Social and Emotional Self-Regulation[a]

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The assumption of some form of frontal dysfunction in emotional disorder has long been important in psychiatry. In the United States during the 1940s and 1950s, this assumption led to many thousands of frontal lobotomies, leukotomies, and tractotomies for the treatment of affective and psychological dysfunctions.[1,2] Yet it was clear at the time that the scientific evidence relating these deliberate frontal lesions to psychiatric symptoms was thin at best.[3] The rationale was that psychosurgery treated the psychotic process by disrupting the "fixed" pathological ideation.[1,2] In fact, however, although orbital frontal and anterior cingulate lesions reliably decreased the symptoms of anxiety and depression, the clinical outcome studies of this era showed consistently that the psychotic disorder of schizophrenic patients was unchanged by the procedure.[4]

In Sweden, physicians evaluated the effects of lobotomy by talking with the patients' family members. The damage to personality was clear.

> The wife of patient 2 says, "Doctor, you have given me a new husband. He isn't the same man." The mother of patient 4 declares, "She is my daughter but yet a different person. She is with me in body but her soul is in some way lost. Those deep feelings, the tendernesses are gone. She is hard, somehow." The brother of patient 3, a clergyman, states that her personality is altered; her interests, her outlook on life, her behavior, are different. "I have lost my husband. I'm alone. I must take over all responsibilities now," says the wife of a schoolteacher. "I'm living with another person," says the friend of patient 7. "She is shallow in some way."[5] (p. 695)

With naturally occurring frontal lesions, such as from stroke or head injury, the psychosocial deficits are often unappreciated by clinicians until they have led to major failures in occupational and social adjustment.[6] For example, one patient appeared to have been a model citizen prior to sustaining a ventromedial frontal injury.[7] After the injury, although this patient scored well above average on standard tests of intelligence, he soon lost his job, his money, and his marriage.

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An effective theory of human frontal lobe function must be able to explain the complex psychological and social skills that are impaired in such patients. The level of psychological description must go beyond the familiar concepts of cognitive neuroscience, such as spatial attention or working memory, and enter the domain of personality. On the other hand, the theoretical challenge at the neural level is to go beyond labeling the functions of the frontal lobe to formulate the key neurophysiological mechanisms. These mechanisms link the operations of frontal cortex to the multiple systems of the brain's control hierarchy, ranging from the control of arousal by brain-stem projection systems to the control of memory by reentrant corticolimbic interactions. When sufficiently understood, these mechanisms must be found to regulate not only the physiology of neural tissue, but the representation and maintenance of the self.

In this paper, we consider the social and emotional functions of the frontal lobe in terms of three anatomical dimensions. The first might be described as the "vertical" dimension because it emphasizes the integration of the lower functions—brain stem and limbic—with the highest operations—cognitive and motor planning—of the frontal neocortex. For this dimension, we provide a brief overview of theoretical approaches to vertical integration. Frontal lesions may disrupt self-regulation at the most elementary level by impairing the capacity to engage and maintain adequate levels of activation and arousal in service of long-range goals. At more complex levels, the adaptive control of frontal lobe contributions to attention and memory may be traced to the limbic networks that form the adaptive base for the operations of frontal neocortex.

The second dimension examines the functional differentiation between the dorsal and ventral anatomical pathways linking frontal cortex to the limbic structures, reflecting the dual origins of frontal cortex in the archicortical and paleocortical divisions of paralimbic cortex. We consider the differing clinical syndromes resulting from lesions to these pathways. Dorsomedial lesions may lead to apathy and a loss of initiative. Orbital (ventral) lesions may be more likely to lead to behavioral disinhibition. We interpret these syndromes in terms of a theory of differing motivational biases that shape the differential forms of motor control emerging in the dorsal and ventral pathways.

The third anatomical dimension is lateral, reflecting hemispheric specialization for emotion. We review the increasing evidence that the left and right frontal lobes contribute differently to emotional self-regulation. This evidence includes not only brain lesion studies, but brain function studies with both normal and psychiatric subjects using EEG, cerebral blood flow, and cerebral metabolism measures. The recent blood flow studies are particularly important in addressing the key theoretical issue of whether the emotional effect of a hemispheric lesion results from the disinhibition of the opposite hemisphere, or the disinhibition of the ipsilateral subcortical structures.

