

HEART-BRAIN NEURODYNAMICS: THE MAKING OF EMOTIONS

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*Emotions are...
the function where mind and body most closely
and mysteriously interact.*

—Ronald de Sousa, *The Rationality of Emotion*

As pervasive and vital as they are in human experience, emotions have long remained an enigma to science. This monograph explores recent scientific advances that clarify central controversies in the study of emotion, including the relationship between intellect and emotion, and the historical debate on the source of emotional experience. Particular attention is given to the intriguing body of research illuminating the critical role of ascending input from the body to the brain in the generation and perception of emotions. This discussion culminates in the presentation of a systems-oriented model of emotion in which the brain functions as a complex pattern-matching system, continually processing input from both the external and internal environments. From this perspective it is shown that the heart is a key component of the emotional system, thus providing a physiological basis for the long-acknowledged link between the heart and our emotional life.

The Mental and Emotional Systems

The relationship between mind and emotions has been deliberated at length throughout history, with most schools of thought drawing a boundary between them. Perception, appraisal, arousal, attention, memory, thinking, reasoning, and problem solving are often grouped together under the broader heading of cognition, or the mental system. The emotional system, on the other hand,

encompasses feelings, which can span a range of intensity. The importance of gaining a deeper understanding of the emotional system has become increasingly recognized as an important scientific undertaking, as it has become clear that emotions underlie the majority of the stress we experience, influence our decisions, provide the motivation for our actions, and create the textures that determine our quality of life. In recent years, the concept of “emotional intelligence” has emerged, claiming that emotional maturity is as important as are mental abilities in both personal and professional spheres, and that emotional competencies often out-weigh the cognitive in determining success.¹

The tendency to view emotions as operating separately and apart from rational or intellectual capacities dates back to the times of the ancient Greeks. Thus, historically, thinking and feeling—or intellect and emotion—have often been portrayed as opposing forces engaged in an incessant battle for control over the human psyche. Plato maintained that strong emotions made it impossible for him to think and described emotions as wild horses that had to be reined in by the intellect, while Christian theology has traditionally regarded many emotions as sins and temptations to be overcome by reason and will-power. Traditionally, the intellect was held in high regard, while emotions were considered “irrational” and received little recognition. However, a modern-day examination of emotions presents us with an entirely new perspective, providing a more comprehensive understanding of the emotional system and illuminating the critical roles that emotions play in human experience, performance, and rationality.

Most contemporary researchers agree that cognition and emotion are distinct functions mediated by separate but interconnecting neural systems. A number of research centers, rather than studying these systems in isolation, are attempting to understand the essential dynamic interactions that occur between them. From a neuroscience perspective, several intriguing forms of interaction have been discovered that link the cognitive centers with the emotional processing areas of the brain. For example, bidirectional neural connections that exist between the frontal cortex and the amygdala permit emotion-related input from the amygdala to modulate cortical activity and cognitive input from the cortex to modulate the amygdala’s emotional information processing.²⁻⁴

Beyond these hard-wired neural connections, biochemical bridges also link key components of the mental and emotional systems. The cortex, for instance, has been found to contain a high density of receptors for many neuropeptides that are also heavily concentrated in the brain’s subcortical areas, which are associated with emotional processing.⁵ Evidence suggests, moreover, that communication channels linking the mental and emotional systems are essential for the expression of our full range of mental capacities.⁶

In his book, *Descartes’ Error*, neurologist Antonio Damasio presents evidence

that patients with brain damage in the frontal lobes, a key site of integration of the cognitive and emotional systems within the brain, can no longer function effectively in the day-to-day world, even though their intellectual abilities are perfectly intact. Damasio presents a powerful argument supporting the seemingly counter-intuitive position that input from the emotional system to our thought centers not only facilitates, but is actually indispensable to, the process of rational decision-making.⁷

Emotions influence nearly every type of cognitive activity in subtle yet crucial ways. Emotions can direct attention. This phenomenon is known as the *mood-congruity effect*⁸ Thus, people in a given emotional state pay more attention to stimuli that are emotionally congruent with their current emotional state. Emotions also influence memory and learning, an effect known in neuroscience as *emotion state-dependent memory*.⁹ This is why information learned or obtained in a given emotional state may be more easily retrieved if the individual returns to an emotional state similar to the one that prevailed during the original learning. Emotions can also affect judgment, as well as the cognitive processing style employed during problem solving. This effect is readily demonstrable in the laboratory, as well as in everyday life.¹⁰

While two-way communication between the cognitive and emotional systems is hard-wired into the brain, the actual number of neural connections going from the emotional processing areas to the cognitive centers is greater than the number going the other way.⁴ This goes some way to explain the powerful influence of emotions on thought processes. It also provides insight into how emotional experience, in contrast to thought alone, can often be a powerful motivator of future attitudes and behavior, influencing moment-to-moment actions as well as both short-term and long-term performance. While emotions can easily dispel non-emotional events from conscious awareness, non-emotional forms of mental activity, such as thoughts, do not so easily displace emotions from the mental landscape. Likewise, experience reminds us that the most pervasive thoughts, least easily dismissed, are typically those fueled by the greatest intensity of emotion.

Interestingly, the seventeenth century philosopher René Descartes noted this same point over three hundred years ago. In commenting on the function of human emotion in his *Treatise on the Passions of the Soul*, Descartes wrote:

The utility of all passions consists alone in their fortifying and perpetuating in the soul thoughts, which it is good it should preserve, and which without that might easily be effaced from it. And again, all the harm which they can cause consists in the fact that they fortify and conserve these thoughts more than necessary, or that they fortify and conserve others on which it is not good to dwell.¹¹ (art. 74)

Descartes' views on emotions were clearly more sophisticated than the simplistic notion that emotions are antagonists to rational thought. Descartes considered emotions a double-sided coin. They give substance and sustenance to what otherwise may have been ephemeral thoughts. As a result, they can work both for and against us. Descartes was really highlighting the contrast between the potential of effectively managed emotions and the harm caused by unmanaged emotions. Whereas effectively managed emotions work in synchrony with the mind to facilitate its activity, unmanaged emotions can be the source of mental chaos.

Mental and Emotional Coherence

To further refine Descartes' premise and express it within the context of the concepts discussed here, we can say that when there is *coherence* within and between the mental and emotional systems, they interact constructively to expand awareness and permit optimal psychological and physiological functioning. Conversely, when the mental and emotional systems are out-of-phase, they lack synchronization and thus interact in a conflicting manner, thereby compromising performance. For example, people commonly tell themselves to "think positive" about a challenging task, yet emotionally they may still dread doing it. When our emotions are not aligned with getting the task accomplished we lack motivation and enthusiasm, which limits our access to creativity and insight, and thus impedes our overall performance. In other words, as many of us have likely experienced, positive thoughts or affirmations are often only superimposed on an underlying internal environment of emotional turmoil. In such cases, "positive thinking" is rarely able to produce an enduring shift in the negative feelings.

To better understand an experience such as this, it is important to realize that many common emotion regulation strategies operate on the assumption that all emotions follow thought, and thus by changing one's thoughts, one should be able to gain control over one's emotions. However, in the last decade, research in neuroscience has made it quite clear that emotional processes operate at a much higher speed than thoughts, and frequently bypass the mind's linear reasoning process entirely.⁴ In other words, emotions do not always follow thought; in many cases, in fact, emotions occur independently of the cognitive system and can significantly bias or color the cognitive process and its output or decision.^{3,4}

Since the mind and emotions affect a wide range of abilities and responses, mental and emotional coherence are of the utmost importance. Vision, listening ability, reaction times, mental clarity, problem solving, creativity, and per-

formance in a wide range of tasks are all influenced by the degree of coherence of these two systems at any given moment. Because emotions exert such a powerful influence on cognitive processes, emotional incoherence often leads to mental incoherence. Furthermore, emotional incoherence is often the root cause of “mental” problems and stress. Mental health is maintained by emotional hygiene—emotional self-management—and mental problems, to a large extent, reflect a breakdown of emotional order or stability.

On the other hand, increasing stability in the emotional system can often bring the mind into a greater sense of peace and clarity as well. When the mental and emotional systems are in sync, we have greater access to our full range of potential and a greater ability to manifest our visions and goals, as the power of emotion is aligned with the mind’s capacities. Even more intriguing, we can gain more conscious control over this process than previously believed through the application of tools and techniques designed to increase emotional stability. Empirical research on the outcomes of such techniques indicates that increased mental and emotional coherence, in turn, can lead to a higher degree of physiological coherence, manifested as increased efficiency and synchronization in the functioning of physiological systems.¹²

The positive emotion-focused coherence-building techniques developed by the Institute of Heart-Math engage the heart as a point of entry into the psychophysiological networks that underlie emotional experience.¹²⁻¹⁴ One of the research focuses of our laboratory over the last decade has been the study of the patterns and rhythms generated in various physiological systems during the experience of different emotions. Through experimenting with numerous physiological measures, we have found that heart rate variability (heart rhythm) patterns are consistently the most dynamic and reflective of changes in one’s emotional state. We have demonstrated that positive and negative emotions can be readily distinguished by distinct changes in heart rhythm patterns. Sustained positive emotions are associated with a noticeably coherent (*i.e.*, ordered, smooth, and sine wave-like) heart rhythm pattern, whereas negative emotions are characterized by a jagged, erratic pattern in the heart’s rhythms.¹⁵ Moreover, further exploration led us to discover that unhealthy individuals could be greatly facilitated towards improved physical and emotional health through learning how to generate the coherent heart rhythm patterns displayed by healthy, high-functioning individuals.

