Forebrain psychophysiology of feelings: interest and involvement

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1. Introduction

The current presentation stems from two apparent paradoxes that must still be addressed by any comprehensive theory of emotion. One concerns the history of psychophysiological research: experiments undertaken to study the relationship of brain systems to emotional responses have more often than not told us more about types of attention than about types of emotions. The other paradox is that the limbic forebrain is commonly referred to as the neural substrate of emotions, yet massive lesions in both non-human primates and in humans fail to impair emotional experiences or expressions.

The history of psychophysiological research has been summarized in a series of papers by McGuinness and Pribram from 1975 to 1992 (McGuinness and Pribram, 1977, 1980). In these papers we resolved the paradox (emotion/attention) by reviewing the evidence regarding the motivational and emotional controls on attention. In Languages of the Brain (1971), I labeled the complex emotion-motivation/attention 'interest'. In view of the second paradox, it is timely to review the experimental results that led to these conclusions as much of this work is no longer acknowledged.

The second paradox concerns the almost universal belief that the limbic forebrain mediates emotional experience and expression. This belief stems from the observations that electrical stimulation of some limbic structures such as the amyg-
dala produce viscero-autonomic responses (Kaada et al., 1949) presumably supporting the James–Lange theory of the biological substrate of emotional experience. The paradox arises when emotions remain intact after massive limbic lesions that grossly impair episodic memory (while sparing semantic memory). Such observations have been made on H.M. and, more recently, on children with large medial forebrain cysts that resulted in complete loss of episodic memory while sparing semantic memory.

Another reason for the paradox is that electrical stimulation of certain limbic structures do produces a continuum from arousal to rage. And resections in non-human primates change their feeding, fighting, fleeing and sexual behavior (the 4 Fs). But these changes were shown to be part of a larger syndrome based on the disruption of the familiarization process.

In this, as in previous essays (Pribram, 1970), I will use the term feelings, a term often used synonymously with emotion. This provides the opportunity to distinguish emotional from motivational feelings, emotions as relating to internal coping while motivations go beyond the skin to provide practical involvement with the organism’s environment. On this basis, a resolution of the paradoxes can be attained by subsuming the feelings generated by emotions and motivations under some larger categories such as ‘interest’ and ‘involvement’ and to discern whether the brain systems coordinate with these larger categories are the ones popularly assigned to a more restricted role in governing emotion and motivation.

2. Historical perspective

Some two decades before I wrote the quotation introducing this essay, 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 is certainly an up and down person—so moody’. ‘He’s completely hung up on death; it would help him if he were not so emotional about it’. ‘Are not 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. Today’s scientists have attained a considerable body of evidence about the regulation of these states.

Current scientific knowledge regarding emotion has it 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. Modern biomedical research has supplanted these primitives with a host of endocrine hormones. The hormones must, of course, be seriously considered in any comprehensive treatment of the biological regulations that determine emotions.

Two other major developments have occurred in the scientific study of the biology of emotions. One of these developments points to the role of non-humoral mechanisms in the emotional process: One is Lange’s (1887) ‘visceral’ theory, made famous by James (1890). The other is Nina Bull’s (1951) ‘muscle’-based attitude theory, which is probably the most neglected and important of these.

The second major development is the realization that the brain is involved in the experience and expression of emotions. This realization 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, 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 predetermined channels, alter the condition of muscle, skin, and viscera; 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 centers admitted in one shape or another by all to exist (Vol. II, pp. 472–74).

Freud (1895/1966); see also Pribram and Gill, 1976) 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' neurons 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 consist of chemical products, of which there may be a considerable number (S.E., pp. 320–321). 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 that 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 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 DeValois (1960) shows us that cells in the lateral geniculate nucleus reflect an opponent process which accounts for otherwise inexplicable perceptual phenomena (Hering, 1964) (according to the Hering theory, 1964). More recently, DeValois and DeValois (1988) were able, on the basis of further experimental results, to provide a comprehensive theory of color and figural visual perception.

In a similar manner, a theory of what is loosely called emotion must begin by specifying the manner in which emotional behavior differs from other behavior, the stimulus dimensions that elicit such behavior, and the brain systems that process these dimensions. During the decades since the introductory statement was written a great deal of excellent experimental work and theory building has begun to fulfill these requirements.

