1953; Pribram and Bagshaw, 1953), and is also involved in olfactory (Brown, 1963; Brown, Rosvold and Mishkin, 1963), and, as noted earlier, temperature discriminations (Chin, Pribram, Drake, and Green, 1976).

By contrast to these clearcut results of experiments relating the perifissural cortex to processing of "external space," the evidence for processing "self" by the remaining cortex is somewhat more difficult to interpret. Initially, data were believed to point to the anterior frontal cortex as the main source of an image of self. Recent experimental results show, however, that this conclusion was oversimplified and to a large extent erroneous (Brody and Pribram, 1978). Furthermore, clinical evidence has shown the inferior parietal lobule to be concerned with body-image: Lesions of this cortex lead to severe "neglect" of the opposite side of the body and this is especially severe when the lesion is in the right hemisphere. The lesions are often deep involving the precuneus and its connections (see Pribram and MacLean, 1953) with the cingulate and retrosplenical portions of the limbic cortex (Geschwind, 1965).

What seems to be a more accurate reading of current available evidence is that there is a balance between the parietal and frontal (including temporal pole) portions of this cortex which processes "self." While lesions of the parietal cortex lead to "neglect," lesions of the frontal and temporal poles lead to its opposite (Teuber, 1972; Geschwind, 1965). Patients with frontal and temporal lobe involvement tend to talk and write voluminously about themselves and, as noted, to lose control over behavior which is context-sensitive, i.e., depends on some stable mnemonically organized self. (See Fig. 10.1.)

To summarize: The cortical contribution to emotion relates the affective-effective dimension to the protocritic-epicritic in such a way that a new dimension, labeled ethical-esthetic, emerges. This new dimension is based on the construction of a self-concept which is organized and enhanced by parietal and selectively inhibited (made context sensitive) by frontal cortical functioning. The construction is achieved in man by combining a frontolimbic protocritic versus cortical convexity epicritic axis with a right hemisphere affec-

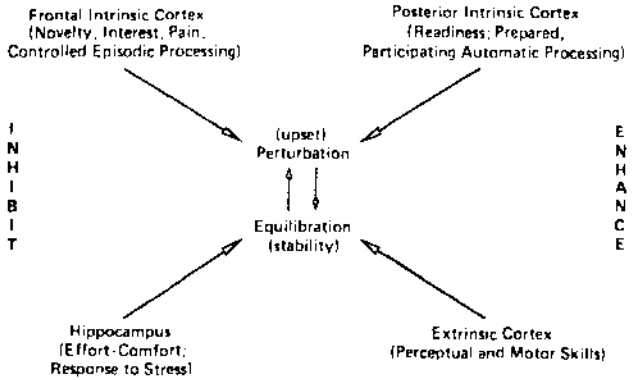

FIGURE 10.1 A highly schematic diagram that portrays cortical influences on the basic neuronal mechanisms that regulate stable states. The Perturbation ⇄ Equilibration couplet is composed of inhibitory interactions. Lateral inhibition pushes the couplet toward perturbation while recurrent inhibition acts as a negative feedback to stabilize and equilibrate. The data base for the arrows from the various cortical regions to the central couplet is derived from recovery cycle experiments. When a cortical stimulation increased the variance of an initial potential evoked by abrupt peripheral stimulation (in the visual, auditory, and somatosensory modes), the stimulus was considered to inhibit equilibration. This effect was obtained when the hippocampus was electrically stimulated. Conversely, a decrease in such variability signaled an enhanced equilibration, a result obtained when the primary sensory and motor cortical regions were stimulated. Furthermore, the second of a pair of such stimuli ordinarily evokes a diminished response (due to a lag in recovery of the channel) when administered over short interstimulus intervals (the recovery cycle). When the duration and level of diminution are increased, the input channel becomes desynchronized; that is, perturbation is enhanced. This effect is obtained when the posterior intrinsic cortex is electrically excited. The converse, inhibition of perturbation, is signaled by a more rapid recovery of the second potential evoked by the sensory stimulation. Such an effect is produced by electrical excitation of the frontal intrinsic cortex. Thus, the intrinsic cortical regions manifest their effect in the recovery cycle data; the extrinsic and hippocampal formations influence the variability of the initial evoked response directly. Both the intrinsic- and extrinsic-hippocampal effects are balanced: Frontolimbic formations exert their influence through inhibition (INHIBIT, a result that has been independently obtained in several series of experiments), whereas the cortical convexity operates via excitation (ENHANCE). This fourfold mechanism thus provides exquisite control over central neural stability and its potential and actual perturbations.