An important implication of this work, in relation to the ideas developed in this article, is that the rhythmic patterns generated by the heart are not only *reflective* of emotions, but actually appear to play a key role in *influencing* moment-to-moment emotional perception and experience. In short, through its extensive interactions with the brain and body, the heart emerges as a critical component of the emotional system. Before developing this concept further,

we place it in perspective by offering a brief historical review of the evolution of scientific thinking about emotions, leading up to a summary of current scientific understandings in this field.

The Source of Emotional Experience: An Evolving Model

Current scientific knowledge regarding the physiology of emotions has its roots in Galenic medicine. Galen's influence on scientific thinking persisted well into the 1800s, with the notion that thoughts ("spirits") circulate in the ventricles of the brain, and emotions circulate in the vascular system. Medical thinking at that time maintained that temperament was determined by four "humors" or secretions: sanguine, choleric, phlegmatic, and melancholic. Modern biomedical research has supplemented this simplistic model with a rich array of endocrine and exocrine hormones, which are invoked in any serious biological discussion of emotion. According to neuropsychologist Karl Pribram, who oversaw the brain research center at Stanford University for 30 years, the retreat from this perspective has been slow and guarded for two reasons: Old theories do not die easily, and there is an aspect of truth to this view.¹⁶ The "spirits" circulating in the ventricles have turned out to be neural electrical activity, and the "humors" flowing through the vascular system, endocrine secretions.

An arguably defining characteristic of emotions is that they involve greater activation of the autonomic nervous system and more conspicuous participation of the body than do mental states. This intimate relationship between emotions and physiology has been expressed for centuries in song, poetry, and prose. Even ordinary conversation pertaining to emotional experiences contains numerous physiological allusions. There is no question that emotions are accompanied by a vast array of physiological changes. This is why people so often tend to describe emotional experiences in physiological terms, such as "My heart was pounding," "My throat went dry," "My blood ran cold," "My skin crawled," "It was gut-wrenching," and "It took my breath away." That these figures of speech have become so engrained in everyday language attests to our experience of emotional states being intricately intertwined with, if not inseparable from, their bodily manifestations.

But is what is the ultimate *source* of emotions—the body or the brain? Do emotions originate as bodily sensations that are then perceived by the brain, or do they originate in the brain as a product of cognitive processes and only then trickle down into the body? This fundamental controversy has formed the core of a lively debate that has raged for over a century, yielding a fascinating and illuminating progression of ideas.

The James-Cannon Debate

In 1884, the debate over the source of emotional experience formally began with a proposal by psychologist-philosopher William James in his seminal article entitled “What is an Emotion?”¹⁷ James believed that emotional experience is not only accompanied by, but actually *arises from* organic changes that occur in the body in response to an arousing stimulus. These physiological signals (e.g., racing heart, tight stomach, sweaty palms, tense muscles, and so on) are subsequently fed back to the brain, and only *then* felt consciously as a true emotion. James proposed that we can sense what is going on inside our body much the same as we can sense what is going on in the outside world. The awareness of the immediate sensory and motor reverberations that occur in response to a perception (e.g. the pounding heart, the clenched jaw, etc.) is what makes that perception emotional. Thus, the feeling aspect of emotion is dictated by the physiology and not vice-versa. According to James:

Our natural way of thinking about...emotions is that the mental perception of some fact excites the mental affection called the emotion, and that this latter state of mind gives rise to the bodily expression. My thesis on the contrary is that *the bodily changes follow directly the PERCEPTION of the exciting fact, and that our feeling of the same changes as they occur IS the emotion*¹⁷ (pp. 189-190)

James maintained that the precise pattern of sensory feedback relayed from the body to the brain gives each emotion its unique quality. Thus, anger feels different from sadness or love because it has a characteristic physiological pattern or signature. James maintained that physiological responses contributing to emotion were “almost infinitely numerous and subtle,”¹⁷ (p. 191) reflecting the nuances of physiology and its emotional counterpart.

In fairness to James, it should be noted that his original premise—that the sensation of bodily changes is a *necessary condition* of emotion—was subsequently oversimplified by many of his contemporaries, as well as by many modern authors.¹⁸ The oversimplification of James’ views suggested that emotions are *nothing but* the sensation of bodily changes. In fact, when using the term “perception” in his writings, James did acknowledge the role of interpretation or cognitive appraisal of the exciting stimulus in the initiation of emotional experience. However, he argued that the emotional “feeling” was not a primary feeling directly aroused by appraisal, but rather a secondary feeling indirectly aroused by the organic changes that occurred following the appraisal.

James’ perspective was called into question in the 1920s by the prominent experimental physiologist Walter Cannon.¹⁹ Cannon believed that the essential mechanisms of emotion occurred within the brain alone and that bodily

responses and afferent input to the brain were not needed to fully experience emotions. He argued, in brief, that bodily feedback, especially from the viscera, was both too slow and not sufficiently differentiated to explain the dynamic range and variety of emotional expression. Though Cannon felt that bodily sensations could not account for differences between emotions, he believed that they nevertheless played an important role in giving emotions their characteristic sense of intensity and urgency.

To support his views, Cannon demonstrated that artificially induced visceral responses alone do not produce emotions and that animals still show “emotional behavior” when feedback from the viscera is surgically eliminated. Of course, here Cannon was forced to rely solely on behavioral evidence to define the parameters of emotion in his animal subjects. In place of the visceral theory, Cannon proposed a brain (thalamic) theory of emotions. He suggested that emotional expression results from the operation of hypothalamic structures, while emotional feeling results from stimulation of the dorsal thalamus. This theory was based on the observation that emotion-like behavior could be elicited in decorticated and decerebrated animals, but not when thalamic structures were ablated as well. Further, a variety of expressive and bodily responses were obtained when the thalamus was electrically stimulated.²⁰

In Cannon’s view, the thalamus and hypothalamus discharged simultaneously to the body to produce physiological responses and to the cortex to produce emotional experiences. In measuring the amount of time it took for electrical stimulation of the hypothalamus to produce visceral changes, Cannon concluded that these bodily responses were too slow to be the cause of emotions. He saw them rather as the effect, since his measurements suggested that we would already be feeling the emotion by the time these responses occur.

Much of Cannon’s experimental research centered on autonomic nervous system (ANS) responses that occur in states of hunger or intense emotion.²¹ His research led him to propose the concept of an emergency reaction—the “fight-or-flight response”—to describe a specific physiological response that accompanies any state in which physical energy must be expended. The sympathetic division of the ANS, which he believed to act in a uniform way regardless of how or why it was activated, mediated this response. Cannon held that the visceral changes accompanying emotion were part of this nonspecific arousal, and thus that all emotions had the same ANS signature.

Cannon’s arguments won over the weight of scientific opinion of the day, and his view consequently spawned a search for emotional mechanisms in the brain. Others such as Lindsley and Papez built upon Cannon’s theory by mapping out additional sub-cortical and limbic structures and communication pathways involved in the brain’s emotion-regulating networks.^{22,23} Experimental evidence demonstrated the existence in the hypothalamic region of an energy-conserv-

ing or *trophotropic* process working primarily through the parasympathetic branch of the ANS, and a mobilizing or *ergotrophic* system working through the sympathetic branch.²⁴ It was assumed that the hypothalamus and dorsal thalamus were at the apex of the hierarchy of control of visceral and autonomic functions and were the key to understanding emotional processes.

Neuropsychologist Karl Lashley was the first to criticize this assumption. He pointed out several flaws in the theory by using lesion studies showing that emotional disturbances (on which the Cannon theory was based) could also be observed following lesions elsewhere, such as in the afferent paths in the nervous system or between the forebrain and thalamic structures.²⁵ He also noted that neither the James nor Cannon theories could account for the dissociation between outward emotional expression and inner feelings, which is a common clinical and experimental observation.

The Limbic Theory

An important breakthrough came in 1937 when James Papez, a professor of neuroanatomy at Cornell University, described a circuit between centers in the brain and suggested that it might constitute the neural substrate for emotion, thus introducing the idea of a circuit or system rather than a single center. He suggested that blockage of information flow at any point along this circuit would cause disorders of emotions. Now known as the *Papez circuit*, this model described the flow of information from the hippocampal formation to the thalamus, then to the cingulate gyrus, and back again to the hippocampal formation.

This was later elaborated on by Paul MacLean, chief of the laboratory for brain evolution and behavior at the National Institute of Mental Health. In the 1950s, MacLean introduced the concept of the *limbic system* to denote the interacting regions of the brain involved in emotional processing.^{26,27} In addition to the areas of the Papez circuit, MacLean included regions such as the amygdala, septum, and prefrontal cortex in the limbic system. Later, he also originated the *triune brain* model, which delineated three functional brain systems that he believed developed successively in response to evolutionary needs.^{28,29} Although MacLean's theory has had little impact on neurobiology, it has become popular in the lay press and with psychotherapists. However, it should be noted that extensive work in comparative neurobiology unequivocally contradicts the evolutionary aspects of his theory.³⁰

MacLean believed that emotional experience could be most accurately described as a response to the *composite* of stimuli the brain receives from the external environment, as a result of ongoing perceptions of the outside world, *and* internal sensations or feedback transmitted to the brain from bodily organs and systems. The limbic system came to be viewed as the receiving station or site for the association and correlation of these varied stimuli, being strategical-

ly located to correlate every form of *internal* and *external* perception. MacLean also emphasized the importance of memory and provided data showing that the limbic cortex exceeds the neocortex in the turnover of protein, a measure of the demand for new RNA in memory formation.³¹

Here at last was the seat of emotion—the visceral brain. Karl Pribram summed it up with the following:

The persuasive power of this suggestion is great: Galen, James..., [and] Cannon...are all saved; visceral [bodily] 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...The path from the “emotions in the vascular system” to “emotions in the fore-brain” had finally been completed, and each step along the way freed us from preconceptions popularly current when the step was taken.¹⁶ (p. 16)

Despite its popularity, there are problems with the limbic theory of emotions and it falls heir to the same criticisms leveled against Cannon. The idea of a specific center (*i.e.*, the thalamus) as a privileged site for emotional experience did not hold up; and the same problem arises with relations between the limbic structures and bodily input, and for that matter, the limbic system itself and emotions. For example, it was found that emotional changes can be observed to accompany lesions in parts of the brain other than limbic areas. Further, ablation and stimulation of limbic structures influence problem solving and other cognitive behaviors in selective ways that cannot be attributed to changes in emotion. In fact, obvious and specific “memory” defects follow limbic lesions, while changes in emotions cannot be found.²⁰ Obviously, the Papez-MacLean theory, like its predecessors, presented only part of the picture.