3. Recent decades

In this essay I will mention only a few of the most comprehensive of these formulations and indicate consonances or dissonances with my own. Then I can go on to review the relevant work from my laboratory that, with other work of the 1950s, 1960s and 1970s has somehow slipped out of current awareness. Here I will indicate findings of that period that are important to current formulations, then review the body of evidence and theory that has led to the paradoxes that must be now resolved.

Edmund Rolls (Rolls, 1999) has published a comprehensive review of the work of his laboratory in the context what other research has produced. In conjunction with Rolls and Treves, 1998 he has developed a neural network model that shows how the theory he has drawn from his work can be described quantitatively in computational and mathematical terms. The wealth of experimental findings which he reviews is impressive as is the theoretical formulation he espouses. This formulation is based on instrumental conditioning of response initiated stimuli that increase or decrease the probability that further similar responses will be made by the organism. Those stimuli that increase that probability are termed positive reinforcers and those that decrease the probability are negative reinforcers. Positive and negative are values that comprise emotions and motivations.
Roll's formulations fit my own but the work performed in my laboratory went a step further in one direction. Bob Douglas, Abe Spevack and I (Douglas and Pribram, 1966; Spevak and Pribram, 1973) showed that while the amygdala is concerned in the processing of reinforcers, the hippocampus is concerned with processing the non-reinforced aspects of a situation that forms the context within which reinforcers operate. This was confirmed by showing that the electrical activity of the hippocampus reflected the contextual, nonreinforced aspects of a task.

Further, using a utility formulation derived from Von Neuman and Morgenstern, I showed (Pribram, 1957, 1998) that desirabilities (reinforcers) are processed by the amygdala system while the probabilities (entailed in a fixed interval schedule) are processed by the prefrontal cortex. Mathematical models of these findings are delineated in the appendices of my book on Brain and Perception (1991). These findings and theoretical formulations flesh out the theory proposed by Rolls.

Le Doux (1996) takes a classical (rather than instrumental) conditioning approach to the study of the brain systems involved in emotional behavior. Again, work in my laboratory (Bagshaw and Coppock, 1968; Pribram et al., 1974) has something to add to his excellently carried out program. Normal monkeys distribute their responses to a to-be conditioned (the conditional) stimulus. Amygdalectomized monkeys have a much closer time frame for such responses and the coincidence between unconditional and conditional responses are rare. Thus Hebbian S–S conditioning fails to occur. The distribution of timing rather than the value of the unconditional stimulus is responsible.

Along the same lines, an incidental observation made in an avoidance conditioning experiment. Monkeys after limbic and frontal resections of various kinds (Pribram and Weiskrantz, 1957) fail to avoid the alerting stimulus. In our avoidance situation (for good reason) we eliminated the sliding door between compartments and got good avoidance conditioning nonetheless. After amygdalectomy, the monkeys responded to the conditional stimulus by sitting on the barrier between the compartments and reached over to with their hands to test which compartment contained the unconditional stimulus. Le Doux (2002) is correct in questioning whether the avoidance paradigm really tests ‘fear’. Weiskrantz, Mishkin and I, debated often whether we should use escape as a sign of pain, avoidance, a sign of fear, and conditioned suppression as a sign of anxiety. In those behaviorist days we refrained and I am happy we did. But, in the light of our experiment, is the disruption of classical conditioning a sure sign of fear conditioning as Le Doux (1996) holds?

This brings me to question the current popularity of classifying the emotions portrayed by faces. With simple cartoons this works. But when I have shown some faces that presumably portray fear, most responders have reacted with ‘how weird’. Interpreting facial expression is not a simple matter. Children who have seen the faces of their mothers during coitus have become severely frightened, thinking the father is violently attacking. I have asked students in class whether they are in pain on the basis of their facial expression: the answer has invariably been surprise and ‘oh no, just thinking’.

