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1 Emotion: A Neurobehavioral Analysis

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Current scientific knowledge regarding the physiology of emotion has its roots in Galenical medicine. Four "humors"—sanguine, choleric, phlegmatic and melancholic—were considered to determine differences in temperament. The humors were believed to be organ secretions, and modern biomedical research has supplemented these primitives with a host of endocrine and exocrine hormones that even today must be seriously considered in any comprehensive treatment of the biological regulations that determine feelings of emotion and motivation.

In addition to the multiplication and specification of humors, several other major developements have occurred in the scientific study of the biology of emotions. The first of these developments points to the role of nonhumoral mechanisms in the emotional process: Lange's "visceral" theory made famous by William James (1890), and Nina Bull's "muscle" based attitude theory (1951) are probably the most important of these. Furthermore, brain mechanisms have been shown to be central to understanding emotions. 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 19th century, and achieved considerable sophistication by its end.

Currently, these developments have become enshrined in two central themes that can be identified in practically all biological approaches to emotion: One theme explores the relationship between visceral-glandular reactions and the brain in producing emotion; the other deals with the quantitative relationship between neural excitation and emotion. As we see later, these relationships, although substantial, by themselves form neither an adequate framework for understanding the complexities of emotional processes nor an outline for understanding the intricacies of the relevant neural apparatus. Nonetheless, they do

provide a familiar starting point for inquiry, and the basis for developing a more comprehensive view that encompasses the results of recent neurophysiological research.

A LABILE-STABILE DIMENSION

The Visceral Theme

The impact of the visceral theme has been great and 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 guy has no guts"; "he sure is bilious today," and so on. In fact, until 1800 A.D. the Galenic medical world subscribed to the notion that while thoughts circulate in the ventricles of the brain, emotions circulate in the vascular system. Gradually, medical and psychological science has become liberated from this view by the accrual of facts showing it to be in error. But the retreat has been a slow and guarded one, partly because old theories do not die easily, and partly because this view has hold of an important part of the truth. The most famous formulations that signal a step-wise retreat and liberation from this view are those of James and Lange, of Cannon and Bard, and of Papez and MacLean.

James and Lange fully faced the accumulated knowledge of the functions of the circulatory and nervous systems of the previous century. They offered the following postulates: 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 examinations of the James–Lange theory (1927) is paradigmatic in showing the theory's weaknesses: (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.

In place of the visceral theory, Cannon proposed a brain (thalamic) theory: emotional *expression* results from the operation of hypothalamic structures; emotion *feeling* results from stimulations of the dorsal thalamus. This theory was based on the observations that "sham," emotionlike behavior, could be elicited in decorticated and decerebrated preparations, but not when thalamic structures are additionally ablated (Bard & Rioch, 1937). Further, a variety of expressive and visceral responses were obtained when the thalamus was electrically stimulated (Von Bechterev, 1911). Finally, patients with unilateral lesions in the thalamic regions were described as excessively sensing what were to others

ordinary cutaneous stimulations. For example, a pin prick would elicit excruciating pain, warmth, intense delight, and so on (Head, 1920).

Probably more is known about the functions of these core thalamic portions of the brain than about any other. This stems in part from the fact that these mechanisms are relatively "peripheral" in the sense that they are relatively directly connected to the organism's receptor mechanisms. In fact, 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 control through feedback was termed "homeostasis" by Cannon and has proved to be a powerful conception in a variety of biological and engineering applications.

But of equal importance is the fact that the processes which are controlled by these mechanics are highly autonomous, that is, self-regulating. Visceral and endocrine regulation is performed with a light hand via two distinct portions of the autonomic nervous system, the sympathetic and the parasympathetic, which balance each other. Experimental evidence was accumulated, especially by Hess (1954), to demonstrate the existence in the hypothalamic region of a trophotropic, energy-conserving process, working primarily through the parasympathetic peripheral division of the autonomic nervous system, and an ergotrophic or mobilizing system, working through the sympathetic division.

The balance between ergo- and trophotropic is not static, of course. When tipped in one direction or the other, a temporary rebound or an "answering effect" (Fair, 1963) could occur as the balance was restored. And indeed both processes could be activated simultaneously so that they would, in effect, work additively. And this was not all. When such activation occurred, somatic, as well as visceral, musculature was 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 substrata of emotions, this assumption followed logically.