tive versus left hemisphere effective axis. The poles of each axis have been found to oppose each other (Lassonde and Ptito, in preparation; Pribram, Lassone, and Ptito, in preparation; Spinelli and Pribram, 1967) in such a way that a combinatorial balance of control is achieved (Jackson, 1873).

CONCLUSION

Clearly, considerable progress has been achieved in recent years in understanding the biology of emotion and other feelings. The intensive (protocritic) dimension of emotional experience is being correlated with

neurochemical states and factors that control the changes in those states. Emotional expressions are related to these controls and are found to "stop at the skin" and, ordinarily, not to get into "*practical* relations with the environment," as William James so aptly stated. An exception to this occurs in social behavior, where emotional expressions are "read" by other persons and thus exert a practical influence through the exercise of the recipient rather than the emoting organism.

These experimentally based definitions of emotions distinguish them from motivational feelings and behavior. An affective-effective dimension is thus recognized as grounded in biological as well as in social fact.

The biological roots of the protocritic (intensive) dimension of emotional and motivational feelings concern two interrelated sets of systems:

1. Neurochemical (i.e., neurohumoral) control systems establish relatively enduring configurations, that is, stable states, by way of homeostatic regulations. To date, the catechol and indole amines and several peptides have been found to be especially important components of the neurohumoral configurations that determine such states.

2. Neuroelectric control systems ensure the stability and smooth changes of these states and involve arousal, activation, and effort. Arousal has been shown to be a phasic response to input experienced as interesting and novel or as disruptive. Arousal is regulated by a system whose forebrain locus is in the amygdala. Neurochemically this system is rich in sex hormones (among others) and in the neuropeptide endorphin, a regulator of pain. By contrast, activation is a tonic phenomenon whose forebrain locus centers on the basal ganglia, which are part of a dopaminergic neurohumoral system. Psychophysiological and neurobehavioral experiments have shown this system to provide continuing readiness to respond; it is therefore more directly involved in motivational than in emotional experience and expression.

3. A third control system whose forebrain locus centers on the hippocampus has been shown to regulate the coordination of arousal and activation. This system is intimately involved in the pituitary-adrenal cortical humoral response to stress by way of adrenocorticosteroid receptors and the pituitary secretion of adrenocorticotrophic hormones (ACTH) and peptides related to ACTH, the enkephalins. The system is thus responsible for the experiences (feelings) of effort and comfort and their expression.

Furthermore, current evidence attests to the involvement of body mechanisms in these brain regulations of state. Already noted is the role of the pituitary-adrenal axis in determining effort and comfort. Visceral involvement has been demonstrated as important in the registration of novelty (and familiarity). And the role of the motor system in attitudinal activation is clear-cut. These muscular, skin, and visceral inputs are not directly responsible for emotional and motivational feeling as proposed by James and Lange, however. Rather, they are involved in the registration in memory of changes,

so that the organism can habituate to them. They are thus the components of which stable neural (core-brain) representations of bodily states are put together. Changes in these core-brain states, not in the somatic inputs per se, appear to be related to the experiencing of the protocritic dimension of emotion and emotional feelings.