The problem is that labeling emotions and emotional expression is a learned, cognitive-like process. Thus, social agreement as to labels may override some deeper understanding of the ‘actual’ situation producing the facial expression. If everyone agrees that Le Doux’s rats are ‘really’ afraid of the unconditional stimulus once they have been conditioned, then perhaps so be it. But there should be some lingering doubt that the normal rats are just smarter (are able to ‘learn’) the S–S conditional association—which the amygdalectomized rats are unable to do, for reasons that have little to do with fear. The responses of my monkeys have alerted me to such a possibility, and the fact that classical conditioning to food in my experiments was also impaired, support such a conjecture. I do not really want to attribute to my monkeys that they have developed an ‘appetite’ for the conditional stimulus.

With respect to food conditioning, Le Doux (2002) points out that the amygdala does other things than to make fear conditioning possible. He attributes this to the involvement of other parts of the amygdala. Without denying this, my interest has been to subsume the essence of all amygdala
processing (aversive and appetitive) under a larger umbrella such as an intensive dimension based on the habituation (familiarization) to a novel stimulus. But this is the burden of my argument and makes up the body of this essay.

This is not to deny the importance of visceral processing in the habituation of novelty. Kaada et al. (1949), described a mediobasal motor cortex devoted to visceral and autonomic regulation and Fulton et al. (1949), showed the intimate relationship of this cortex to hypothalamic nuclei. Porges (2001) has recently developed a vagotonic theory of social processing. Bailey and Bremer (1938) showed that vagal afferents reach the region of the amygdala (and orbitofrontal cortex) a finding confirmed by Paul MacLean and myself (unpublished). As Vinegradova (personal communication) once put it, in learning, an ounce of emotion is worth a pound of repetition.

Frijda (1986) as well as Oatley (Oatley and Jenkins, 1996; Oatley and Johnson-Laird, 1987) have provided theoretical frames consonant with those reviewed so far. For instance, Oatley and Johnston-Laird discuss evaluation as an event relevant to a concern that is important to an organism. A positive emotion occurs when the concern is advanced, a negative emotion occurs when the concern is impeded.

By contrast, Damasio (1994) has emphasized somatic markers, not as reinforcers of behavior but as essential components of feelings. In a 1970 paper entitled ‘Feelings as Monitors’ (Pribram, 1970) I make a similar point but restrict the monitoring to changes in brain representations of somatic signals. Damasio, as do Rolls, LeDoux and I, distinguishes between emotional and motivational feelings. I differ from the others in that they consider emotions as preludes to motivation whereas I consider them separate, within the body, coping processes that can, but need not lead to motivations. Antonio and Hannah Damasio and their group (e.g. Damasio et al., 1991; Bechara et al., 2002) are showing in humans much of what my laboratory showed in monkeys and have taken the monkey (reviewed below) results several steps forward.

However, I do differ from Damasio in his presumed anti-Cartesian stance. I say presumed because Damasio speaks of movies in the brain which are not altogether different from what Dennett (1991) has called the Cartesian Theater. But that is not the point. Damasio argues that feelings and their unconscious counterpart are constantly informed by stimuli from the body. The work from my laboratory reviewed below indicates that this is not so. Signals from the body build in the brain an apperceptive mass, a neural model, a representation of somatic and visceral input and it is only when there are changes from the homeorhetic steady state, that feelings become informed.

The clinic furnishes ample evidence that only such changes are felt. Phantom limbs are a case in point. Early on after an amputation injecting the stump with analgesics will relieve the phantom symptoms. If left undisturbed for several weeks, injections of the stump no longer work, and the effective site has moved to the cauda equina. Some months or years later even such injections no longer work: cordotomy of the ascending pain and temperature tracts in the spinal cord is necessary. Finally, still later, the only effective treatment is a frontal leucotomy.

The same course can be seen in testing monkeys: for instance in the delayed response procedure. At first the animals will use external cues such as pacing along the side of the cage where the reward is hidden. As training progresses these external crutches fade away and behavior is guided by their brain representations. Learning to play a piano piece follows a similar course: at first the score is necessary, later the score can be dispensed with.