But Lashley (1960) tellingly criticized the evidence on 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 [p. 352]." He also raised the question of 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 [p. 351]." Lashley agrees 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 phenomena of hyperexcitability 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 [p. 348]." Clearly, the dissociation between emotional expression and feeling, which is such a common clinical and experimental observation, can be leveled against both the James–Lange and Cannon–Bard theories. Unfortunately, Lashley provided no alternative to the theories he so effectively deprecates.

Recently, the James–Lange and the Cannon–Bard views have been superceded by the one proposed by Papez (1937) and elaborated by MacLean (1950). 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 functions. With the development of modern techniques for electrical brain stimulation, viscera were shown to be under the surveillance of the cerebral cortex (Kaada, Pribram & Epstein, 1949). One portion of this cortex came into focus for special attention: the limbic portion of the forebrain. Papez (1937) suggested that the anatomical interconnections among limbic structures were ideally constituted to handle the long-lasting, intense aspects of experience which are usually associated with emotion. MacLean added to this idea the facts of the relationship between this part of the brain and viscera, thus suggesting that here at last is *the* visceral brain—the seat of emotions. The persuasive power of this suggestion is great: Galen, James and Lange, Cannon and Bard, are all saved; visceral processes are the basis of emotion; and an identifiable part of the brain is responsible for emotional control and experience because of its selective relations with viscera. James and Lange were wrong only in leaving out the brain; Cannon and Bard were wrong only in the part of the brain they had identified with emotion; the limbic forebrain, not the thalamus, is the responsible agent. The path from the "emotions in the vascular system" to "emotions in the forebrain" had finally been completed, and each step along the way freed us from preconceptions popularly current when the step was taken.

Despite its persuasiveness and still-present popularity, there are some important criticisms to be levied against the visceral brain theory of emotions. Just as the theory gains in power from its implicit acceptance of the James–Lange and Cannon–Bard views, so it falls heir to the criticisms leveled against the earlier theories. In the same way that the relationship between thalamic structures and emotion fails to be an exclusive one, so the relationship between limbic structures and viscera, or, for that matter, limbic structures and emotions fails to be exclusive. It has been demonstrated experimentally (Wall & Pribram, 1950) that other parts of the cerebral mantle, when electrically stimulated, also give rise to visceral response. Emotional changes are observed to accompany lesions in parts of the forebrain other than the limbic areas. Further, ablation and stimulation of limbic structures influence problem-solving (cognitive) behavior in selective ways that cannot be attributed to changes in emotion. In humans, in fact, a very

obvious and specific "memory" deficiency follows limbic lesions, while changes in "emotion" cannot be ascertained. Obviously, the Papez–MacLean theory, like its predecessors, has only a part of the problem in hand.

The Activation Theme

As one turns from the visceral to the activation theories, one can again distinguish between peripheral and central subtheories. Here, however, the argument has not been so sharp. Peripheralists have gladly accepted the diffuse nonspecific reticular activating system as the central locus on which and from which peripheral excitation focuses. And centralists, in turn, have been as concerned with the peripheral as with the central effects of adrenergic and cholinergic substances (e.g., Arnold, 1960). Activation theory can be said, on the whole, to be less specific, less controversial, and considerably more factually oriented than visceral theories (cf. Lindsley, 1951). For example, a classical visceral theorist would have to say that a certain amount of adrenocortical hormone circulating in the blood stream would be correlated with a specific pattern of peripheral and central neural response (in hypothalamus or visceral brain), which in turn corresponds to one or another of the varieties of emotional experience or expression. An activation theorist merely states 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.

However, 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 to the notion of quantitative continuity within each of these processes. Once more, the suggestion arises that activation theory, although part of the story, is not in itself the whole story.