Thus, there is a convergence of earlier biological theories of emotion into a current, more comprehensive view. The medieval humoral theories find a berth in current endocrine research. Lange's (1887) visceral theory is confirmed as taking part in the arousal mechanism that provides feelings of interest, novelty, and familiarity and more painful disruptions of stable states. James's (1890) emphasis on a report to the brain of bodily responses is shown to be a manifestation of the brain's representation of familiar body responses so that departures from the familiar are arousing. Cannon's (1929) thalamic theory is supported by the documentation that the diencephalon is a prime locus for receptors sensitive to the hormones that determine the body's chemical response systems. The diencephalon is central to the (homeostatic) maintenance of the stable representations of body states. Papez's (1937) and MacLean's (1949) extension of Cannon's brain locus to the limbic forebrain is also amply supported—and one must now add (as foreshadowed by Nina Bull's [1951] attitude theory) the basal ganglia as well—in that limbic formations and basal ganglia are shown to be part of the neurochemical regulating mechanisms responsible for stable states and the sensing of departures from such stable states. Such departures were at an earlier time encompassed by Lindsley (1951), by Duffy (1934), and by Malmo (1963) under the banner of activation theory.

Finally, as demanded by clinical evidence (e.g., psychoanalysis and other verbal psychotherapies [see Pribram & Gill, 1976]) and social psychological experimentation (e.g., Schachter & Singer, 1962), the cerebral isocortex is not exempt from playing a critical role in the organization of emotional and motivational experience and expression. Laboratory evidence has shown that the intrinsic portions of the cerebral cortex, the "association" cortices of the posterior convexity and of the frontal pole, exert their influence via the basal ganglia, the forebrain focus of the arousal and readiness systems. This cortical control accounts for the specific epicritic "labeling" of feelings.

In short, as foreshadowed by William James, the entire brain is involved in the regulation of emotional experience and expression. But each part of the brain has a very specific role in the totality, and this role involves the sensing and control not only of other neural events but of body functions as well. The essence of an intensive protocritic dimension emotion—as experienced and expressed by the man in the street—is this regulation of changes in stable states, corebrain states, and body states. Brief episodic mild changes in stability are experienced as arousing and interesting (novel); more severe disruptions are painful and frightening. When control is exercised to contain such changes, it is experienced as effortful; when little control is needed, behavior is automatic

and the experience is one of comfort. The intensive dimension of feeling is not the only dimension, however. Epicritic, specific labeling is also a prominent feature of emotional and motivational experience and expression. Evidence has been reviewed that shows a cortical contribution by way of input to core-brain structures to be responsible for the epicritic aspect, the labeling of feeling states.

But, in concluding, it should again be pointed out that a significant portion of the controls exercised are not through neural circuitry but by way of chemical regulations, that is, secretions or hormones in different concentrations that lay the foundation for differences in reactivity and thus differences in disposition and temperament. A single exposure to testosterone during a critical embryological period will change a female's reactivity to that of a male for the rest of its life (Young, 1961; Phoenix, Goy, & Young, 1967). Presumably that exposure programmed a brain mechanism that thereafter controlled behavior according to the testosterone program. Changes in adrenal cortical hormone level during disease (Cushing's syndrome), or by virtue of experimental excision, alter sensory thresholds and tolerances in several modalities (Henkin, 1970). Thyroid deficiency in infants produces cretins; in adults, activity levels are related to the amount of circulating thyroid hormone (Vernikos-Danellis, 1972a). Disturbed insulin and therefore sugar metabolism has been related to feelings of irritability and malaise (Vernikos-Danellis, 1972b). Could it be that Galen's theory was but a degenerate view of some more precise knowledge attained by the ancients, which we are only now regaining? Or is it that, like the concept of the atom, vague formulations which were held for centuries finally reached precision when subjected to scientific exploration? In either case, the search continues with just enough yield of novelty to maintain the interest of explorers of emotion—ensuring that profitable change in understanding continues.

REFERENCES

Bagshaw, M. H., & Benzies, S. Multiple measures of the orienting reaction and their dissociation after amygdalectomy in monkeys. *Experimental Neurology*, 1968, *20*, 175–187.