I have puzzled and written, and continue to write about just where to place what is now called the Cartesian cut. Briefly: I use computer programming as a metaphor. Machine language is isomorphic with the operations of the computer. But any higher order languages (even hexadecimal) are transformations of that isomorphism until the word processing language (which is isomorphic with my natural language) is so different from the operations of the computer that one can excuse someone (such as DesCartes) for becoming a machine vs. language (thinking) dualist. Of course there is no ‘language substance’ etc. I cover my views by
being epistemologically pluralistic and ontologically monistic. (Pribram, 1986, 1997).

4. Emotional behavior and the experience of changes in internal states

But to get back to the point of this essay: Since Darwin’s classical treatise on the expression of emotion (Darwin, 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, ‘Unhumm’, 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 concepts. Are we to say that the patient felt hungry because she ate ravenously despite her verbal denial? Or are we to take her statement at face value and seek elsewhere for an explanation for 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 (Pribram, 1971, pp. 192–194).

As a result of this and other observations George Miller, Eugene Galanter and I called ourselves ‘Subjective Behaviorists’—an oxymoron at the time we wrote ‘Plans and the Structure of Behavior’ (Miller et al., 1960). In keeping with this insight, I will describe the expressions of emotional and motivational processes in terms of observed behavior and assess emotional and motivational experience—feelings—primarily on the basis of verbal statements of humans.

5. Stop and go

At the hypothalamic level, another paradox has emerged. 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 et al., 1950).

It was also found that the more palatable the food, the more the lesioned subject would eat (Teitelbaum, 1965), giving rise to the notion that the lesioned animals did not show greater ‘drive’ to eat but were actually more ‘finicky’ than their controls. Experimental results obtained 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 can be made: 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 re-stabilization.

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 (as measured by skin conduction and the de-synchronization of the EGG) to distracting stimuli (see the following and Pribram and McGuinness, 1975a), and more generally when behavior is interrupted (Mandler, 1964). By contrast, the organism is considered motivated when his readiness mechanisms are activated (see also Pribram and McGuinness, 1975a), 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 and 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. H, 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 by internal body mechanisms' while effective interactions generate 'implicit' (i.e. readiness) or overt responses toward the stimulus object (Kantor and 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 act or is in the process of acting on the environment, whereas emotional antecedents imply 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'.

6. Communication of feelings

An important consideration arises at this point. If the expression of emotions is affective (rather than effective), that is, emotional expression defines internal coping, how then can we observe and work with such expressions in terms of their 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, boles of rat feces and the like, have been used, but they fail to reflect the richness of (especially the pleasant and positive) emotional processes 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 expres-
sions 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 (Miller et al., 1960, Chapter 5). By contrast, organized motivations (‘plans’) are constructed within the organism’s brain and mean to enter into practical relationships with the exciting object.

7. The hedonic dimension

The importance of humeral factors in determining emotional states has already been noted and is amply documented by Pert (1997) in her book ‘Molecules of Emotion’ (1967). 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 control mechanism is sensitive to the partial pressure of CO₂; 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, by the kidney, and by 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 (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) 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.

Cannon (1927), in his classical studies, took off from Bernard’s (1858) experiments delineating the regulation of the ‘milieu interieur’. Cannon 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 signal 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. Waddington (1957) has suggested the term homeorhesis because the stable states are not static but, in today’s language, are non-linear stabilities far from equilibrium.

Individual homeorhetic mechanisms are multiply interlinked into complex organizations. Thus, the thermostat regulating temperature is linked to the glucostat regulating food intake (in part via control of insulin secretion), 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 CO₂, etc. (Brobeck, 1963, for a review).
In short, the core of the brain (mesencephalon, diencephalon, the basal ganglia, and the 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'. Changes in these states are apparently experienced as hunger, thirst, sleepiness, elation, depression, effort, comfort, etc. (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 and insulin are involved in the hunger mechanism, the concentration salt in the thirst mechanism, the concentration of the indole amine serotonin and the catechol amine norepinephrine 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 and pain, and the concentrations of endorphins (and 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 pain falls into the homeostatic mold. But temperature and pain are also skin senses that share a common spinal pathway, and the question 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 skin components of temperature and pain sensitivity that are processed in the parietal lobes of the cortex are characterized by what is called in neurology 'local sign'. This means that the sensation can be located on the skin, and the duration of the sensation is limited. Head (1920) labeled such sensory experiences 'epicritic' to distinguish them from more diffuse experiences that are obtained during early re-growth 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 et al., 1974), and forebrain and are not affected by stimulations of the parietal cortex or the tracts leading to it (Chin et al., 1976; Richardson and 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 (hedonic) dimension of experience, devoid of epicritic local sign, is therefore characteristically dependent on the quantity (the intensity) of the stimulus (see e.g. Mangina and Beuzeron-Mangina, 1996 for direct stimulation effects on electrodermal activity; and Beuzeron-Mangina, 1996 for basal forebrain electrical activity dependent on memory workload). 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 (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 and Akil, 1974). In short, are there two neural systems, one for pain and one for temperature, or is there only one?