A New Approach

A part of the difficulty comes from the view of activation as an elementary process opposed only to 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 ineractions. Also, disequilibration is often sudden, explosive, and has about it the feel of 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 provided by the process of habituation of the orienting reaction. Experimental evidence has accumulated in the past two decades (Sokolov, 1960) to show that habituation of orienting is not due to a progressive 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 configurations form the sum of an organism's expectancies. The evidence runs like this: A person is subjected to an irregular repetition of a sound stimulus of constant intensity, frequency, and duration. Initially, the person shows a set of physiological and behavioral reactions that together form the orienting response. Among these reactions is "cerebral activation"—that is, a desynchronization of the electrical rhythms recorded from the brain. As the repetition of the sound stimulus proceeds, less and less orienting takes place. This lessening of orienting is called "habituation." For many years it was thought to be due to a simple rise in threshold to input. But "dishabituation"—that is, a recrudescence of the orienting responses—occurs when the intensity of the sound stimulus is decreased or if the duration of sound is shortened. In this latter situation, the orienting reaction occurs at the offset of the stimulation—to the "unexpected" silence.

There can thus be no question about the configurational nature of activation. But these experiments, and the many everyday experiences that they confirm, also account for the importance of visceral and autonomic functions in providing the stable baseline from which the organism's reactions can take off.

Each interaction between environment and organism involves at least two components: (1) discrete interaction by way of the brain's sensory-mode specific classical projection systems and its core homeostats, and (2) a "nonspecific," relatively diffuse interaction by way of reticular and related formations. These nonspecific systems act as a bias on the specific reactions: the set point or value toward which a specific interaction tends to stabilize is determined by the nonspecific activity. Visceral feedback, by the nature of its receptor anatomy and diffuse afferent organization, constitutes a major source of input to this biasing mechanism; it is an input that can do much to determine set-point. 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. Therefore, habituation to visceral and autonomic activity makes up a large share, although by no means all, of the stable baseline from which the organism's reactions can take off.

Another major source of recurrent input that determines bias or sets the level at which change can be sensed is input from the somatic musculature and skin. These somesthetic and proprioceptive inputs give rise to baseline configurations

that have been conceptualized in terms such as "body image" and "perceptual-motor organization." Configurational changes in these inputs can also give rise to incongruities that disturb the stable baseline.

Whenever the reaction to incongruous input is sufficient to disturb these baselines, 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 discrepencies is the basis for the visceral theories of emotion.

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, overall 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 (Adey, Kado, & Didio, 1962; Li, Cullen, & Jasper 1956a, 1956b) that the slow graded activity of neural tissue, rather than the overall 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 the 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 limited by input. An input that may ordinarily be processed smoothly may perturb the system if that system is already unstable; or an internal change in the organism may initiate incongruity where match had previously existed. Thus, the configuration of activation of the nervous system 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 nonspecific neural systems are primarily involved in setting the bias toward which more specific organism-environmental 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 the important locus of action of the pharmacological tranquilizers and energizers that have been so successful an adjunct in altering maladaptive emotional reaction.

But these are not the only chemical influences at play. 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 constitutuents of the internal environ-

ment of the organism. A respiratory control 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. Peptides secreted by the walls of the gut and by the kidney and a host of others are being investigated because some experiments indicate that they too are sensed by cells in the core of the brain (see Pribram, 1971/1982, chaps. 9 & 10, for review).

Further, 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—adrenaline—secreted by the adrenal medulla) and dopamine (which metabolizes into norepinephrine); indole amines such as serotonin; and peptides such as endorphine (an endogenous morphine-like substance) abound. As might be expected, sensitivities to these neurohumors are also built into the mechanism.

As noted earlier, Walter Cannon, in his classical studies (1927), determined that the relationship between the sensor and its chemical was such that the concentration of the chemical, although 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 ways 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 the 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 both of these in turn are linked to osmoreceptors (the salt-water receptors) which control thirst. This set of linkages is additionally connected to the thyroid sensitive mechanism controlling activity. Through various metabolic interrelationships, such as breathing, that take place in the body these homeostatic mechanisms also regulate the partial pressure of CO_2, and so on (see Brobeck, 1963 for review).

In short, the core of the brain (mesencephalon, diencephalon, and the basal ganglia and limbic systems of the forebrain) uses chemical regulations to control body functions. The configuration of concentrations of these chemicals, although fluctuating around some set point, is sufficiently stable over periods of time to constitute steady "states." These states apparently are experienced as hunger, thirst, sleepiness, elation, depression, effort, comfort, and so on. 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 concentrations 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—that is, of elation and depression; the concentration of endorphins in those of temperature, novelty, and pain; and the concentrations of the enkephalins (adrenocorticotrophic hormones of the pituitary) in those of effort and comfort (for reviews, see Pribram, 1971/1982, 1977a; Stein, 1978).