With the development of newer techniques for electrical brain stimulation, Pribram and others showed that the so-called “limbic” brain regions were under the surveillance and control of the neocortex.³² Brain structures such as the hippocampus, amygdala, cingulate cortex, septum, thalamus, hypothalamus, and prefrontal cortex came to be viewed as interpreting experience in terms of feelings rather than “intellectualized” representations. It now appears that the whole brain as well as the ascending input from the body, both neurological and hormonal, are necessary in the full experience of emotion.

Memory

An important aspect of emotional experience is memory. The first associations of memory with specific parts of the limbic system appear to have been made in 1900 by the Russian neurologist-anatomist Vladimir Mikhailovich Bekhterev when he observed memory deficits in a patient with hippocampal

degeneration.²⁰ The story of the search for memory is far beyond the scope of this monograph; however, the work of Canadian psychologist Donald Hebb has special relevance to this paper's theme. In 1949, Hebb predicted a form of synaptic plasticity based on temporal activity, which was verified decades later with the discovery of long-term potentiation.³³ Hebb believed that synaptic connections were the material basis of mental associations; however he went well beyond the naïve connectionism theories of that time period in two important respects. First, he argued that an association could not be localized to a single synapse. Instead, neurons were grouped in "cell assemblies," and an association was distributed over their synaptic connections. Secondly, Hebb rejected the concept that input-response behaviors could be explained by simple reflex arcs connecting sensory neurons to motor or output neurons. He believed that sensory stimulation could initiate patterns of neural activity that were maintained by circulation in synaptic feedback loops. This reverberatory activity made it possible for a response to follow a delay that was characteristic of thought. In essence, Hebb argued for a dual-trace mechanism of memory. Reverberatory neural activity was the trace for short-term memory, and synaptic connections were the trace for long-term memory. He hypothesized the conversion of short-term memory into long-term memory by the stabilization of reverberatory activity patterns. Once such an activity pattern was stored, in a redistribution or change in the strength of synaptic connections, it could be recalled repeatedly by an excitation from sensory neurons or from other reverberatory activity patterns occurring in other cell assemblies that provide inputs. In the past fifty years, several aspects of Hebb's theory have been confirmed, while the technology needed to prove or disprove other aspects does not yet exist.

In the 1970s, new insights into the question of what happens in the brain during the time interval between stimulus and response were made possible with the discovery of long-term potentiation. This and the first neural network models of delay activity provided a candidate for Hebb's "reverberatory" activity. For example, it has been demonstrated that certain prefrontal cortex neurons remain active during delays of many seconds and encode information about the preceding stimulus or the impending response. Changes in distribution and strength of synapses have been confirmed, and this aspect of his theory is not in doubt. What remains unknown is whether the delay between stimulus and response is truly due to a reverberatory type of activity, and if so, if the reverberatory activity is stabilized by long-term potentiation. Also, Hebb's concept of only two memory traces may be incorrect, as it is now known that synaptic plasticity involves many processes operating on different time scales.³⁴

Current Perspectives on the Nature of Emotion

Most theorists now agree that emotion involves, at the most fundamental level, the registration and interpretation of a stimulus based on memory processes in addition to information from physiological responses and subjective feeling states. In more recent years, attempts have been made to determine the “correct” sequence of these components in the generation of emotional experience. However, when interpretation, subjective feeling, and bodily responses are all considered as *processes*, rather than discrete events or simple input-output relations, the source of a large part of the controversy dissolves.¹⁸

We find that it is indeed possible to have emotional processing in specific brain areas simultaneously with input from the body to the brain, each building on the other to contribute to the dynamic process of emotion. Recent elucidation of the numerous afferent pathways through which the body transmits signals to the brain and the interaction of this information with higher-level brain processes provides strong support for this perspective. Elmer Green, Menninger Clinic physician and pioneer of the biofeedback approach to treatment of disease, offered an astute summation of this highly debated topic: *“Every change in the physiological state is accompanied by an appropriate change in the mental emotional state, conscious or unconscious, and conversely, every change in the mental emotional state, conscious or unconscious, is accompanied by an appropriate change in the physiological state.”*³⁵

The remaining element of the controversy, namely the specificity of physiological responses, must now take into account new data revealing that communication between the body and the brain is much more sophisticated and complex than previously imagined. The generation of such data has been made possible, in part, due to the development of more sophisticated recording techniques and instrumentation that more clearly capture the subtleties and complexities of communication between different bodily systems and between the body and brain. In addition, technological advances have enabled us to achieve finer measurements of neuroendocrine and immune activity, thereby offering a wider view into the array of physiological responses at the cellular level that accompany different emotional states.

Before introducing a new model of emotion that synthesizes and further develops many of the perspectives discussed here thus far, a brief review of the role played by activity in both the efferent and afferent pathways of the nervous systems in emotional experience is relevant.

Specificity of Autonomic Responses

Let's return to Cannon's assumption that all emotions are associated with the same basic state of non-specific arousal or activation of the ANS. In the 1960s, Stanley Schachter and Jerome Singer, social psychologists at Columbia University, embraced this view by suggesting that a cognitive interpretation of a basically undifferentiated state of physiological arousal within the social or environmental context of the arousing stimulus was the missing factor in determining the specificity of emotion.³⁶ Schachter and Singer's model, called the *two-factor theory* proposed that emotions are produced by both feedback from the body and the cognitive appraisal of what caused those responses. In other words, we label the response according to what we think is causing the response. This theory had a profound influence on the thinking on the subject of emotion at the time. However, in the last thirty years the tide has turned, as increased evidence has emerged to indicate that autonomic responses in different emotional states are much more complex than previously assumed, and certainly far from uniform.

In contrast to the thinking in Cannon's day, which attributed emotional arousal to sympathetic nervous system activation alone, we now understand that simultaneous and complex changes in the patterns of efferent activity in both the sympathetic and parasympathetic branches of the ANS are involved in the experience of different emotions. The sensations produced in any given emotional state depend on the extent to which sympathetic effects are balanced by parasympathetic influences; thus sympathetic/parasympathetic balance has become an important measure in psychophysiological research.

Many emotional states are associated with complex patterns of sympathetic/parasympathetic activity in different tissues. For example, in states of aggression and resentment, increased sympathetic discharges occur in the vascular system while parasympathetic discharges predominate in the gastrointestinal tract. Conversely, increased sympathetic activity occurs in both the cardiovascular and gastrointestinal systems in states of fear. Further, autonomic responses vary both quantitatively and qualitatively with the degree of emotional intensity.³⁷

A number of experiments conducted in the 1950s provided evidence that different emotions could be differentiated psychophysiologicaly³⁸⁻⁴¹ These findings have been confirmed recently.⁴²⁻⁴⁶ For example, in an experiment by Ekman and colleagues at the University of California in San Francisco, subjects experienced different emotional states (happiness, surprise, disgust, sadness, fear, and anger) both by reliving past emotional experiences and by constructing facial prototypes of emotion, muscle by muscle, according to instruction. Specific

differences in the autonomic parameters of heart rate, finger temperature, and skin resistance were found among the six different emotions measured. The response patterns differed not only between positive and negative emotions, but also among the negative emotions of disgust, sadness, anger, and fear. These differences were consistent across profession, age, gender, and culture.⁴³ While this and other research provided convincing evidence of autonomic variation among different emotional states, the variation measured was often small and present in only some of the physiological parameters, or experienced by only a subset of subjects.

A more recent study measuring multiple autonomic parameters showed that six basic emotions (happiness, surprise, anger, fear, sadness, and disgust) could be fully differentiated on the basis of electrodermal variables (skin resistance, skin conductance, and skin potential), thermovascular variables (skin blood flow and skin temperature), and a respiratory variable (instantaneous respiratory frequency).⁴⁵ These results clearly support the concept of emotion-specific ANS activity, which can be demonstrated with the aid of careful experimental procedures providing that a sufficient number of autonomic variables are considered.

Individual differences in patterns of autonomic discharge during emotional states have also been identified and associated with personality characteristics. For instance, individuals who have been characterized as “impulsive” personality types display rhythmic bouts of palmar sweat secretion and increases in heart rate even at rest, while in others, little change occurs in these physiological parameters under similar circumstances.³⁷

The Importance of Afferent Input

In addition to understanding how complex patterns of efferent autonomic activity correlate to differing emotions, many scientists are beginning to understand the critical role played by the afferent neural signals that flow from the body to the brain. Afferent feedback from bodily organs has been shown to affect overall brain activity and to exert a measurable influence on cognitive, perceptual, and emotional processes.

Physiology textbooks are replete with diagrams that illustrate nervous system pathways from the brain to autonomically innervated organs. However, many of these illustrations do not complete the communication circuit. They frequently omit the extensive systems of visceral afferent fibers, which carry messages from receptors in the body to the brain. The nerve pathways connecting most organ systems to the brain are, in fact, composed of as many afferent fibers as there are efferent connections;⁴⁷ while in some visceral nerves, such as the abdominal vagus, up to ⁹⁰ percent of the fibers are afferent.⁴⁸ Remarkably, we now know that the heart sends more neural traffic to the brain than the

brain sends to the heart. While afferent pathways were identified during the early years of autonomic research, their study was not emphasized. However, research conducted primarily in the 1950s through the 1970s began to illuminate the importance of afferent input from the thoracic, abdominal, and neck cavities back to the brain—and the effects of this input on brain activity and emotional experience.