8. Basal forebrain controls: resolving the first paradox

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 hind-brains, 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 output to and input from the body, including the viscera through the autonomic nervous system, is involved in the organization of neurochemical states rather than acting directly 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 and McGuinness, 1975a) showed that three separate systems could be discerned to influence electrocortical desynchronization. One system regulates basic 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 et al., 1965; Bagshaw et al. 1965; Bagshaw and Benzies, 1968; Pribram et al., 1974 reviewed by Pribram and McGuinness, 1975b). Furthermore, this elimination of the viscero-autonomic responses apparently leads to a failure of behavioral habituation, which normally occurs rapidly when the novel stimulus is repeated: the viscero-autonomic 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 (familiarization) 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 interest and, when the novelty exceeds certain limits, the experiencing of 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 non-limbic 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) and motor (i.e. attitudinal) sets (Lassonde et al., 1975; Reitz and

It is this second system that forms the neural basis for an ‘attitude’ theory of emotion and motivation, much as suggested by Bull (1951), 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 includes the n. accumbens septi, as well as the entorhinal and cingulated corteces). This system coordinates arousal and readiness (Pribram and McGuinness, 1975b, 1992 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 lngvar and Lassen, 1975).

Neurochemically the three systems also differ (reviewed by Pribram, 1977a). To oversimplify, essentially, the amygdala is rich in endorphins, and essentially, the caudate and putamen are characterized by dopamine. The hippocampal system is especially involved in the pituitary–adrenal hormonal controls, selectively absorbing adrenocortical hormone (see e.g. Bohus, 1976; McEwen et al., 1976) and being acted upon by ACH (adrenocorticotropic hormone) and related enkephalins (Reizen et al., 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 interest (arousal) produced by novelty appears to be related to endorphin homeostasis, the activation of involvement, that is, 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 brain model of 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 stabilization—that is, 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 disturbing events, and what is perceived as novel, or disturbing, is dependent on the configuration of the states that determine what is familiar. The distinction between a feeling of being merely interested or being disturbed is one of intensity only—e.g. electrical stimulations of the amygdala in animals and humans produce orienting, investigating, avoidance, escape, and attack as a function of ascending stimulus intensity (Gastaut, 1954).

In contrast to the phasic destabilizing of familiarity is the maintenance of attitudinal involvement by tonic operations of the basal ganglia readiness system.

When the demands of arousal are pitted against those of readiness the organism can be subject to stress that calls for effortful coping. There is considerable evidence that the maintenance of a stable basal temperature (in warm-blooded animals) involves the food appetitive, water balance, and tonic muscular readiness systems, among others (see Brobeck, 1963, for a review). When effort is involved the coordinating process centers on the hippocampal system that ordinarily adjudicates the smooth transition from state to state within some comfortable band width of tolerance.

In short, the first paradox is resolved by considering the basal forebrain control systems to be involved in initiating emotion related feelings of attentional interest and maintaining motivational related intentional involvement. Interest and involvement conjoin cognitive processes such as memory and expectancy to raw hedonic experience and expression.

9. Self interest and involvement: resolving the second paradox

Jaak Panksepp (2001) has emphasized the distinction between emotions (which he uses synonymously with feelings) that are based on a perception-action cycle and those that are based on an action-perception cycle. This is an important distinction that, I believe, characterizes the difference between the construction of a ‘me’ using the parietal (perception-action) systems of the brain
and the construction of an 'I' using the frontal (action-perception) systems.