Although these three dimensions may seem to divide the frontal lobes along separate axes, they are not necessarily independent. The dorsal and ventral pathways may incorporate different forms of activation and arousal control, leading to different modes of vertical integration. Furthermore, lateral specialization for both cognition and emotion may involve differential elaboration of the dorsal and

ventral corticolimbic pathways within each hemisphere, leading to different patterns of frontolimbic interaction on the two sides of the brain.

CONCEPTS OF VERTICAL INTEGRATION

To frame the problem of self-control in terms of the relevant neurophysiological systems is to face the theoretical problem of vertical integration. This is the problem of functionally coordinating the multiple levels of the vertebrate neural hierarchy, from brain stem through midbrain, striatal, and limbic to the extensive paralimbic and neocortical networks.[8] The frontal cortex appears able to recruit the multiple levels of the hierarchy in support of extended, goal-directed behavior. Whereas the perceptual systems of the posterior cortex are dedicated to *representational* operations, developing the internal model of the environmental context within each sensory modality, the networks of the frontal cortex are uniquely suited to achieve *regulatory* operations, linking the multiple levels of the neural hierarchy in service of effectively motivated actions.[9] In the human brain, the extensive frontal cortex also provides representational capacity—working memory—that is dedicated not just to the representation of the sensory context, but to a complex and flexible organization of the regulatory functions across the neural hierarchy. In this sense, the working memory of the frontal lobe could be described as the representation of the regulatory process. The most basic level of the regulatory process is the control of arousal.[10]

Self-Regulation through Activation and Arousal

Observations of frontal-lesioned patients have suggested that frontal cortex plays an integral role in the self-regulation of arousal in light of behavioral demands.[11] Mechanisms of this control may include both influences on brain-stem neuromodulator projection systems and frontal regulation of nonspecific thalamic projections.[12]

Psychological concepts of arousal have typically considered autonomic signs as the critical component.[13,14] The concept of the brain-stem reticular activating system was important in moving beyond the nineteenth century notion of arousal as a visceral mechanism, in order to consider neural mechanisms that regulate alertness as a function of both external and internal events.[15] However, even when framed within neurophysiological terms, a unidimensional construct of arousal has proven inadequate to account for the range of specific controls on the activity and attentional capacity of the brain. An important theoretical challenge has been to find concepts that bridge between the control of level of neural activity and the control of qualitative features of attention and memory.

Pribram and McGuinness[16] differentiated between an *arousal* system, centered on the amygdala, that responds in a phasic fashion to changes in stimulus input and an *activation* system, centered on the basal ganglia, that maintains the motor circuitry in preparation for action. In addition, an *effort* system regulated by the hippocampus was proposed to coordinate between arousal and activation.

Building upon this formulation, Tucker and Williamson[17] theorized how qualitative changes in attention could be produced by brain-stem neuromodulator systems regulating activation and arousal. Operating to apply a *redundancy bias* on working memory, the dopaminergic activation system routinizes actions and focuses attention. Operating under an opposite control system principle, a *habituation bias*, the noradrenergic arousal system allocates attention to a broad array of novel events, leading to an expansive, holistic perceptual mode.

Although these models remain controversial, they provide ways of understanding how elementary neurophysiological mechanisms could have fundamental psychological roles for the self-regulation not only of attention and cognition, but of personality. For example, a person who relies strongly on the phasic arousal system for self-control would be strongly regulated by external events. A child with this dominant mode of arousal control may be described as having an attention deficit.[18] An adult whose personality was dominated by this mode may be described as extraverted.[17]

The frontal lobe may fine-tune these qualitative controls on attention in accordance with ongoing adaptive demands. For example, lesions of the right frontal lobe may result in particularly severe cases of the neglect syndrome, in which the patient ignores objects, and even body parts, in the half of sensory space opposite to the lesion.[19] This syndrome appears to involve dysfunction of brain-stem, thalamic, and cortical alerting systems.[19] Positron emission tomography (PET) studies of attention have shown increased blood flow in right frontal cortex in a number of experiments that require orienting to targets.[20] The noradrenergic (NE) brain-stem projection system courses through the frontal lobe before projecting caudally to posterior cortex, primarily of the dorsal (archicortical) pathway.[21] For human attention, Tucker and Williamson[17] proposed that the NE phasic arousal system is particularly important to the holistic attentional mode of the right hemisphere. Although many issues remain to be worked out, theoretical models that link frontal control to arousal regulation provide ways of understanding the deficits of motivation and initiative that may follow frontal lesions.[22] Further clarification of theoretical issues may help explain the normal role of frontal cortex in recruiting the appropriate state of arousal and alertness in service of effective behavior.