AN EPICRITIC-PROTOCRITIC DIMENSION: BRAIN STEM SHELL AND CORE

Pain and Temperature as Protocritic Processes

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

The part of the skin components of temperature and pain sensitivity processed separately (in the parietal lobes of the cortex) from the homeostatic mechanism is characterized by what is called in neurology the "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 periqueductal grey of the midbrain, and the amygdala of the forebrain) 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 simply sensitive to amounts, to quantities, of chemical and neural excitation. Processing does not lead to identi-

fication 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 should 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. Chin et al. (1976) and Pribram (1977a) provide a more complete review.

The protocritic dimension of experience, devoid of epicritic local sign, is therefore characteristically dependent on the quantity, the intensity of the stimulus. Quantity [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, whereas 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. Brain stimulations in human beings that protect against pain are accompanied by the feeling of cold (Richardson & Akil, 1974). 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? 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?

Although there are no definitive answers to these questions yet, it has become clear that a host of neural systems become engaged in the rostral extensions of the pain and temperature mechanisms. At the brainstem level up to the forebrain, electrical excitations of these systems produce self-stimulation in animals and hedonic experiences in man. Closely intertwined, but perhaps more laterally placed, are locations from which electrical stimulations produce aversive effects—turning off the stimulus and, in the more caudal placements, evidence of discomfort in animals.

Arousal, Activation and Effort

In the forebrain, these systems focus on structures such as the basal ganglia and limbic formations about which we have a considerable amount of information regarding their relationship to emotion (and motivation). The evidence involved can be organized (detailed by Pribram & McGuinness, 1975) to show that three separate categories of systems can be discerned to influence electrocortical desynchronization—evidence that goes beyond that reviewed in the previous sec-

tion, of "The Activation Theme." One system regulates phasic desynchronization (i.e., brief, lasting at most several seconds), another tonic (on the order of minutes) desynchronization, while a third system coordinates the other two (over a longer period of time—the duration of an attention span).

Phasic desynchronization we called "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 (Bagshaw & Benzies, 1968; Bagshaw, Kimble, & Pribram, 1965; Kimble, Bagshaw, & Pribam, 1965; 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 that normally occurs when the novel stimulus is repeated. The visceroautonomic reaction appears necessary for familiarization with the stimulus to occur. 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 noted, Sokolov showed that (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 to 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 maintaining 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 (1951).

A third system centers on the hippocampus and coordinates arousal and readiness. 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 this function (Isaacson & Pribram, 1976). Coordination has been shown to involve neural work, that is, to take effort (Benson, 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 pituitary–adrenal hormonal controls, selectively absorbing adrenocortical hormone (Bohus, 1976; McEwen, Gerlach, & Micco, 1976) and being acted on by the adrenocorticotrophic hormone (ACTH) and related enkephalins (Riezen et al., 1977).

A Comprehensive Physiological Theory of Emotion

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 (or its complement, familiarity) appears 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 emotional feelings that emerges from these data centers on a set of corebrain neurochemical states that comprise the experience of "familiarity." Familiarity implies equilibrium, a feeling of reasonable amount of stability and 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 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 mechanisms, the maintenance of states is effected by tonic operations of the readiness system. As noted earlier, there is a considerable body of 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 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 smooth transition from state to state within some confortable band width of tolerance.

The data briefly noted in this section make it necessary to look carefully at another often neglected distinction. Ordinarily, we use the terms emotion and feelings synonymously. Feelings generated by readiness to respond are more akin to motivations and intentions than to emotions, however. The next section makes explicit therefore a distinction between emotions and motivations and the feelings that are generated by both.

AN EFFECTIVE–AFFECTIVE DIMENSION: BASAL GANGLIA AND LIMBIC FOREBRAIN

The Experiencing and Expressing of Emotions

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,

1971/1982) made the case for using the category "feelings" to encompass a range of experiences which 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 years 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 the 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 concepts. 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 of her voracious eating? The paradox is resolved if 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, & 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 produces fighting if the animal is provoked (King & Hoebel, 1968).