The objective ‘me’ and its projection onto other ‘me-s’) is what Freud termed ‘the object’ in psychoanalytic theory. Freud’s conception was based on Brentano’s concept of ‘intentional inexistence’ which later was reformulated by Husserl (1962) as ‘intentionality’. The basic theme of these formulations was that every perceptual act had both a semantic content (what is being perceived) and a perceiver who intends to perceive. Such intended perceptual acts may or may not be matched (realized) by the sensory input (as, for instance in ‘perceiving’ a unicorn, the example Brentano uses).

Conflated (not distinguished) in these earlier formulations is a narrative ‘I’ based on episodic processing. Episodes and their constituent events are personal records monitoring the outcomes of actions. In terms of operant conditioning, these outcomes (events, from the Latin ex venire, out come), are either reinforcing or deterrent (in more ordinary terminology, they place a hedonic value on the behavior) depending on the situation (the consequences, the sequence of contexts) they evoke.

The evidence for processing ‘self’ in terms of a hedonic vs. epicritic dimension has come mainly from the clinic; experimental findings with non-human primates pointed the way. 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 repeatedly shown the parietal cortex 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 and MacLean, 1953) with the cingulate and retrosplenial 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 polar frontal and polar temporal lobe involvement tend to obsessively talk and write voluminously about themselves and, as noted, to lose control over behavior that is context-sensitive, i.e. depends on some stable mnemonically organized self (Pribram, 1999).

This reading of forebrain control over emotional and motivational processes goes back to my earlier experiences with H.M., the now famous ‘case history’ of bilateral medial temporal lobe reception. I saw H.M. within a few days of his surgery, expecting to see a flattening of feelings, both emotional and motivational. Instead, we literally had fun, excitedly reminiscing about actual and projected high school adventures. And then, after I briefly left the room to answer a telephone call, I stumbled into his total lack of episodic memory. This experience has been highlighted and expanded recently by the following case history:

T.J. had an agenesis of the corpus callosum with a midline cyst at birth. During the first 6 months of his life, two surgical procedures were carried out to drain the cyst. Recently performed magnetic resonance imaging showed considerable enlargement of the frontal horns of the lateral ventricle—somewhat more pronounced on the right. The orbital part of the frontal lobes appeared shrunken as did the medial surface of the temporal pole. T.J. appears to have no ability for quantifying the passage of time (what Bergson (1911/1959) called durée) and no experiential appreciation of the meaning of time units. For example, a few minutes after tutoring begins, he cannot say—even remotely—how long it has been since the session started. He is as apt to answer this question in years as in minutes. He does always use one of seven terms of time quantification (seconds, minutes, hours, days, weeks, months or years) when asked to estimate the duration of an episode but uses them randomly. He can put these terms in order, but does not have any sense of their meaning or their numerical relationships to one another. When T.J. returned from a trip to the Bahamas he did recall that he had been on the trip; however, the details he could recount about the trip numbered fewer than 5. His estimates of how long it had been since his trip, were typical in that they were inaccurate and wildly inconsistent on repeated trials. Also, the first five times back at tutoring he stated that he had not been at tutoring since his trip. It appears that he is unable to place in sequence those few past events that he can recall. Nonetheless, he can answer questions correctly based on his application of general knowledge about development, e.g. he knows he was a baby before he could talk because ‘everyone starts as a baby’. But, one day he asked his tutor if he knew
him when he was a kid, indicating, I think, his incomprehension of the duration of each of these developmental periods and his unawareness of what events constituted such a period for him. T.J. is aware that he has a past, that events have happened to him but he cannot recollect those events. He also spontaneously speaks of events in his future such as driving an automobile and dating and growing a beard. He has play-acted on separate occasions his own old age and death. T.J. is capable of excitement about the immediate future. On the very day that he was going to the Bahamas he was very excited as he exclaimed repeatedly: 'I'm going to the Bahamas. 'But when his tutor asked him when, he said blankly; 'I do not know'. He also displayed keen anticipation when one day he saw a helicopter preparing to take off from the hospital. The helicopter engines revved approximately 13 min before it took off and T.J. become increasingly more vocal and motorically active, laughing as he repeated 'When's it going to take off?' He also anticipates future punishment when he is 'bad'. He is aware, on some level, of the immediate future in his constant question 'what's next' which he asks his mother at the end of each activity. There are a variety of other occasions on which he demonstrated this capacity regarding tempo (as opposed to evaluating the duration of an experience). There have been several breaks in his usual thrice weekly tutoring schedule. Each of four times this schedule has been interrupted, he has run to meet his tutor when he approached rather than waiting inside as he usually does. Also, on these occasions he has typically asked if his tutor missed him. However he states he does not know how long it has been since his last session, and there was no evidence that he knew it had been longer than usual. T.J. compares who walks faster or who draws faster. He has at least a basic sense of sequencing as when he says 'I'll take a turn and then you take a turn'. He also uses terms like 'soon' and 'quick' correctly in conversation. For example, when he wanted to do a drawing at the beginning of a session, and his tutor said that we needed begin to work and he countered 'this will be quick'. Unsurprisingly, he finished his drawing at his normal pace. He somehow seems to use such terms correctly without any experiential appreciation of them—Modified from Ph.D. thesis by Chuck Ahern, Supervised by Karl H. Pribram