One of the earliest contributors to our understanding of the importance of afferent neural traffic was the German internist, Ludwig van Müller. He was particularly interested in the perception of sensory stimuli arising from internal organs and their role in the regulation of different bodily states and sensations.⁴⁹ He pointed out, in 1906, that emotions influence heart rate, and conversely, that heart rate influences emotions. For example, he observed that cardiac palpitations can induce emotions.⁵⁰

Early neurophysiological evidence of the influence of afferent input on brain activity dates back to 1929. Tournade and Malméjac, followed by Koch two years later, showed that stimulation of the carotid sinus nerve (contributing afferent fibers which enter the brain stem), or an increase in pressure in the carotid sinus itself, produced a decrease in muscle tone in anaesthetized animals.⁵¹ Koch also demonstrated that by sharply increasing the pressure within the carotid sinus he could inhibit motor activity and induce prolonged sleep. These results were confirmed in later investigations, which showed that distension of the carotid sinus produced marked changes in cortical electrical activity, from low-voltage fast to high-voltage slow waves (characteristic of sleep), and inhibited activity of the pyramidal nerve cells in the motor cortex, which control muscle movement.⁵¹

In the 1950s, French and Italian neurophysiologists performed a variety of experiments investigating the effects of changes in heart rate and blood pressure on brain activity. Changes in heart rate and blood pressure are detected by receptors in the heart, the aortic arch, and the carotid sinus. Information from these receptors is transmitted to the brain stem via the vagal and glossopharyngeal nerves.⁵² In one study, Bonvallet and Allen demonstrated that elimination of the glossopharyngeal and vagal input to the brain resulted in a prolongation of cortical activation and skeletal muscle activity.⁵³ Then in 1974, French researchers Gahery and Vigier, working with cats, found that stimulating the vagus nerve reduced the electrical response in the cuneate nucleus of the brain to about half its normal rate.⁵⁴ Since that time, extensive experimental data have been gathered documenting the role played by afferent input in modulating such varied processes as pain perception,⁵⁵ hormone production,⁵⁶ electrocortical activity, and cognitive functions.⁵⁷⁻⁵⁹ Animal studies have now demonstrated that a variety of brain regions are involved in the processing of visceral afferent information, including the hypothalamic and thalamic nuclei, amygdala, hip-

pocampus, cerebellum, somatosensory cortex, prefrontal cortex, and insula.⁶⁰ Thus, it has become clear that the influence of cardiovascular afferent signals on the brain is far more pervasive than previously considered.⁶¹

Uncovering conversations between the heart and brain

Among the first modern psychophysiological researchers to systematically examine the “conversations” between the heart and brain were John and Beatrice Lacey.⁶² During 20 years of research throughout the 1960s and 1970s, they observed that afferent input from the heart and cardiovascular system could significantly affect *perception and behavior*. Their research produced a body of behavioral and neurophysiological evidence suggesting that sensory-motor integration could be modified by cardiovascular activity.^{52, 63-66}

The Laceys’ observations directly challenged the “arousal” or “activation” theory proposed by Cannon. In essence, Cannon believed that all of the physiological indicators underlying emotion—heart rate, blood pressure sweating, pupil dilation, narrowing of certain blood vessels, and so on—moved predictably *in concert* with the brain’s response to a given stimulus. Thus, Cannon had suggested that when we are aroused, the sympathetic nervous system mobilizes us to fight or flee. In contrast, in quieter moments, the parasympathetic nervous system relaxes our inner systems. Presumably, autonomic responses all increased together when we were aroused and decreased in unison when we were at rest, and the brain was entirely in control of both these processes.

The Laceys noticed that this view of activation as a single dimension only partially matched actual physiological behavior; they observed that all physiological responses did not always move together. As their research evolved, they found that the heart, in particular, seemed to have its own peculiar logic that frequently diverged from the direction of other ANS responses. In essence, the heart seemed to behave as if it had a mind of its own. In laboratory studies of reaction time and operant responses, the Laceys observed that in response to certain stimuli, all autonomic variables recorded did not exhibit the expected response pattern typical of arousal. At times, for example, heart rate decelerated and blood pressure *decreased*, while simultaneously recorded parameters such as skin conductance, respiration rate, and pupillary dilation all increased as expected. The Laceys called this phenomenon *directional fractionation* and noted that it appeared to be dependent upon the nature of the stimulus and the type of mental processing involved.⁶³

The Laceys found that tasks requiring mental concentration or attention to *internal* stimuli (*e.g.* mental arithmetic, reverse spelling, or making up sentences) produced an acceleration in heart rate and an increase in skin conductance. In contrast, tasks requiring attention to the external environment (*e.g.*, detecting colors and patterns or empathizing with a dramatic recitation) produced a

marked deceleration in heart rate, although skin conductance still increased. The Laceys also showed that patterns of physiological responses were affected as much by the context of a specific task and its requirements as by emotional stimuli. Thus, heart rate, for example, tends to decrease, even in the presence of a distressing emotional context, when subjects are attending visually or auditorially to events in their external environment; on the other hand, heart rate accelerates when subjects mentally recall and think about the very same unpleasant emotional material.⁶³⁻⁶⁵ Subsequent research also revealed an intriguing link between the heart rate response (but not other autonomic responses) to different environmental stimuli and an individual's cognitive style, or attitude toward the external environment.⁶⁷⁻⁶⁸

The selectivity of the heart's response indicated that *it was not merely mechanically responding to an arousal signal from the brain*. Even more intriguing, in simple reaction time experiments, which required attention to external cues, an *anticipatory* deceleration in heart rate was observed during the preparatory interval, and subjects' reaction times were faster during periods when their heart rate was slowing.⁵² This led the Laceys to propose that cardiovascular afferent feedback to the higher brain centers plays a role in *facilitating either the intake or rejection of environmental stimuli*, in accordance with the nature of the mental processing required for a given task.⁶⁶ In brief, such a mechanism would permit us effectively to "tune out" potentially disruptive external environmental events when performing tasks requiring internal cognitive elaboration, and, conversely, to focus in on external inputs when our activities demanded close attention to our environment.

To support this hypothesis, the Laceys and others found evidence that in humans under normal physiological conditions, brain activity varies in relation to cardiovascular events.^{52, 69} Thus, increased heart rate and the resulting increased afferent discharge inhibits (desynchronizes) cortical activity. Conversely, decreased heart rate occurring prior to sensory intake promotes cortical facilitation and processing by reducing brain inhibition.⁶⁶ In their reaction time experiments, the Laceys discovered that the greater the cardiac deceleration, the greater the cortical activation, and the greater the behavioral efficiency (*i.e.*, the faster the speed of response). In other words, *afferent input to the brain from the heart can either inhibit or facilitate the brain's activity, which, in turn, can affect perception and motor activity*

Evoked potential studies

A useful technique for the study of how and where information flows through the brain is evoked potential analysis. Evoked potentials (also sometimes referred to as event-related potentials) are obtained using signal averaging, a procedure for separating a known repetitive signal from other signals.

Evoked potential analysis can be used to study the flow of information through many different pathways in the brain. Common applications of the technique are to study the visual, auditory, and somatosensory systems. In the case of the visual system, for example, the flow of information through the nervous system produced in response to a series of light flashes or a changing visual stimulus of any kind can be traced through the different visual pathways as it is processed. In this case, the resulting waveforms are called visual evoked potentials. It is also possible to examine the flow of afferent input through the brain from many other sensory systems, such as the auditory and tactile systems, or to assess how a change in afferent signals generated by one system affects the processing of information in another system.

For example, the effects of cardiac afferent input on sensory perception have been studied by looking at how these signals affect processing in the visual system. It has been shown that the processing of visual information is significantly changed as heart rate and carotid pressure change. These findings provide confirmation of the Lacey's' earlier behavioral evidence that cardiovascular activity influences sensory intake.⁷⁰

While these data indirectly support the view that cardiovascular afferent information interacts with higher central nervous functions, experiments by the German researcher Rainer Schandry and others have provided more direct psychophysiological evidence for this perspective. Their work has demonstrated that cardiovascular events like heartbeats are detectable as a signal in the EEG and evoke cortical responses analogous to "classical" sensory event-related potentials.^{60, 71, 72} When the heart's afferent signals are being studied, the ECG R-wave is used as the timing source for the signal averaging and the resulting waveforms are called heartbeat evoked potentials (HBEPs). These experiments have shown that the processing of afferent input from the cardiovascular system is accompanied by specific electrical activity in the brain. This processing of cardiovascular afferent information is most pronounced at the frontocortical areas, a brain region known to be particularly involved in the processing of visceral afferent information. Recent findings have demonstrated that the HBEP is significantly diminished in diabetic patients with autonomic neuropathy, and reduced amplitude of the HBEP is significantly correlated with reduced awareness of body sensations.⁷³ In other words, when the communication of afferent signals from the heart to the brain is compromised, there is less awareness of feeling sensations in the body.

Furthermore, psychological factors, such as motivation, attention to cardiac sensations, and general perceptual sensitivity, have been found to alter HBEPs in the brain in a manner analogous to the cortical processing of external stimuli.^{60, 72} These findings confirm our own data demonstrating that focusing attention in the area of the heart and generating a positive emotion alters HBEPs,

thus indicating an modulation of cortical processing. Taken together, these data suggest that perception and processing of information arising from bodily processes is comparable to perception and processing of external events, and the effects of both sources of input on perceptual and emotional experience must be considered.