Bagshaw, M. H., Kimble, D. P., & Pribram, K. H. The GSR of monkeys during orienting and habituation and after ablation of the amygdala, hippocampus and inferotemporal cortex. *Neuropsychologia*, 1965, *3*, 111–119.

Basgshaw, M. H., & Pribram, K. H. Cortical organization in gustation (Macaca mulatta). *Journal of Neurophysiology*, 1953, *16*, 499–508.

Bohus, B. The hippocampus and the pituitary adrenal system hormones. In R. L. Isaacson & K. H. Pribram (eds.), *The hippocampus*, pp. 323–353. New York: Plenum, 1976.

Bonin, G. von, & Bailey, P. The neocortex of Macaca mulatta. *Illinois Monographs in the Medican Sciences*, Vol. 5, No. 4. Urbana: University of Illinois Press, 1947.

Brobeck, J. R. Review and synthesis. In M. A. Brazier (ed.), *Brain behavior*, Vol. II, pp. 389–409. Washington, D.C.: American Institute of Biological Sciences, 1963.

Brody, B. A., & Pribram, K. H. The role of frontal and parietal cortex in cognitive processing: Tests of spatial and sequence functions. *Brain*, 1978, *101*, 607–633.

Brown, T. S. Olfactory and visual discrimination in the monkey after selective lesions of the temporal lobe. *Journal of Comparative and Physiological Psychology*, 1963, *56*, 764–768.

Brown, T. S., Rosvold, H. E., & Mishkin, M. Olfactory discrimination after temporal lesions in monkeys. *Journal of Comparative Physiological Psychology*, 1963, *56*, 190–195.

Bull, N. *The attitude theory of emotion*. Nervous and Mental Disease Monograhs Series, New York: Johnson Reprint Corp., 1969.

Cannon, W. B. The James-Lange theory of emotions: A critical examination and an alternative theory. *American Journal of Psychology*, 1927, *34*, 106–124.

Cannon, W. B. *Bodily changes in pain, hunger, fear and rage*. New York: D. Appleton, 1929.

Chin, J. H., Pribram, K. H., Drake, K., & Greene, L. O., Jr. *Disruption* of temperature discrimination during limbic forebrain stimulation in monkeys. *Neuropsychologia*, 1976, *14*, 293–310.

Darwin, C. *The expression of the emotions in man and animals*. Chicago: University of Chicago Press, 1965.

De Valois, R. L. Color vision mechanisms in monkey. *Journal of General Physiology*, 1960, *43*, 115–128.

Dewson, J. H. III. Preliminary evidence of hemispheric asymmetry of auditory function in monkeys. In S. Harnard, R. W. Doty, J. Jaynes, L. Goldstein, G. Crauthamer (eds.), pp. 63–71. *Lateralization in the nervous system*. New York: Academic Press, 1977.

Dewson, J. H. III., & Cowey, A. Discrimination of auditory sequences by monkeys. *Nature*, 1969, *222*, 695–697.

Dewson, J. H. III., Pribram, K. H., & Lynch, J. Effects of ablations of temporal cortex on speech sound discrimination in monkeys. *Experimental Neurology*, 1969, *24*, 579–591.

Douglas, R. J., & Pribram, K. H. Learning and limbic lesions. *Neuropsychologia*, 1966, *4*, 197–220.

Duffy, E. Emotion: An example of the need for reorientation in psychology. *Psychology Review*, 1934, *41*, 184–198.

Freud, S. *Project for a scientific psychology* [orig. publ. 1895], Standard Edition, Vol. 1. London: The Hogarth Press, 1966.

Galin, D. Lateral specialization and psychiatric issues: Speculations on development and the evolution of consciousness. In S. J. Dimond & D. A. Blizard (eds.) *Evolution and lateralization of the brain*. Vol. 299, pp. 397–411. New York: Annals of the New York Academy of Sciences, 1977.