Grossman (1966) 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 that tend to starve), and the medial portions contain the "stop" mechanism, the interruption of behavior which we have identified as leading to emotional arousal.

The paradox is thus resolved by the hypothesis that processes ordinarily involved in taking the organism "out of motion" also generate affects or feelings of emotion. In this fashion, 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, 1971/1982 pp. 192–194).

Emotion and Motivation

These 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 foregoing quotation, arousal as in the orienting reaction to distracting stimuli, and more generally when behavior is interrupted (Mandler, 1964). By contrast, the organism is considered motivated when his readiness mechanisms are activated, when he is ready to "go" and to continue "going." These responses are (as noted in the previous section) critically organized by the basal ganglia (Pribram, 1977b) and have as their physiological indicators the CNV (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 (1890) 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 [p. 442]." In a similar fashion, J. R. Kantor (Kantor & Smith, 1975), 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 primarily with internal body mechanisms, while effective interactions generate readiness or overt responses toward the stimulating object.

In short, for behavior, as well as for the neurophysiology of feelings, it becomes useful to distinguish emotional from motivational antecedents. Moti-

vational 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, are 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."

The Expression of Emotions in a Social Context

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 behavior? Ordinarily, an experimentalist is concerned with the environmental consequences of behavior (e.g., the cumulative record in an operant situation). In this situation, 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 (internal) states (reactions that terminate at the skin) which 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.

Thus, emotional expression does have a practical influence beyond the emoting organism, but only in a social 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 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 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, 1954b, 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 (Miller, Galanter, & Pribram, 1960, Chap. 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 consequences of motivated behavior is a function of that behavior. The adaptive consequence of emotional expression is a function of the social matrix in which it occurs or of a stability attained in the evolutionary process, which eliminates the occurrence of the expression of nonadaptive situations.

In summary, *emotional behavior* is defined as an expression of positive and negative emotional feelings that are inferred to reflect 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 experiences and 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, although not always, highly adaptive. Note, however, that the adaptation is due not directly to the expression of emotion but to the forces operating in the social and physical environment. We have already dealt with these "triggering" stimuli for emotional expression: The protocritic dimension was seen to be critical. But what neural control mechanisms determine how an emotional feeling will be experienced?

AN ETHICAL-ESTHETIC DIMENSION

The Cortical Contribution to a Labeling of Feelings

The biological contribution to an understanding of feelings in general and emotional feelings in particular can therefore not rest here. A basic problem set out by William James and also by Freud has to be faced. Freud (1895/1966) proposed that the critical neurological mechanisms involved in emotion are neurochemical and derive from body stimulation (the endogenous paths), which affect a certain portion of the brain. The work reviewed here has given substance to Freud's proposal and enlarged 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. Further, the processing of this protocritic dimension was found to take place in limitec

portions of the brain—the core brain systems of the brain stem and the limbic forebrain.

William James (1890) faces 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 [pp. 472–474].

Thus, James opts for the cortex. Was he wrong? I do not believe so. There is more to feeling that the protocritic dimension. Schachter (Schachter & Singer, 1962) has delineated two aspects to feeling: one that devolves on its intensity and the other that "labels" the feeling. With regard to his intensity dimension, Schachter, using only adrenalin, was trapped by this unidimensional approach in the activation theme current at the time he performed his experiments. As previously detailed, a richer protocritic dimensionality better describes this aspect of his experimental results. But Schachter's data do provide evidence for another set of dimensions, those that determine whether an emotional or motivational feeling or expression is "good" or "bad." Such evaluative labels are specific; they identify the feeling with respect to a qualitative context. In short, labeling can encompass epicritic as well as protocritic factors, and we should turn, as James proposed, to the cerebral convexity in the search for the neural mechanisms that are involved.

According to James (1890b), 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 [pp. 472–474]."

Review of the foregoing work 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 perturbance) be addressed. The pathways whereby this can occur have now been thoroughly established both anatomically and physiologically (Gold-

man & Nauta, 1977; Kemp & Powell, 1970; Lassonde, Ptito, & Pribram, 1981; Nauta, 1964; Reitz & Pribram, 1969).

Personal and Extrapersonal Processes

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 excitation 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 that are regulated by the more primitive structures.