In summary, evidence now clearly demonstrates that afferent signals from the heart significantly influence cortical activity. Specifically, we now know that afferent messages from the cardiovascular system are not only relayed to the brain stem to exert homeostatic effects on cardiovascular regulation, but also have separate effects on aspects of higher perceptual activity and mental processing. Furthermore, as discussed next, there are now data from both animals and humans to support the premise that central *emotional processing* is also altered by afferent input from the heart.

Afferent input influences emotional processing: The role of the amygdala

The influence of cardiovascular afferent input to the brain on emotional processes is highlighted by recent evidence suggesting that psychological aspects of panic disorder are often created by unrecognized paroxysmal supraventricular tachycardia (PSVT), a sudden-onset atrial arrhythmia. According to one study, DSM-IV criteria for panic disorder were fulfilled in more than two-thirds of patients with these sudden-onset arrhythmias. In those patients in whom PSVT was unrecognized at initial evaluation, symptoms were attributed to panic, anxiety, or stress in 54 percent of the cases. In the majority of cases, once the arrhythmia was recognized and treated, the panic disorder disappeared.⁷⁴ Interestingly, this study confirmed the observations of pioneer ANS researcher Müller, who reported the induction of emotions by cardiac palpitations over 90 years earlier.⁵⁰ Likewise, our research has also shown that changing the pattern of afferent information generated by the cardiovascular system can significantly influence perception and emotional experience.^{12,75}

The amygdala has been the subject of intense scrutiny in recent years. This brain center plays a key role in emotional memory, emotional processing, and dreaming.⁷⁶ Several studies have investigated the effects of cardiovascular afferent input on the amygdaloid complex (*i.e.*, the amygdala and associated nuclei). For example, in cats, spontaneous neural activity in the central nucleus of the amygdala has been shown to be synchronized to the cardiac cycle and to be modulated by afferent input from the aortic depressor and carotid sinus nerves.⁷⁷ Similarly, data from humans undergoing surgery for epilepsy demonstrated that cells within the amygdaloid complex specifically responded to information from the cardiac cycle.⁷⁸ Pribram, who did much of the original mapping of the functions of the amygdaloid complex, found it has extensive projections

to both the brain stem autonomic nuclei and the higher cognitive centers, and is thus uniquely placed to coordinate affective, behavioral, immunological, and neuroendocrine responses to environmental stimuli.^{16,79} The observed interaction of afferent cardiac input with this brain region supports the view that visceral information not only influences emotional processing and emotional experience, but can also influence hormonal and immune responses.⁷⁵

Taken together with the demonstrated role of the amygdala in the regulation of viscer-autonomic activity and the resultant effects on familiarization, considered below, a new view of emotional processing and regulation emerges.

The Role of Familiarization in Emotional Processing

To further unfold our understanding of the emotional system and the heart's role in emotion, we now review the model of emotion first developed by Karl Pribram.⁸⁰ Simply said, in Pribram's model a set of memories, or stable patterns of activity, is formed and maintained in the neural architecture of the brain as we gain experience both in internal self-regulation and in interacting with the external environment. These stable patterns are updated and modified as we encounter new experiences and learn how a certain action usually leads to specific result. All ongoing or current sensory input to the brain, from both the internal and external sensory systems, is compared to these stable patterns. When a mismatch between current input and a stable pattern occurs, novelty is sensed.

These stable patterns create a set of "expectancies" against which breathing, eating, drinking, sleeping, alerting, sexual, and other behaviors are evaluated. The stable neurological pattern acts as a set point against which an input is matched, and therefore determines what is familiar and what is novel, and perhaps exciting.

The set point, based on previous experience, becomes a reference point for evaluating current and future experience, and is biased or adjusted according to ongoing experience. To maintain stability as we encounter life's events, we must make adjustments that return us to the "familiar" set point. These adjustments require us to take an "action"—which can be either an outward action (*i.e.*, control of some kind over the external environment) or an internal adjustment (*i.e.*, self-control of our inner environment). Since our psychophysiological systems are designed to maintain stability and resist change, returning to familiar set points gives us a sense and feeling of security, while remaining in unfamiliar territory causes unrest. Interestingly, this is true even if the established set point is one of chaos and confusion.

Attention

No conscious awareness of anything, including our emotions, is possible until it has captured our attention. Sensory neurons in our eyes, ears, nose, and body are in continuous action, day and night, whether we are awake or asleep. The brain receives a steady stream of information about all the events the sense organs are capable of detecting. It would be bewildering if we were continuously aware of all the incoming information. In fact, we completely ignore most of the information arriving at the brain most of the time. Yet any input is capable of shifting and dominating our attention. In order for this process to function, there must be mechanisms and processes that direct *selective* attention. The attention mechanisms must continuously scan the available information and assign priority, usually based on biological importance. Large, sudden, novel occurrences typically have the ability to grab our attention. Emotions also have the ability to capture and focus attention, and attention is involved in the management of our emotional state.

In 1890, William James described attention thus:

Everyone knows what attention is. It is taking possession of the mind, in clear and vivid form, of one out of what seems several simultaneously possible objects or trains of thought. Focalization, concentration of consciousness, are of its essence. It implies withdrawal from some things in order to deal effectively with others, and is a condition which has a real opposite in the confused, dazed, scatterbrained state...⁸¹ (pp. 403-404)

Many laboratories around the world have investigated the brain structures involved in awareness and attention. Generally there have been two approaches to attention research: (1) recording physiological or behavioral responses against a background of regular, repeating sensory events and (2) pairing of the outcome of the response to sensory events.

When a new stimulus is presented to the brain, a change in activity in the central and autonomic nervous systems is produced. If the response is short-lived (1–3 seconds), it is called *arousal orienting reflex*. If, however, the stimulus or event is recurrent, the brain rapidly adapts and we *habituate*. For example, people who live in a noisy city adapt to the ambient noise and eventually become unaware of it. However, when they take a trip to the quiet countryside, the lack of noise seems strange and noticeable. Thus, any change in the stimulus will cause the reappearance of the arousal response, or the orienting reflex. The arousal reaction therefore reflects a *mismatch* between the new information and the familiar representation stored in the brain. A change in brain potentials can be measured during the arousal response to a novel stimulus, and is called *mismatch negativity*⁸² The observed changes in the nervous system can be sep-

arated into a *phasic* component, which habituates quickly, and a long-lasting *tonic* component, which habituates more slowly.⁸³

James, and more recently Pribram and McGuiness, also distinguished two types of attention. Pribram and McGuiness called these involuntary and voluntary. *Involuntary primary attention* as James called it, is provoked by certain classes of stimuli that are novel, salient, or intense, which impinge upon our awareness regardless of ongoing activity. *Voluntary attention*, on the other hand, describes the process whereby the individual voluntarily determines the contents of his/her own awareness and the duration of focus. In the Pribram and McGuiness model, the distinction between involuntary and voluntary attention identifies two aspects of attentional control: one regulates *arousal* resulting from a mismatch in sensory input; the other controls the preparatory *activation* of potential responses. In addition, there is a third aspect of attention that serves to coordinate involuntary arousal and voluntary activation, and this aspect of attention requires effort.²

Pattern-Matching and the Maintenance of Stability

In their book *Plans and the Structure of Behavior*, Miller, Galanter, and Pribram propose that in order for an organism to maintain continued stability, it must be able to maintain a match between its current experience or “reality” and its neural and hormonal set points and *programs*⁸⁴ These programs consist of hierarchies of nested neural feedback loops that maintain memories of familiar experiences, responses, and outcomes. Incongruities or differences in the input (new experiences) arouse or activate us depending upon the degree of mismatch, and, in most cases, determine what action is needed to reestablish stability. When the differences (mismatch) are of sufficient magnitude, there is a temporary discontinuity; importantly, *it is this discontinuity or mismatch—effectively a departure from the familiar—that gives rise to the experience of emotion*. In this context, it is interesting to note that the word “emotion” derives from the Latin *emovere*, which means “to move out or away from.”

Pribram, in his book *Languages of the Brain* carries the theory further. When the input to the brain does not match the existing program, an adjustment must be made in an attempt to achieve control and return to stability. One way to re-establish control is by taking an outward action. We are motivated to eat if we feel hungry, run away or fight if threatened, do something to draw attention to ourselves if feeling ignored, etc. Alternatively, we can re-establish stability and gain control by making an internal adjustment (without any overt action). For example, a confrontation at work may lead to feelings of anger, which can prompt inappropriate behavior (*i.e.*, outward actions such as yelling, hitting, etc.). However, through internal adjustments, we can self-manage our feelings in order to inhibit these responses, re-establish stability, and maintain our job.

Thus, stabilization is achieved through external action on the environment or through internal self-control. These processes are referred to, respectively, as motivational control and emotional control. Ultimately, when we achieve stability through our efforts, the results are feelings of satisfaction and gratification. By contrast, when there is a failure to achieve stability or control, feelings such as anxiety, panic, annoyance, apprehension, hopelessness, or depression result.

Pribram and many others have conducted numerous experiments providing evidence that these sorts of internal adjustments, although commonplace, represent a complex interplay between peripheral and central processes. For example, the afferent input systems and even their receptors are modulated by the central nervous system, which alters information processing in the sensory input channel.⁸⁶ In other words, the higher brain centers can inhibit or “gate” the information flowing into the brain. There are many examples of how we can control input channels. Where we focus our attention has a powerful effect on modulating inputs and thus on determining what gets processed at higher levels. In a noisy room filled with many conversations, we have the ability to tune out the noise and focus on a single conversation of interest. In a like manner, we can modulate pain from a stubbed toe or headache or desensitize ourselves to sensations like tickling.