Contrast T.J.'s symptoms with the following:

From Mrs C.: I was doing laundry about mid-morning when I had a migraine. I felt a sharp pain in my left temple and my left arm felt funny. I finished my laundry towards mid-afternoon and called my neurologist. He told me to go to the emergency room. I packed a few things and drove approximately 85 miles to the hospital where he is on staff (the nearest was 15 min away). In the E.R. the same thing happened again. And again, the next morning after I was hospitalized, only it was worse. The diagnosis of a stroke came as a complete surprise to me because I felt fine, and I did not notice anything different about myself. I remember having no emotional response to the news. I felt annoyed and more concerned about getting home, because I was in the process of moving. Not until several days later while I was in rehabilitation did I notice strange things happening to me. I was not frightened, angry or annoyed. I did not feel anything—nothing at all. Fourteen days after I was admitted to the hospital, I became extremely dizzy, and I felt I was falling out of my wheelchair. The floor was tilting to my left and the wheelchair was sliding off the floor. Any stimulus on my left side or repetitive movement with my left arm caused a disturbance in my relationship with my environment. For instance, the room would tilt down to the left, and I felt my wheelchair sliding downhill off the floor, and I was falling out of my chair. I would become disoriented, could hardly speak, and my whole being seemed to enter a new dimension. When my left side was placed next to a wall or away from any stimuli, this disturbance would gradually disappear. During this period, the left hand would contract, and the arm would draw up next to my body. It did not feel or look like it belonged to me. Harrison moved the left arm repeatedly with the same movement, and a similar behavior occurred, except I started crying. He asked me what was I feeling, and I said anger. In another test he started giving me a hard time until the same episode began to occur, and I began to cry. He asked me what I was feeling, and I said anger. Actually I did not feel the anger inside but in my head when I began to cry. Not until I went back to school did I become aware of having no internal physical feelings. I call that arm Alice (Alice does not live here anymore)—the arm I do not like. It does not look like my arm and does not feel like my arm. I think it is ugly, and I wish it would go away. Whenever things go wrong, I will slap it and say, 'Bad Alice' or 'It's Alice's fault'. I never know what it is doing or where it is in space unless I am looking at it. I can use it, but I never do consciously because I am unaware of having a left arm. I do not neglect my left side, just Alice. Whatever it does, it does on its own, and most of the time, I do not know it is doing it. I will be doing homework and then I will take a sip of coffee. The cup will be empty. I was drinking coffee with that hand and did not know it. Yet I take classical guitar lessons. I do not feel the strings or frets. I do not know where my fingers are nor what they are doing, but still I play. How do I live with an illness I am not aware of having? How do I function when I am not aware that I have deficits? How do I stay safe when I am not aware of being in danger?