Gall, F. J., & Spurzheim, G. [*Research on the nervous system in general and on that of the brain in particular*, pp. 255–275. Paris: F. Schoell, 1809,] In K. H. Pribram (ed.) *Brain and behavior*, Vol. I, pp. 20–26. Middlesex: Penguin Books, 1969.

Gastaut, H. Interpretation of the symptoms of "psychomotor" epilepsy in relation to physiologic data on rhinencephalic function. *Epilepsia*, Series III, 1954, *3*, 84–88.

Gazziniga, M. S. *The bisected brain*. New York: Appleton-Century-Crofts, 1970.

Geschwind, N. Disconnexion syndromes in animals and man: Part I. *Brain*, 1965, *88*, 237–294.

Goldman, P. S., & Nauta, W. J. H. An intricately patterned prefrontocaudate projection in the rhesus monkey. *Journal of Comparative Neurology*, 1977, *171* (3), 369–384.

Grossman, S. P. The VMH: A center for affective reaction, satiety, or both? *Physiology and Behavior*, 1966, *1*: 10.

Head, H. *Studies in neurology*. Oxford: Oxford Medical Publications, 1920.

Hebb, D. O. Drives and the CNS (conceptual nervous system). *Psychology Review*, 1955, *62*, 243–254.

Henkin, R. I. The neuroendocrine control of perception. In D. A. Hamburg, K. H. Pribram, & A. J. Stunkard (eds.) *Perception and its disorders*, Research Publications, Association for

Research in Nervous and Mental Disease, Vol. XLVIII, pp. 54-107. Baltimore: Williams & Wilkins, 1970.

Hering, E. *Outlines of a theory of light sense*. Cambridge: Harvard University Press, 1964.

Hinde, R. A. Factors governing the changes in strength of a partially inborn response, as shown by the mobbing behavior of the chaffinch (*Fringilla coelebs*). I. The nature of the response, and an examination of its course. *Proceedings of the Royal Society of London, Series B*, 1954, *142*, 306-331. (a)

Hinde, R. A. Factors governing the changes in strength of a partially inborn response, as shown by the mobbing behavior of the chaffinch (*Fringilla coelebs*). II. The waning of the response. *Proceedings of the Royal Society of London, Series B*, 1954, *142*, 331-358. (b)

Hinde, R. A. Factors governing the changes in strength of a partially inborn response, as shown by the mobbing behavior of the chaffinch (*Fringilla coelebs*). III. The interaction of short-term and long-term incremental and decremental effects. *Proceedings of the Royal Society of London, Series B*, 1960, *153*, 398-420.

Ingvar, D. H., & Lassen, N. A. (eds.) *Brain work: Benzon Symposium VIII*. Copenhagen: Munksgaard, 1975.

Jackson, J. H. *Clinical and physiological researches on the nervous system*. London: J. & A. Churchill, 1873.

James, W. *Principles of psychology*, Vols. I and II (compl.). New York: Dover Publications, 1890.

Jones, E. G. The anatomy of extrageniculostriate visual mechanisms. In F. O. Schmitt & F. G. Worden (eds.) *The neurosciences third study program*, pp 215-227. Cambridge, Mass.: The MIT Press, 1973.

Kantor, J. R., & Smith, N. W. *The science of psychology: An interbehavioral survey*. Chicago: Principia Press, 1975.

Kemp, J. M., & Powell, T. P. S. The cortico-striate projection in the monkey. *Brain*, 1970, *93*, 525-546.

Kimble, D. P., Bagshaw, M. H., & Pribram, K. H. The GSR of monkeys during orienting and habituation after selective partial ablations of the cingulate and frontal cortex. *Neuropsychologia*, 1965, *3*, 121-128.

King, M. B., & Hoebel, B. G. Killing elicited by brain stimulation in rats. *Communications in Behavioral Biology*, Part A., 1968, *2*, 173-177.