Corticolimbic Network Architecture

On the basis of both lesion and stimulation evidence and considering the connectivity of frontal cortex, Pribram and his associates have theorized that the frontal lobe is essential to integrating complex behavior because it represents the neocortical extension of the limbic system.[23–25] Recent research continues to confirm that areas of frontal cortex are closely connected to autonomic responses, as are areas of paralimbic cortex.[26] In addition, areas of frontal cortex appear to be important in integrating kinesthetic information with ongoing behavior, and impairment in kinesthetic processing may be a factor in the learning deficits of monkeys with frontal lesions.[27]

Any theory of the motivational basis of the function of the frontal lobe must consider the extensive frontal-limbic connectivity. For example, Nauta's formula-

tion considered the limbic structures as providing interoceptive reference points to serve as adaptive guides for the frontal lobe's direction of behavior.[28] In a manner similar to Teuber's corollary discharge to perceptual systems,[29] an "efferent copy" of a frontal action plan could be evaluated in terms of the limbic reference points for desired outcomes.[28]

In the cognitive neuroscience model of today's research, the function of the frontal lobe is often considered in terms of working memory.[30] Corticolimbic connections are essential for memory as well as emotion. In monkeys, disruption of the cortical pathways linking perceptual systems to the hippocampus, amygdala, and associated paralimbic cortices results in severe memory impairments.[31,32] In humans, the amnesia syndromes can be traced to damage to limbic structures and paralimbic cortex.[33] Corticolimbic connections are often thought of in terms of the perceptual operations of the posterior brain, but they must also be important in guiding action on the basis of experience as well. The frontal neocortex shows a pattern of connectivity that links it to the archicortical and paleocortical paralimbic cortices, just as for the posterior brain.[34] However, the primary direction of information flow for the posterior brain appears to be from neocortical (sensory cortex) to limbic, whereas the primary flow for the anterior brain is from limbic to neocortical (primary motor).[35] An interesting theoretical question is whether the mechanisms of memory operate differently when the dominant direction of control reverses in the corticolimbic pathways.

Recognizing the dual functions of the limbic networks—memory and emotion—suggests important possibilities for theoretical insight into the adaptive base of frontal lobe function. To relate the anatomical and functional evidence to a theory of the cognition of the human frontal lobe, two theoretical questions arise. First, what does it mean that the networks that are most critical for consolidating memory (the paralimbic cortices) are also those that represent kinesthetic and visceral information? Second, how do these primitive regions of cortex shape the organization and control of actions?

For both these questions, psychological theory has provided an important perspective. Heinz Werner proposed that in the child's primitive, syncretic perception, motor attitudes, and bodily feelings form the elementary substrate for experience, the "postural-affective matrix." From this primitive experiential basis, specific thoughts and actions become articulated through a progressive developmental process.[36] Although the articulation becomes more differentiated in the adult's cognition, Werner believed that the adult's cognition still begins at a syncretic, primitive level of organization and becomes progressively articulated into each discrete act. This developmental process is said to be microgenetic, because it occurs within the milliseconds required for the formation of each thought and action.

Drawing from Werner's model, Brown[37] theorized that specific forms of apraxia (motor disorder) result from specific frontal lobe lesions because they represent the disruption of specific stages in the microgenetic process: frontal limbic lesions impair the initiation of actions; prefrontal lesions impair the direction and organization of actions once they are initiated; premotor and precentral lesions impair the final articulation of the action sequence. Each level of the reentrantly connected

limbic-cortical progression thus serves to differentiate the action from the primitive motivational impetus.