Ms C. is obviously intelligent, attending lecture material, asking interesting questions. She is a widowed lady in her mid-fifties, enrolled in adult education, majoring in clinical psychology. She gets around splendidly despite Alice and despite a history of a temporary left hemi-paresis. The diagnosis was damage of the right temporal-parietal cortex confirmed by an abnormal EEG recorded from that location. The damage was not sufficiently extensive to show in a PET scan.
By contrast T.J.’s episodic memory is severely deficient. He has no problem with his egocentric space, nor is he blindsighted (Weiskrantz, 1986)—he has no difficulty in experiencing his allocentric whereabouts. Despite his disability in monitoring, he continually defines his location both in space and in clock time. However, his narrative self is severely limited by his inability to monitor events and place them into sequences of episodes. The narrative self is composed of such sequences of episodes. T.J.’s attempts to do so are contrived and depend on his intact ability to deal with egocentric and allocentric experience.

These two case histories illuminate two very important dimensions of self. One dimension, portrayed by Mrs C., locates us in the world and also with respect to our body’s configural integrity. The other dimension, highlighted by T.J., monitors our experience. Without such monitoring, the events comprising the experience fail to become evaluated and encoded into memory. Location is kin to—but more primitive than—a spatial dimension; monitoring is kin to—but more basic than—a temporal dimension.

To summarize: Two dimensions of self can be distinguished on the basis of selective damage to different parts of the brain. These dimensions concern an objective ‘me’ and a narrative ‘I’.

The objective ‘me’ is characterized as spatio-temporally articulated (egocentrically) and located in the world (allocentrically). The narrative ‘I’, by contrast, is constituted by interest and involvement, an intensive (in both the ordinary and the philosophical sense) hermeneutic monitoring of episodes and events which themselves are the consequences of the monitoring, and thus self-organizing.

The second paradox can therefore be resolved by considering the basal forebrain and related frontal and temporal lobe cortices to control the construction of episodes composing a narrative ‘I’. Such construction is based on a self-organizing process of interest and involvement in which emotional and motivational experiences and expressions are an integral part. However, the immediate moment to moment departures from steady states which characterize emotional and motivational experiences and expressions appear to be mediated by the brain stem and diencephalon rather than by the basal forebrain (limbic and basal ganglia).

In addition, the posterior cortices allow us to locate ourselves as a ‘me’ in physical and social environments. It is these posterior cortices, therefore, that provide the semantics, the labels with which we identify particular feelings of emotion and motivation.

10. Conclusion

The entire brain is involved in the regulation of feeling. The raw hedonic feels of pain and pleasure based on core brainstem and spinal cord systems become conjoined with cognitive processes such as memory and expectancy to create a more complex range of feelings. Each part of the brain has a very specific role in the totality, and this role involves the control of other neural systems. Changes in core-brain controlled body states are monitored by a system centering on the amygdala which contributes an intensive hedonic dimension which ranges from an arousal of interest to a disturbing disruption of ongoing behavior. The hedonic dimension is based on a neural model, a self organizing representation of the familiar. Brief, episodic, mild changes perturbing the model are experienced as interesting and novel; more severe disruptions are disturbing.

Maintaining involvement in the self-organizing process appropriate to a particular action based (attitudinal) feeling state is called being motivated and is controlled by the non-limbic basal ganglia. When a conflict between maintenance and change needs to be mediated, the mediation can become stressful and demand effort; when little mediation is needed, behavior is regulated by pre-established context, becomes more or less automatic, and when experienced feels comfortable. The hippocampal system is especially involved in this effort/comfort dimension.

But, these dimensions of feeling are not their only dimensions. Specific marking is also a prominent feature of emotional and motivational experience and expression. A frontal cortical contribution by way of input to basal forebrain structures (Goldman and Nauta, 1977) is responsible for embracing the feelings within a personal
narrative. In addition, a posterior cortical contribution makes possible an epicritic semantic labeling of feeling states (Gazziniga, 1970; Schachter and Singer, 1962).

Brain systems, body systems (chemical, visceral, muscular), physical (e.g. radiation, gravitation) and social systems thus all become conjoined in the processing of feelings. Specificity arises from the variety of conjunctions in brain systems. This explains the different effects of restricted brain resections and stimulations. In view of what has been reviewed here, what we have loosely come to call the relationship between the limbic system and emotion (presumably emotional feelings) is at once too restrictive and at the same time too inclusive. The relationship between the basal forebrain and feelings needs, at a minimum, to include the concepts 'interest' and 'involvement'.

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