Krasne, F. B. General disruption resulting from electical stimulation of ventro-medial hypothalamus. *Science*, 1962, *138*, 822-823.

Kruger, L., & Michel, F. A single neuron analysis of buccal cavity representation in the sensory trigeminal complex of the cat. *Archives of Oral Biology*, 1962, *7*, 491-503.

Lacey, B. C., & Lacey, J. I. Studies of heart rate and other bodily processes in sensorimotor behavior. In P. A. Obrist, A. Black, J. Bruner, & L. DiCara (eds.), *Cardiovascular psychophysiology: Current issues in response mechanisms, biofeedback and methodology*, pp. 538-564. Chicago: Aldine-Atherton, 1974.

Lange, C. *Ueber Gemuthsbewegungen*. Leipzig, Germany: Uebersetzt von H. Kurell, 1887.

Lassonde, M. C., & Ptito, M. Intracerebral influences on the microstructure of visual cortex. II. Striatal and cortical effects on response properties of visual cortex neurons. (Submitted to Journal of Neurophysiology, June, 1979).

Lassonde, M. C., Ptito, M., & Pribram, K. H. Are the basal ganglia only motor structures? In *Programs and Abstracts*, Washington, D.C.: American Physiological Society, 1975.

Liebeskind, J. C., Mayer, D. J., & Akil, H. Central mechanisms of pain inhibition: Studies of analgesia from focal brain stimulation. In J. J. Bonica (ed.), *Advances in neurology*, Vol. 4 *Pain*. New York: Raven Press, 1974.

Lindsley, D. B. Emotion. In S. S. Stevens (ed.), *Handbook of experimental psychology*. New York: Wiley, 1951.

MacLean, P. D. Psychosomatic disease and the "visceral pain": Recent developments bearing on the Papez theory of emotion. *Psychosomatic Medicine*, 1949, *44*, 338-353.

Malmo, R. B. On central and autonomic nervous system mechanisms in conditioning, learning and performance. *Canadian Journal of Psychology*, 1963, *17*, 1.

Mandler, G. The interruption of behavior. In D. Levine (ed.), *Nebraska Symposium on Motivation*, pp. 163–220. Lincoln: University of Nebraska Press, 1964.

McEwen, B. S., Gerlach, J. L., & Micco, D. J. Putative glucocorticoid receptors in hippocampus and other regions of the rat brain. In R. L. Isaacson & K. H. Pribram, (eds.), *The hippocampus*, pp. 285–322. New York: Plenum, 1976.

Miller, G. A., Galanter, E. H., & Pribram, K. H. *Plans and the structure of behavior*. New York: Henry Holt, 1960.

Miller, N. E., Bailey, C. J., & Stevenson, J. A. Decreased "hunger" but increased food intake resulting from hypothalamic lesions. *Science*, 1950, *112*, 256–259.

Mountcastle, V. B., Lynch, J. C., Georgopoulos, A., Sakata, H., & Acuna, C. Posterior parietal association cortex of the monkey: Command functions for operations within extrapersonal space. *Journal of Neurophysiology*, 1975, *38*, 871–908.

Nauta, W. J. H. Some efferent connections of the prefrontal cortex in the monkey. In J. M. Warren & K. Akert (eds.), *The frontal granular cortex and behavior*, p. 397. New York: McGraw-Hill, 1964.

Neff, D. Neural mechanisms of auditory discrimination. In W. A. Rosenbligh (ed.), *Sensory communication*, pp. 259–278. New York: Wiley, 1961.

Papez, J. W. A proposed mechanism of emotion. *Archives of Neurology and Psychiatry of Chicago*, 1937, *38*, 725–743.

Phoenix, C. H., Goy, R. W., & Young, W. C. Sexual behavior: General aspects. In L. Martini & W. F. Ganong (eds.), *Neuroendocrinology*, Vol. 2, pp. 163–196. New York: Academic Press, 1967.