In reviewing the theoretical issues in the research on frontal lobe anatomy and function, Pribam[27] proposed that Brown's microgenetic model provides a useful model for the development of motor plans across the linked limbic-neocortical networks of the frontal lobe. A microgenetic account might also explain the integration of visceral and kinesthetic representations within the organization of action programs. The frontal cortex may mediate between the interoceptive state represented within limbic networks and the external context as it is interfaced by primary sensory and motor cortices.[27,38]

Derryberry and Tucker[39] developed this line of reasoning by considering the computational architecture of the mammalian cortex. Within a connectionist or parallel-distributed model of information processing, the representational function of a network can be inferred in large part from its pattern of connectivity. The anatomical studies of Pandya and Yeterian[40] have shown that the interconnection among widespread cortical regions is sparse for neocortical networks (which therefore appear to process in a local fashion) and dense for paralimbic networks (which therefore appear to process in a more global fashion). This evidence shows that the most essential "association" cortex may not be that on the lateral convexity of the hemisphere, as traditionally thought. Rather, the greatest integration of sensory, motor, and evaluative information may occur in the primitive paralimbic cortex. In the cognitive domain, a central integrating role for paralimbic networks would be consistent with Brown's[37] observation that semantic language disorders involve damage to limbic cortex.

Thus the connectivity to limbic regions may help explain the cognitive as well as the motivational functions of the frontal lobe. Derryberry and Tucker[41] proposed that the representation of interoceptive information within paralimbic networks provides a reference for evaluating and motivating cognitive representations formed at this holistic level, before they are articulated as realized actions or fully conscious ideas. The limbic base for cognition may not be limited to thought and behavior that is obviously emotional. Tucker and Derryberry[42] theorized that the motivational substrate from limbic networks may be essential for guiding the higher executive functions of the frontal lobe. Thus anxiety, emerging from ventral limbic structures and becoming elaborated within orbital frontal cortex, may be an integral component of the focused attention and anticipation required for effective planning. Although pathologically high anxiety may disrupt frontal lobe function,[43] inadequate anxiety may contribute to the self-control deficits and personality disorder in the pseudopsychopathic syndrome that results from orbital frontal lesions.[44,45]

Although our purpose in this section has been to consider the vertical integration across limbic and neocortical networks in a general sense, the example of anxiety and the role of anticipation and planning pertains to a specific anatomical subdivision, the orbital frontal cortex emanating from the paleocortical limbic networks. The evolutionary parcellation of the cortex shown by the anatomical studies of Pandya and associates has given an important new perspective on the functional anatomy of the frontal lobe.[27] In the following section, we review the anatomy of the archicortical and paleocortical pathways, the interpretation of

differing forms of motor control in each pathway by Goldberg,[46] and we propose that there are unique motivational biases for each pathway that are consistent with the different modes of motor control.

DORSAL AND VENTRAL CORTICOLIMBIC PATHWAYS

If the executive functions of the frontal lobe involve working memory,[27,30,47–52] these functions must be bound by the motivational constraints of limbic networks on one end of the processing stream and by the requirements of motor articulation on the other. The theoretical challenge is to characterize the progressive organization of behavior across frontal networks in a way that captures the integration of diverse motivational constraints, the recruitment of activation and arousal controls integral to the process, the extended working memory made possible by large networks, and the fine differentiation of motor programs that are suitably constrained by the extended representational process. In this section, we consider this process in reverse microgenetic order, beginning with a model of motor articulation, considering the role of working memory in planning action, and then theorizing on the emotional and motivational foundations from which the actions are organized. We argue that the theoretical challenge must be met twice, because different principles may be required to describe the dorsal and ventral limbic-cortical processing streams.

Clinical observations have long suggested that there may be differing motivational disorders resulting from damage to dorsal and ventral areas. Kleist noted in 1931 that patients with damage to the mediodorsal areas of the frontal lobes may show apathy and indifference, whereas patients with damage to orbital areas may show poor inhibition of impulses.[53] A number of recent findings have been consistent with Kleist's observations. A lack of initiative is often seen with bilateral dorsomedial frontal lesions. This condition may be confused with psychiatric depression, and has been called the "pseudodepression" syndrome.[44] An extreme form of apathy may be seen in the syndrome of akinetic mutism, in which the patient does not initiate action or speech even though capable of doing so. Although damage to basal ganglia or rostral brain stem may be required for a chronic form of this condition, it is not uncommon to see this syndrome in the period soon after cingulate and mediodorsal frontal lesions.[54] Thus, consistent with Kleist's formulation, an intact mediodorsal frontal lobe may be required for normal motivational initiative.