Arousal

There is ample support that arousal, measured as EEG desynchronization, occurs in response to novel or unfamiliar input, and that arousal is one of the elements of emotional experience. In classical models of arousal theory, the amount of neural and/or hormonal activity generated in response to a given stimulus or event determines whether the experience leads to familiarization or disruption. Arousal theory states that a correlation exists between the amount of a specific hormone or amount of neural excitation and the amount of emotional arousal.

However, this is only part of the story. Arousal can at times be associated with an increase the *amount* of neural activity, but arousal can also occur without any increase in neural activity. In the latter case what does change, instead, is the *pattern* of activity in the nervous system (for example, variations in the time intervals between sequential firings of a neuron or group of neurons, or in which efferent pathways are active). Therefore, the amount of neural activity does not always necessarily indicate the level of arousal.⁸⁰ This is an important realization, as it shifts the focus from thinking in terms of amount of activity alone to understanding the importance of the pattern of activity. This is also related to the observation that differing emotions are reflected in the patterns

of the heart rhythm. For example, during an emotional state shift, the *pattern* of beat-to-beat heart rate variability can shift dramatically, while the *amount* of variability remains exactly the same. This is not to imply that changes in the amount of neural activity or amount of heart rate variability are not also important sources of information that contribute to ongoing emotional experience. However, in the broader context of the model presented here, such variations can also be considered as changes in pattern relative to a familiar baseline or set point.

The Role of the Heart

Monitoring the alterations in the rates, rhythms, and patterns of afferent traffic is a key function of the cortical and emotional systems in the brain. Pribram was well aware of the influence of afferent input from the heart and other organ systems in determining the set points, or what becomes the familiar pattern, as far back as 1969, when he wrote:

Visceral feedback constitutes, by the nature of its receptor anatomy and diffuse afferent organization, a major source of input to this biasing mechanism; it is an input which can do much to determine set-point. In addition, cardiovascular 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 which vary from occasion to occasion. Habituation to visceral and autonomic activity makes up, therefore, a large share...of the stable base-line from which the organism's reactions can take off.⁸⁰ (p. 322)

These set points establish a background against which blood pressure, hormonal balance, and all regularly recurring behaviors are initiated and maintained. For example, when we sense a mismatch between our actual heart rate and the habituated heart rate, we generate a feeling (*e.g.*, excitement or anxiety if heart rate is accelerated). The specific feeling experienced may reflect the nature of the mismatch. Importantly, a mismatch may be registered not only due to changes in heart rate but also due to changes in the pattern of the afferent traffic.

Although input originating from many different bodily organs and systems is involved in the processes that ultimately determine emotional experience, it has become clear that the heart plays a particularly important role. The heart is the primary and most consistent source of dynamic rhythmic patterns in the body. Furthermore, the afferent networks connecting the heart and cardiovascular system with the brain are far more extensive than the afferent systems associated with other major organs. Additionally, the heart is particularly sensitive

and responsive to changes in a number of other psychophysiological systems. For example, heart rhythm patterns are continually and rapidly modulated by changes in the activity of either branch of the ANS, and the heart's extensive intrinsic network of sensory neurons also enables it to detect and respond to variations in hormonal rhythms and patterns.⁸⁷ In addition to functioning as a sophisticated information processing and encoding center,⁸⁸ the heart is also an endocrine gland that produces and secretes hormones and neurotransmitters.⁸⁹⁻⁹² Thus, with each beat, the heart not only pumps blood, but also continually transmits dynamic patterns of neurological, hormonal, pressure, and electromagnetic information to the brain and throughout the body.⁹³ Therefore, the multiple inputs from the heart and cardiovascular system to the brain are a major contributor in establishing the dynamics of the baseline pattern or set point against which the "now" (current input) is compared.

The repeating rhythmic patterns generated by the heart, whether they are ordered or disordered, become familiar to the brain. At the brain stem level, these patterns are compared to set points that control blood pressure, affect respiration rate, and gate the flow of activity in the descending branches of the autonomic system. From there, these signals cascade up to a number of subcortical centers, such as the thalamus, hypothalamus, and amygdala, which are involved in the processing of emotion. With the understanding that the emotional system operates essentially as a pattern recognition system, the finding that a significant proportion of people diagnosed with panic disorder actually have an unrecognized atrial arrhythmia is easily understandable. When a sudden-onset arrhythmia occurs, there is a large and sudden change in the pattern of afferent signals arriving at the amygdala and hippocampus, resulting in a significant mismatch between the current input and the familiar, stable pattern. The system is unable to achieve stability through an outward action or through an internal adjustment; the mismatch therefore captures attention and gives rise to feelings of fear and anxiety, which build to panic. In cases where the arrhythmia is constant or occurs more frequently, the system adapts or habituates—in other words, the new input pattern becomes familiar.

On the other hand, a change in the pattern of afferent cardiovascular input that accompanies a more coherent or ordered heart rhythm, such as those that occur with certain breathing techniques or the use of HeartMath positive emotion-focused tools, results in a "pattern match" associated with security and positive emotional experience. These coherent rhythms are familiar to a "healthy" system as they have occurred spontaneously many times during sleep and positive emotional states. However, in many individuals, a coherent pattern is rare and relatively unfamiliar to the brain. In this case, with the practice of self-generating coherent rhythms, they become the familiar baseline pattern and that which the system attempts to maintain.

Emotional Instability

When the neural systems that maintain the baseline reference patterns are in an unstable state (due to stress, anxiety, chemical stimulants, etc.), sensory input from either internal or external sources that would ordinarily be processed smoothly can be perceived as a mismatch and give rise to an uncomfortable feeling. Thus, patterns of neural activity in the brain can effectively predispose the individual towards either stability or instability. The reference patterns can be temporarily destabilized by large, sudden changes in the pattern of afferent activity, such as those that occur in the example of a sudden onset arrhythmia or during an emotionally charged situation. If a reference pattern is destabilized, a mismatch can be perceived even in the absence of novel input. This explains why we can have an upsetting interaction with our spouse, and even though things may have been smoothed over and the event consciously forgotten, we could subsequently be set off by what we perceive as a funny look from a co-worker upon arriving at the office. Physiologically, the instability is still in our system. Under normal circumstances, the look would have gone unnoticed. Likewise, had we been able to stabilize our neural systems by clearing the emotional residue on the way to work, the look from the co-worker would not have thrown us off.

In addition to processes that monitor the input and controls for maintaining stability (pattern matching) in the here-and-now, there are also matching processes that appraise the degree of congruity or incongruity between the past and the now and between the now and the projected future. Furthermore, these prospective appraisals can be divided into optimistic and pessimistic.⁹⁴ If the appraisal does not result in a projected ability to return to stability, feelings of fear and anxiety can result. This appraisal could be due to past experience of similar situations or a lack of experience in the projected future situation. However, as we encounter novel situations and learn that we are able to maintain stability, we can apply that experience to similar future situations without fear.

Pribram states that when a homeostatic system becomes stabilized and a new pattern has become familiar, new sensitivities develop and different strategies and programs are added to handle the acquired sensitivities.⁹⁵ In essence, we mature. Encountering novel situations or obstacles requires that we develop new strategies: we either take an external action to gain control or self-manage our internal systems. Once we learn how to handle the new challenge effectively and maintain stability, the strategy (complex pattern) for dealing with the challenge becomes familiar and part of our repertoire. Through this process, we increase our internal self-control and management of emotions as well as our ability to effectively deal with external situations.

The baseline patterns maintained in the neural architecture are modified by

other sources of neural and hormonal input that affect the “bias” or sensitivity of the system. Because the neural systems involved in comparing the incoming sensory information are made up of short, fine fibers with many branches, they are especially sensitive to hormonal influences. Thus, the system is readily affected by changes in the patterns of hormonal input associated with different psychophysiological states. In this way hormones provide important influences on the brain processes involved in the experience of emotion.

The Making of Emotions: A Converging View

In summary, we can see earlier theories of emotion, coupled with current research, converging into a more complete and comprehensive view of emotions. Endocrine research significantly advanced the previous view of emotions as “humors.” The visceral theory acknowledged an arousal mechanism that provides feelings of interest, novelty, and familiarity, as well as more painful disruptions of stable states. James emphasized the communication of bodily responses to the brain. Cannon’s thalamic theory contributed by offering evidence of the thalamus as a prime locus for processing emotional information from the body’s chemical homeostatic systems. Papez and MacLean introduced the idea of emotional circuits and systems instead of a single center and added the possibility of a memory component to the emotional system. With Pribram’s cortical control of afferent input and monitoring of a departure from stable, familiar patterns, it becomes clear that *both the brain and the entire body are involved in the full experience and expression of emotions*

With this understanding in mind, we can view the experience of emotion as emerging from an intricate array of interactions occurring within a complex system. Broadly speaking, its main components include the brain and nervous system, the hormonal system, and body. Although there are numerous sources of bodily input to the brain, the heart is given particular relevance in the emotional system due to its unique degree of afferent input and its consistent generation of dynamic rhythmic patterns that are closely coupled with changes in emotional state. From a generalized perspective, one of the ways an emotion is generated is through the comparison of information received from the external sensory systems, (e.g., sights, sounds, and smells) against pre-existing memories. This processing occurs at unconscious levels, unless attention is captured, and results in changes in the patterns of descending autonomic activity flowing to the body. This leads to a wide variety of specific changes in biochemical outputs and biophysical states, such as alterations in patterns of muscle tension (especially in the face), adrenal secretions, vascular resistance, cardiac output, and heart rhythms. These alterations, in turn, result in changes in the afferent

inputs from the body back to the brain, which are then compared to a set of pre-existing reference patterns. This ascending bodily input is crucial to the felt experience of an emotion, and may or may not reinforce the cognitive level appraisal and labelling of the feeling. The process continues as the system makes external and internal adjustments in order to maintain stability, and, depending upon the outcome, can further color and add textures to the emotional experience. Of course, this is only one example, as the process can also be initiated by changes in the internal systems alone as well as through many combinations of the internal and external sensory systems' interactions with the reference patterns and memories.