The far frontal cortex receives abundant connections from both the limbic and basal ganglia systems (Pribram, 1954, 1958a). Anatomically the far frontal (frontal intrinsic) cortex receives projections from the n. medialis dorsalis of the thalamus (an "intrinsic" nucleus, since it is only indirectly connected with extracerebral inputs), which lies embedded within nuclei that project to limbic cortex. Behaviorally, resections of far frontal cortex result in deficits in delayed alternation performance, deficits also obtained when lesions are made of limbic and basal ganglia (caudate) structures but not when the posterior cortical convexity is damaged. By contrast, damage to regions located in the posterior cortical convexity (the posterior intrinsic cortex that receives its input from the pulvinar, another intrinsic thalamic nucleus) produces deficits in discrimination learning and performance, which remain unaffected by frontal, limbic and caudate lesions. The difference between alternation and discrimination has been conceptualized to reflect the difference between context-sensitive reactions on the one hand, and context-free information processing on the other (Pribram, 1978).

The far frontal cortex thus operates to combine the protocritic with the effective poles of the protocritic–epicritic and affective–effective dimensions. This combination produces one pole of a new dimension that I have labeled esthetic–ethical (Pribram, 1967), based on the distinction between the processing of "external space" and the processing of a "body image" or "self." Processing that 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 perirolandic and periocalcarine 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 perisylvian and perirolandic 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 divisions:

1. the far frontal cortex discussed earlier which is interconnected by the fibers of the uncinate faciulus to the adjacent orbitofrontal, anterior insular and periamygdaloid cortex of the temporal pole, which are a part of the limbic system.

2. a posterior division focused on the inferior parietal lobule on the lateral surface and the precuneus on the medial which is connected to the inferior parietal lobule via the medial extension of the confluence between intraparietal and lunate sulci. The functional connectivities of these divisions of the cortical mantle have been most clearly demonstrated by strychnine neuronography (Pribram & MacLean, 1953; Von Bonin & Bailey, 1947) 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 (1956) showed that the environmental consequences of movement, not movements or muscle constructions per se, are encoded in this "motor" cortex (see review by Pribram, 1971/1982). (2) The postcentral and superior parietal cortex deals with the somatosensory (haptic) discrimination of objects in external space (Brody & Pribram, 1978; Kruger & Michel, 1962; Mountcastle, Lynch, Georgopoulos, Sakat, & Acuna, 1975; Pribram & Barry, 1956; Wilson, 1957). (3) The pericalcarine cortex deals with visual processing (see Weiskrantz, 1973, 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 Neff, 1961, pp. 259–278 for review) and its extension into the superior temporal gyrus with auditory discriminations (Dewson, 1977; Dewson & Cowey, 1969; Dewson, Pribram, & 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 & Pribram, 1953; Pribram & Bagshaw, 1953) and is also involved in olfactory (Brown, 1963; Brown, Rosvold, & Mishkin, 1963), and, as noted earlier, temperature discriminations (Chin, Pribram, Drake, & Green, 1976).

By contrast to these clear-cut results of experiments relating the perisfissural 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 far frontal cortex as the sole source of an image of self. Recent experimental results show, however, that this conclusion was oversimplified and to a large extent erroneous (Brody & 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 (Pribram & 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 far frontal and temporal polar 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 (Geschwind, 1965; Teuber, 1972). Patients with far frontal and temporal lobe involvement tend to talk and write voluminously about themselves and, as noted, to lose control over behavior that is context-sensitive, that is, behavior which depends on some stable mnemonically organized self. These observations fit the suggestion made earlier on anatomical grounds that the far frontal and temporal cortex operates to combine the protocritic and effective poles of the respective dimensions.

The cortical contribution to emotion thus 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 human beings by combining a frontolimbic protocritic versus cortical convexity epicritic axis with limbic affective versus a basal ganglia effective axis. The poles of each axis have been found to oppose each other (Lassonde, Ptito, & Pribram, 1981; Pribram, Lassonde, & Ptito, 1981; Spinelli & Pribram, 1967) in such a way that a combinatorial balance of control is achieved (Jackson, 1873).

CONCLUSION

Current physiological data support a multidimensional view of the organization of emotional and motivational feelings and expressions. A *labile-stabile dimension* is discerned as operating by virtue of corebrain homeostatic feedback mechanisms. However, homeostats become biased by brain stem activation. This results in mechanisms which can be reset by a variety of internal and external contingencies.