Pribram, K. H. Toward a science of neuropsychology (method and data). In R. A. Patton (ed.) *Current trends in psychology and the behavioral sciences*, pp. 115–142. Pittsburgh: University of Pittsburgh Press, 1954.

Pribram, K. H. Neocortical function in behavior. In H. F. Harlow & C. H. Woolsey (eds.), *Biological and biochemical bases of behavior*, pp. 151–172. Madison: University of Wisconsin Press, 1958. (a)

Pribram, K. H. Comparative neurology and the evolution of behavior. In A. Roe & G. G. Simpson (eds.) *Behavior and evolution*, pp. 140–164. New Haven: Yale University Press, 1958. (b)

Pribram, K. H. The new neurology and the biology of emotion: A structural approach. *American Psychologist*, 1967, *22*, 830–838.

Pribram, K. H. Mind, brain and behavior: Excursions into paradox. *Psychological Scene*, 1968, *2*, 30–33.

Pribram, K. H. Neural servosystems and the structure of personality. *Journal of Nervous and Mental Diseases* (Kubie issue), 1969, *140*, 30–39.

Pribram, K. H. Feelings as monitors. In M. B. Arnold (ed.), *Feelings and emotions*, pp. 41–53. New York: Academic Press, 1970.

Pribram, K. H. *Languages of the brain: Experimental paradoxes and principles in neuropsychology*. Englewood Cliffs, N.J.: Prentice-Hall, 1971. (2nd ed. Monterey, Calif.: Brooks/Cole, 1977).

Pribram, K. H. How is it that sensing so much we can do so little? In F. O. Schmitt (ed.) *The neurosciences third study program*, pp 249–261. Cambridge, Mass.: The MIT Press, 1974.

Pribram, K. H. Peptides and protocritic processes. In L. H. Miller, C. A. Sandman, & A. J. Kastin (eds.), *Neuropeptide influences on the brain and behavior*. New York: Raven Press, 1977. (a)

Pribram, K. H. New dimensions in the functions of the basal ganglia. In C. Shagass, S. Gershon, & A. J. Freidhoff (eds.), *Psychopathology and brain dysfunction*, pp. 77–95. New York: Raven Press, 1977 (b)

Pribram, K. H. Modes of central processing in human learning. In T. Teyler (ed.), *Brain and learning*, Stamford, Conn.: Greylock, 1978.

Pribram, K. H., & Bagshaw, M. Further analysis of the temporal lobe syndrome utilizing fronto-temporal ablations. *J. Comparative Neurology*, 1953, *99*: 347-375.

Pribram, K. H., & Barry, J. Further behavioral analysis of the parieto-temporo-preoccipital cortex. *Journal of Neurophysiology*, 1956, *99*, 99-106.

Pribram, K. H., & Gill, M. M. *Freud's "project" reassessed*. London: Hutchinson, 1976; New York: Basic Books, 1976.

Pribram, K. H., Kruger, L., Robinston, F., & Berman, A. J. The effects of precentral lesions on the behavior of monkeys. *Yale Journal of Biology and Medicine*, 1956, *28*, 428-443.

Pribram, K. H., Lassonde, M. C., & Ptito, M. Intracerebral influences on the microstructure of visual cortex: I. Classification of receptive field properties. (submitted to the Journal of Neurophysiology, June, 1979).

Pribram, K. H., & MacLean, P. D. Neuronographic analysis of medial and basal cerebral cortex. II. Monkey. *Journal of Neurophsyiology*, 1953, *16*, 324-340.

Pribram, K. H., & McGuinness, D. Arousal, activation and effort in the control of attention. *Psychology Review*, 1975, *82*(2), 116-149.

Pribram, K. H., & Melges, F. T. Psychophysiological basis of emotion. In P. J. Vinken & G. W. Bruyn (eds.), *Handbook of clinical neurology*, Vol. 3. Amsterdam: North Holland, 1969.