Within the context of the model of emotion developed here, we can also gain new insight into the mechanisms underlying the efficacy of the HeartMath emotional restructuring techniques, which produce a positive emotion-driven shift in the heart's rhythmic patterns, and thus a change in the pattern of cardiac afferent input to the brain. The coupling of a more organized pattern of afferent input with an intentionally self-generated positive emotion reinforces the natural conditioning between the coherent physiological mode and the positive emotion. This subsequently strengthens the ability of a positive emotional shift to initiate a physiological shift towards increased coherence, and a physiological shift to facilitate the experience of a positive emotion.

From the perspective presented in this article, HeartMath interventions affect several aspects of the emotional process. First, by reducing nervous system chaos, they stabilize the neural systems that maintain the baseline or reference patterns against which incoming information is compared. They also modify the baseline patterns by reinforcing the coherent psychophysiological patterns associated with positive emotions and allowing these patterns to become familiar, thus effectively establishing a new baseline or norm. Once this new reference pattern established, the system then automatically strives to maintain this state.

With practice of these techniques, as the neural architecture comes to recognize the patterns associated with coherent heart rhythms as familiar, it becomes progressively easier to intentionally generate coherent rhythms and their psychophysiological benefits, even during experiences of stress or challenge. Moreover, we have demonstrated that as people continue to practice intentionally self-generating states of psychophysiological coherence using heart-based techniques, they also begin to demonstrate a greater frequency of *spontaneous* heart rhythm coherence, without conscious use of the interventions. These data support the concept the techniques facilitate an actual re-patterning process at the level of the neural architecture, which can be objectively assessed using electrophysiological measures.

In sum, consistent use of heart-based positive emotion-focused techniques reinforces existing neural pathways that the brain uses to control its input

(self-manage) and facilitates the establishment of new control pathways, thus improving our ability to self-manage our emotions and regulate our physiological state. Experientially, the occurrence of a system-wide re-patterning process with consistent use of the HeartMath interventions is supported by reports from thousands of individuals who have noted enduring improvements in many aspects of health, well-being, and performance, increased emotional stability and new capabilities for dealing with stress and challenges. In a very real sense, we become the architects of our own neural landscape.

Acknowledgments

I would like to express my appreciation to Dr. Karl Pribram for his careful review of this monograph and his insightful input on its content.

References

1. Goleman D. *Working with Emotional Intelligence* New York: Bantam Books, 1998.
2. Pribram KH, McGuinness D. Arousal, activation, and effort in the control of attention. *Psychological Review* 1975;82(2):116-149.
3. LeDoux JE. Cognitive-emotional interactions in the brain. In: Ekman P, Davidson RJ, eds. *The Nature of Emotion: Fundamental Questions*. New York: Oxford University Press, 1994: 216-223.
4. LeDoux J. *The Emotional Brain: The Mysterious Underpinnings of Emotional Life*. New York: Simon and Schuster, 1996
5. Pert CB, Dreher HE, Ruff MR. The psychosomatic network: Foundations of mind-body medicine. *Alternative Therapies in Health and Medicine* 1998;4(4):30-41.
6. Damasio AR. *The Feeling of What Happens*. Orlando, FL: Harcourt, 1999.
7. Damasio AR. *Descartes' Error: Emotion, Reason and the Human Brain*. New York: G.P. Putnam's Sons, 1994.
8. Bower GH. Mood-congruity of social judgements. In: Forgas J, ed. *Emotions and Social Judgements*. Oxford: Pergamon, 1990.
9. Bower GH. How might emotions affect learning? In: Christianson SA, ed. *The Handbook of Emotion and Memory: Research and Theory*. Hillsdale, NJ: Lawrence Erlbaum, 1992: 3-31.
10. Clore GC. Why emotions are felt. In: Ekman P, Davidson RJ, eds. *The Nature of Emotion: Fundamental Questions*. New York: Oxford University Press, 1994: 103-111.
11. Descartes R. *Treatise on the Passions of the Soul* In: Haldane E, Ross G, trans. *The Philosophical Works of Descartes*. Cambridge: Cambridge University Press, 1649.

12. McCraty R, Childre D. *The appreciative heart: The psychophysiology of positive emotions and optimal functioning*. Boulder Creek, CA: HeartMath Research Center, Institute of HeartMath, Publication No. 02-026, 2002.
13. Childre D, Martin H. *The HeartMath Solution* San Francisco: HarperSanFrancisco, 1999.
14. Childre D, Rozman D. *Overcoming Emotional Chaos: Eliminate Anxiety, Lift Depression and Create Security in Your Life*. San Diego: Jodere Group, 2002.
15. Tiller WA, McCraty R, Atkinson M. Cardiac coherence: A new, noninvasive measure of autonomic nervous system order. *Alternative Therapies in Health and Medicine* 1996;2(1):52-65.
16. Pribram KH. Emotions: A neurobehavioral analysis. In: Scherer KR, Ekman P, eds. *Approaches to Emotion* Hillsdale, NJ: Erlbaum, 1984.
17. James W. What is an emotion? *Mind* 1884; 9(34): 188-205.
18. Ellsworth PC. William James and emotion: Is a century of fame worth a century of misunderstanding? *Psychological Review* 1994; 101(2):222-229.
19. Cannon WB. The James-Lange theory of emotion: A critical examination and an alternative theory. *American Journal of Psychology* 1927;39: 106-124.
20. Marshall LH, Magoun HW. *Discoveries in the Human Brain*. Totowa, NJ: Humana Press, 1998.
21. Cannon WB. *Bodily Changes in Pain, Hunger, Fear and Rage: An Account of Recent Researches into the Function of Emotional Excitement*, 2nd edition. New York: D. Appleton & Company, 1929.
22. Lindsley DB. Emotion. In: Stevens SS, ed. *Handbook of Experimental Psychology*. New York: Wiley, 1951: 473- 516.
23. Papez JW. A proposed mechanism of emotion. *Archives of Neurological Psychiatry* 1937;38: 725-743.
24. Hess WR. *Diencephalon: Autonomic and Extrapyrarnidal Functions*. New York: Grune & Stratton, 1954.
25. Lashley KS. The thalamus and emotion. In: Beach FA, Hebb DO, Morgan CT, Nissen HS, eds. *The Neuropsychology of Lashley*. New York: McGraw-Hill, 1960.
26. MacLean PD. Psychosomatic disease and the "visceral brain": Recent developments bearing on the Papez theory of emotion. *Psychosomatic Medicine* 1949; 11:338-353.
27. MacLean PD. Some psychiatric implications of physiological studies on frontotemporal portion of limbic system (visceral brain). *Electroencephalography and Clinical Neurophysiology* 1952;4:407-418.
28. MacLean PD. The triune brain, emotion and scientific bias. In: Schmitt FO, ed. *The Neurosciences: Second Study Program*. New York: Rockefeller University Press, 1970: 336-349
29. MacLean PD. *The Triune Brain in Evolution: Role in Paleocerebral Functions*.

New York: Plenum, 1990.

30. Butler AB, Hodos W. *Comparative Vertebrate Neuroanatomy: Evolution and Adaptation*. New York: Wiley-Liss, 1996
31. Flanigan S, Gabrieli ER, MacLean PD. Cerebral changes revealed by radioautography with S35-labeled l-methionine. *Archives of Neurological Psychiatry* 1957; 77:588-594.
32. Kaada BR, Pribram KH, Epstein JA. Respiratory and vascular responses in monkeys from temporal pole, insula, orbital surface and cingulate gyrus. *Journal of Neurophysiology* 1949;12:347-356.
33. Seung HS. Half a century of Hebb. *Nature Neuroscience* 2000; 3:1166.
34. Abbott LF, Nelson SB. Synaptic plasticity: Taming the beast. *Nature Neuroscience* 2000; 3:1178-1183.
35. Green E, quoted in CB Pert, *Molecules of Emotion* New York: Scribner, 1997, p. 137.
36. Schachter S, Singer JE. Cognitive, social, and physiological determinants of emotional state. *Psychological Review* 1962;69:379-399.
37. Gellhorn E, Loofbourrow GN. *Emotions and Emotional Disorders: A Neurophysiological Study*. New York: Harper & Row, 1963.
38. Malmo RB, Shagass C, Davis FH. Symptom specificity and bodily reactions during psychiatric interview. *Psychosomatic Medicine* 1950; 12:362-376.
39. Ax AF. The physiological differentiation between fear and anger in humans. *Psychosomatic Medicine* 1953; 15: 433-442
40. Schachter J. Pain, fear, and anger in hypertensives and normotensives: A psychophysiological study. *Psychosomatic Medicine* 1957; 15:17-29.
41. Graham DT, Stern JA, Winokur G. Experimental investigation of the specificity of attitude hypothesis in psychosomatic disease. *Psychosomatic Medicine* 1958; 20:446-457.
42. Levenson RW, Ekman P, Friesen WV. Voluntary facial action generates emotion-specific autonomic nervous system activity. *Psychophysiology* 1990; 27:363-384.
43. Ekman P, Levenson RW, Friesen WV. Autonomic nervous system activity distinguishes among emotions. *Science* 1983; 221(4616):1208-1210.
44. Hubert W, de Jong-Meyer RD. Psychophysiological response patterns to positive and negative film stimuli. *Biological Psychology* 1991; 31(1):73-93.
45. Collet C, Vernet-Maury E, Delhomme G, Dittmar A. Autonomic nervous system response patterns specificity to basic emotions. *Journal of the Autonomic Nervous System* 1997; 62:45-57.
46. Sinha R, Lovallo WR, Parsons OA. Cardiovascular differentiation of emotions. *Psychosomatic Medicine* 1992; 54(4): 422-435.
47. Leek BF. Abdominal visceral receptors. In: Neil E, ed. *Handbook of Sensory Physiology*, Vol. 3. Heidelberg: Springer, 1972: 113-160.