Neither the homeostats nor the mechanisms that determine their setpoints are unimodal. A variety of emotional and motivational processes (e.g., elation, depression, hunger, thirst) have been shown to be intimately related to specifyable neurochemical organizations. This is a very active field of investigation which holds that the neurochemistry of many more feeling states will be specified.

The determination of setpoints has been shown to be guided by at least three distinct processes: phasic arousal, chronic activation, and tonic effort. The mech-

anisms involved are located in the forebrain; all of them regulate and are regulated by structures which compose the territory of the mesencephalic reticular formation.

The forebrain arousal, activation and effort mechanisms which determine setpoints on homeostatic regulations are intimately connected, as well, with spinal and brainstem pain and temperature systems. Sensory processes can be divided into those which display local sign (can be located in time and place) and those which do not. Following Henry Head (1920), those which display local sign are called epicritic. These sensory processes have been traced to the parieto-temporo-occipital portion, i.e., the posterior convexity of the cerebral hemisphere.

By contrast, those aspects of pain and temperature sensibility (and perhaps these same aspects of other senses) which do not display local sign, have been traced to the far frontal and temporal polar portions of the cerebral hemispheres. In a slight modification of Head's terminology, these aspects of sensation are called protocritic.

Thus, the second dimension of the organization of emotional and motivational feelings and expression is the *epicritic–protocritic*. As in the case of the labile–stabile dimension, the epicritic–protocritic dimension applies to the variety of specific emotional and motivational feelings and expressions.

A third dimension is embodied in the distinction between emotion and motivation, between feelings and expressions, and between arousal and activation. This dimension is an *affective–effective dimension*. Neurologically it is manifest in the distinction between limbic (including the amygdala, n. accumbeus and mesolimbic systems) forebrain and the caudate nucleus of the basal ganglia.

Finally, at the cortical level, still another dimension is introduced. I have called this an *esthetic–ethical dimension* but a somewhat more descriptive term would be a dimension which at one pole constructs a world beyond the skin and at the other, a self-reflective world within. The construction of the external pole of this dimension is based on the cortical systems composing and surrounding the major cerebral fissures. Its internal pole is based on the cortex that lies between the perifissural cortex with strong connections to the limbic forebrain.

The identification by neurophysiological and neuropsychological methods of these dimensions still leaves unexplained how it is that we can distinguish anger from fear, acceptance from rejection, joy from sadness, and each of these pairs from one another. Schachter and others have suggested that environmental context determines the labels that we place on some more basic physiological processes. But, as noted, Schachter (1967), by using only adrenalin in his experiments, came to focus on only one such basic process, one which determines the intensity of experience. Others, e.g., Tomkins (this volume) and Koestler (1967) have also called attention to the amplifying dimension which emotions and motivations exercise. But as we have seen this amplifying, intensive aspect of emotions and motivations, subsumed here under the rubric "protocritic," is only one of a set of dimensions that characterize these processes.

A more promising lead (which also takes situation into account) for relating the dimensions identified by neuroscience research to the nuances of feelings comes from the work of Plutchik, Tomkins, Ekman and others (this volume) who have used scaling techniques and facial expression in an attempt to classify the indicators of feelings and to relate this classification to the psychophysiological variables (such as GSR and heart rate) which were also used in the neuroscience studies reviewed here. Plutchik, for example, has identified four processes basic to his classification: (1) control–dyscontrol; (2) toward–away; (3) gain–loss; and (4) in–out. It is tempting to identify the control–dyscontrol process with the stabile–labile dimension; the toward–away process with the effective–affective dimension; the gain–loss process with the protocritic–epicritic dimension; and the in–out process with the ethical–esthetic dimension. Ekman is currently linking facial expression of specific emotional and motivational experiences to *patterns* of psychophysiological indicators. This research is in a position to validate the identity between Plutchik's processes (derived from factoring the reports of specific emotional and motivational experiences) and the neurologically based dimensions described in this chapter (which were derived by relating manipulations of neural systems to patterns of psychophysiological indicators). Should such a convergence of results of different research programs materialize a major step will have been achieved in understanding the physiology of the emotions and motivations which so enrich our personal and interpersonal lives.

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