Pribram, K. H., Reitz, S., McNeil, M., & Spevack, A. A. The effect of amygdalectomy on orienting and classical conditioning. In *Mechanisms of formation and inhibition of conditional reflex* (Asratyan Festschrift). Moscow: Publishing office "Nauka" of the U.S.S.R. Academy of Sciences, 1974.

Reitz, S. L., & Pribram, K. H. Some subcortical connections of the inferotemporal gyrus of monkey. *Experimental Neurology*, 1969, *25*, 632-645.

Richardson, D. E., & Akil, H. Chronic self-administration of brain stimulation for pain relief in human patients. In *Proceedings of the American Association of Neurological Surgeons*. St. Louis, Mo.: AANS, 1974.

Riezen, van H., Rigter, H., & Greven, H. M. Critical appraisal of peptide pharmacology. In L. H. Miller, C. A. Sandman, & A. J. Kastin (eds.), *Neuropeptide influences on the brain and behavior*, pp. 11-27. New York: Raven Press, 1977.

Schachter, S., & Singer, T. E. Cognitive, social and physiological determinants of emotional state. *Psychology Review*, 1962, *69*, 379-397.

Schwartz, G. E. Biofeedback, self-regulation and the patterning of physiological processes. *American Scientist*, 1975, *63*(3), 314-324.

Sokolov, E. N. Neuronal models and the orienting reflex. In M. A. B. Brazier (ed.), *The central nervous system and behavior*. New York: Josiah Macy, Jr. Foundation, 1960.

Sperry, R. W. Lateral specialization in the surgically separated hemispheres. In F. O. Schmitt & F. G. Worden (eds.), *The neurosciences third study program*, pp. 5-19. Cambridge, Mass.: The MIT Press, 1974.

Spinelli, D. N., & Pribram, K. H. Changes in visual recovery functions produced by temporal lobe stimulation in monkeys. *Electroencephalography and Clinical Neurophysiology*, 1966, *20*, 44-49.

Spinelli, D. N., & Pribram, K. H. Changes in visual recovery function and unit activity produced by frontal and temporal cortex stimulation. *Electroencephalography and Clinical Neurophysiology*, 1967, *22*, 143-149.

Stein, L. Reward transmitters: Catecholamines and opioid peptides. In M. A. Lipton, A. DiMascio, & K. R. Killam (eds.), *Psychopharmacology: A generation of progress*. New York: Raven Press, 1978.

Teitelbaum, P. Sensory control of hypothalamic hyperphagia. *Journal of Comparative Physiological Psychology*, 1965, *59*, 275-279.

Teuber, H. L. Unity and diversity of frontal lobe functions. *Acta Neurobiologiae Experimentalis*, 1972, *32*(2), 615-656.

Vernikos-Danellis, J. Effects of hormones on the central nervous system. In S. Levine (ed.), *Hormones and behavior*, pp. 13-14. New York: Academic Press, 1972. (a)

Vernikos-Danellis, J. Effects of hormones on the central nervous system. In S. Levine (ed.), *Hormones and behavior*, pp. 29-39. New York: Academic Press, 1972. (b)

Walter, W. G. Electrical signs of association, expectancy, and decision in the human brain. *Electroencephalography and Clinical Neurophysiology*, 1967, *25*, 258-263.

Weiskrantz, L. The interaction between occipital and temporal cortex in vision: an overview. In F. O. Schmitt & F. G. Worden (eds.), *The neurosciences third study program*. pp. 189-204. Cambridge, Mass.: The MIT Press, 1974.

Wilson, M. Effects of circumscribed cortical lesions upon somesthetic and visual discrimination in the monkey. *J. Comparative Physiology and Psychology*, 1957, *50*, 630-635.

Young, P. T. *Emotion in man and animal, its nature and dynamic basis*. Huntington, New York: Robert E. Krieger, 1973.

Young, W. C. The hormones and mating behavior. In W. C. Young (ed.), *Sex and internal secretions*, pp. 1173-1239. Baltimore: Williams & Wilkins, 1961.