48. Andrews PLR. Vagal afferent innervation of the gastrointestinal tract. *Progress in Brain Research* 1986; 67:65-86.
49. Neundörfer B, Hilz MJ. Ludwig Robert Müller (1870- 1962) – A pioneer of autonomic nervous system research. *Clinical Autonomic Research* 1998; 8:1-5.
50. Müller LR. Ueber die Beziehung von seelischen Empfindungen zu Herzstörungen. *Münchener Medizinische Wochenschrift* 1906; 53:14-16.
51. Heymans C, Neil E. *Reflexogenic Areas of the Cardiovascular System*. Boston: Little, Brown, 1958.
52. Lacey JI, Lacey BC. Some autonomic-central nervous system interrelationships. In: Black P, ed. *Physiological Correlates of Emotion*. New York: Academic Press, 1970: 205-227.
53. Bonvallet M, Allen MB. Prolonged spontaneous and evoked reticular activation following discrete bulbar lesions. *Electroencephalography and Clinical Neurophysiology* 1963; 15:969-988.
54. Gahery Y, Vigier D. Inhibitory effects in the cuneate nucleus produced by vago-aortic afferent fibers. *Brain Research* 1974; 75:241-246.
55. Randich A, Gebhart GF. Vagal afferent modulation of nociception. *Brain Research Reviews* 1992; 17:77-99.
56. Drinkhill MJ, Mary DA. The effect of stimulation of the atrial receptors on plasma cortisol level in the dog. *Journal of Physiology* 1989; 413:299-313.
57. Rau H, Pauli P, Brody S, Elbert T. Baroreceptor stimulation alters cortical activity. *Psychophysiology* 1993; 30: 322-325.
58. Sandman CA, Walker BB, Berka C. Influence of afferent cardiovascular feedback on behavior and the cortical evoked potential. In: Cacioppo JT, Petty RE, eds. *Perspectives in Cardiovascular Psychophysiology*. New York: The Guilford Press, 1982: 189-222.
59. van der Molen MW, Somsen RJM, Orlebeke JF. The rhythm of the heart beat in information processing. In: Ackles PK, Jennings JR, Coles MGH, eds. *Advances in Psychophysiology*, Vol. 1. London: JAI Press, 1985: 1-88.
60. Schandry R, Montoya P. Event-related brain potentials and the processing of cardiac activity. *Biological Psychology* 1996; 42:75-85.
61. Montoya P. *Herzwehrnehmung und hirnelektrische Aktivität. Eine Analyse der topographischen Verteilung von herzs Schlag-synchron evozierten Potentialen (HEP)*. Frankfurt/Main: Peter Lang, 1994.
62. Rosenfeld SA. *Conversations between heart and brain* Rockville, MD: National Institute of Mental Health, 1978.
63. Lacey JI. Psychophysiological approaches to the evaluation of psychotherapeutic process and outcome. In: Rubinstein E, Parloff M, eds. *Research in Psychotherapy* Washington, DC: American Psychological Association, 1959: 160-208.
64. Lacey JI, Kagan J, Lacey BC, Moss HA. The visceral level: Situational determi-

- nants and behavioral correlates of autonomic response patterns. In: Knapp PH, ed. *Expression of the Emotions in Man*. New York: International Universities Press, 1963: 161-196.
65. Lacey JI. Somatic response patterning and stress: Some revisions of activation theory. In: Appley MH, Trumbull R, eds. *Psychological Stress: Issues in Research*. New York: Appleton-Century-Crofts, 1967: 14-42.
 66. Lacey BC, Lacey JI. Studies of heart rate and other bodily processes in sensorimotor behavior. In: Obrist PA, Black AH, Brener J, DiCara LV, eds. *Cardiovascular Psychophysiology: Current Issues in Response Mechanisms, Biofeedback, and Methodology*. Chicago: Aldine, 1974: 538-564.
 67. Israel NR. Cognitive control and pattern of autonomic response. Paper presented at meeting of the Eastern Psychological Association, Washington, D.C., April, 1968.
 68. Israel NR. Leveling-sharpening and anticipatory cardiac response. *Psychosomatic Medicine* 1969; 31:499-509.
 69. Koriath JJ, Lindholm E. Cardiac-related cortical inhibition during a fixed foreperiod reaction time task. *International Journal of Psychophysiology* 1986; 4:183-195.
 70. Walker BB, Sandman CA. Visual evoked potentials change as heart rate and carotid pressure change. *Psychophysiology* 1982; 19(5):520-527.
 71. Schandry R, Sparrer B, Weitkunat R. From the heart to the brain: A study of heartbeat contingent scalp potentials. *International Journal of Neuroscience* 1986; 30:261-275.
 72. Montoya P, Schandry R, Muller A. Heartbeat evoked potentials (HEP): Topography and influence of cardiac awareness and focus of attention. *Electroencephalography and Clinical Neurophysiology* 1993; 88:163-172.
 73. Schandry R, Leopold C. The severity of diabetic autonomic neuropathy is reflected in the heartbeat evoked brain potential [abst.]. *Clinical Autonomic Research* 1997; 7(5): 249-250.
 74. Lessmeier TJ, Gamperling D, Johnson-Liddon V, Fromm BS, Steinman RT, Meissner MD, Lehmann MH. Unrecognized paroxysmal supraventricular tachycardia: potential for mis-diagnosis as panic disorder. *Archives of Internal Medicine* 1997; 157:537-543.
 75. McCraty R, Barrios-Choplin B, Rozman D, Atkinson M, Watkins AD. The impact of a new emotional self-management program on stress, emotions, heart rate variability, DHEA and cortisol. *Integrative Physiological and Behavioral Science* 1998; 33(2):151-170.
 76. Aggleton JP, ed. *The Amygdala: Neurobiological Aspects of Emotion, Memory and Mental Dysfunction*. New York: Wiley-Liss, 1992.
 77. Zhang JX, Harper RM, Frysinger RC. Respiratory modulation of neuronal discharge in the central nucleus of the amygdala during sleep and waking

- states. *Experimental Neurology* 1986; 91:193-207.
78. Frysinger RC, Harper RM. Cardiac and respiratory correlations with unit discharge in epileptic human temporal lobe. *Epilepsia* 1990;31(2):162-171.
 79. Pribram KH, Bagshaw MH. Further analysis of the temporal lobe syndrome utilizing fronto-temporal ablations. *Journal of Comparative Neurology* 1953; 99:347-375.
 80. Pribram KH, Melges FT. Psychophysiological basis of emotion. In: Vinken PJ, Bruyn GW, eds. *Handbook of Clinical Neurology*. Amsterdam: North-Holland Publishing Company, 1969: 316-341.
 81. James W. *Principles of Psychology*. New York: Holt, 1890
 82. Näätänen R. *Attention and Brain Function*. Hillsdale, NJ: Lawrence Erlbaum Associates, 1992.
 83. Sharpless S, Jasper H. Habituation of the arousal reaction. *Brain* 1956;79:655-680.
 84. Miller GA, Galanter EH, Pribram KH. *Plans and the Structure of Behavior*. New York: Henry Holt & Co., 1960
 85. Pribram KH. The new neurology and the biology of emotion: A structural approach. *American Psychologist* 1967; 22(10):830-838.
 86. Pribram KH. *Languages of the Brain*. New York: Brandon House, 1971.
 87. Armour JA. Peripheral autonomic neuronal interactions in cardiac regulation. In: Armour JA, Ardell JL, eds. *Neurocardiology*. New York: Oxford University Press, 1994: 219-244
 88. Armour JA. *Neurocardiology—Anatomical and functional principles*. Boulder Creek: CA, HeartMath Research Center, Institute of HeartMath, Publication No. 03-011, 2003
 89. Cantin M, Genest J. The heart as an endocrine gland. *Scientific American* 1986; 254(2):76-81.
 90. Mukoyama M, Nakao K, Hosoda K, Suga S, Saito Y, Ogawa Y, Shirakami G, Jougasaki M, Obata K, Yasue H, et al. Brain natriuretic peptide as a novel cardiac hormone in humans. Evidence for an exquisite dual natriuretic peptide system, atrial natriuretic peptide and brain natriuretic peptide. *Journal of Clinical Investigation* 1991; 4:1402-1412.
 91. Gutkowska J, Jankowski M, Mukaddam-Daher S, Mc-Cann SM. Oxytocin is a cardiovascular hormone. *Brazilian Journal of Medical and Biological Research* 2000; 33: 625-633.
 92. Huang M-H, Friend DS, Sunday ME, Singh K, Haley K, Austen KF, Kelly RA, Smith TW. An intrinsic adrenergic system in mammalian heart. *Journal of Clinical Investigation* 1996;98(6):1298-1303.
 93. McCraty R, Atkinson M. *Psychophysiological coherence* Boulder Creek, CA: HeartMath Research Center, Institute of HeartMath, Publication 03-016, 2003.

94. Pribram KH. Feelings as monitors. In: Arnold MB, ed. *Feelings and Emotions*. New York: Academic Press, 1970: 41-53.
 95. Pribram KH. Reinforcement revisited: A structural view. In Jones MR, ed. *Nebraska Symposium on Motivation* Lincoln: University of Nebraska Press, 1963: 113-159.
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