In contrast, lesions of the orbital frontal lobe may produce a deficit in controlling motivational impulses. Although the "disinhibition syndrome" has traditionally been related to frontal lesions generally, the classical neurological literature shows disinhibition of impulses, puerility, and euphoria to be associated with damage to the orbital surface specifically.[53] In the "pseudopsychopathic syndrome," damage to the orbital frontal region leads to the inability to maintain normal social constraints on behavior.[7,44] These deficits of inhibition form an interesting counterpoint to the overly restricted behavior of the anxious person, which may be associated with exaggerated activity of the orbital frontal lobe.[42,43]

We propose that understanding the role of the frontal lobe in human social

and emotional behavior may require an appreciation of the differing modes of motivational control applied by the dorsal and ventral pathways that lead from limbic networks to the motor cortex. The anatomical differentiation of these pathways reflects the fact that the neocortex evolved from two points of origin: the dorsal, archicortical limbic cortex connected with the hippocampus and the ventral, paleocortical region associated with olfactory cortex.[55] This evolutionary perspective has provided new insights into the connectional architecture of the frontal lobe.[35]

In this section, we briefly outline the anatomical evidence for dual evolutionary origins of frontal cortex. We then propose that, just as the dorsal and ventral pathways display unique modes of motor control as they culminate in motor cortex,[46] these pathways may stem from unique modes of motivational control as each thought and action emerges from the archicortical and paleocortical substrate. Consistent with the principle of vertical integration, the unique motivational biases of the dorsal and ventral pathways may extend below the limbic networks, engaging differential modes of controlling activation and arousal by the brain-stem neuromodulator projection systems.

Dorsal and Ventral Cortical Moieties

The archicortical trend begins in the medial aspect of each hemisphere and projects to the mediodorsal surface of the frontal lobe.[34,56] The evolution of the archicortical moiety from the hippocampus gave rise to proisocortical areas (cingulate cortex, Brodmann areas 24, 25, and 32) and finally to the isocortical areas (9, 10, 46, and 8 on the dorsal surface). Within motor cortex, area 24 differentiated into premotor cortex,[6] which includes the supplementary motor area (SMA) and primary motor cortex (area 4). As it differentiated from primitive paralimbic cortex, the neocortex for both dorsal and ventral moieties accentuated the supragranular layers. Within the dorsal trend the architectonic differentiation emphasized the pyramidal cells. Within the ventral, paleocortical trend the differentiation emphasized the granular cells.

The paleocortical trend differentiated from the paleocortex on the ventral surface of the frontal lobe, into the proisocortex of the orbital and rostral insular regions, and finally into the isocortex on the ventrolateral surfaces of the prefrontal cortex, including Brodmann areas 10, 12, 46, 14, 8, and 11.[56] Paralleling the evolution of the dorsal trend, the paleocortical trend gave rise to the motor cortex on the ventral surface (area 6), which includes the face, head, and neck representations. Reflecting the shared paleocortical origin, researchers have noted the similarities between the insula and orbital cortex in terms of both architectonics and projections.[25,57]

Projectional and Responsive Modes of Motor Control

Luria and Homskaya[58] proposed that every frontal lesion may be understood as impairing the "psychological control of action" or the "synthesis of directed

movements." Similarly, Pribram[27] proposed that the function of the frontal lobe may be discerned by understanding how internal experiences are translated into motor actions. An instructive theory of how motor control may be effected differentially by the archicortical and paleocortical limbic-cortical pathways was proposed by Goldberg.[46]

Goldberg suggested that the mediodorsal frontal pathway, derived from archicortex, is concerned with projecting actions based on probabilistic models of the future. Within this network, motor behavior is organized according to the organism's internal model of the world that is based on experience in similar contexts.[59] In this pathway, the control of action is achieved through a projectional or "feedforward" mode, in that the motor plan is directed by a preexisting model of the action rather than ongoing feedback about the course of the action in the environmental situation.[46] The entire action sequence is organized and launched as a holistic unit.

The ventrolateral motor system, in contrast, appears to link motor sequences to perceptual objects in a responsive manner.[46] This system must be able to identify objects and their motivational significance, and then a "feedback" guidance of motor action causes the motor plan to be articulated with specific reference to the ongoing perceptual input. The ventral motor plan seems to be more differentiated in time than that in the dorsal stream, in that each segment can be linked to perceptual data about its progress.

Observing the separation of these systems of motor control in the frontal lobe suggests that evolution has encountered the same dilemma faced by artificial intelligence researchers in designing intelligent machines (Hendler, this volume). A deliberate system, one that projects actions in future scenarios, is poorly suited to reacting to unforeseen events. A reactive system, on the other hand, is geared for feedback control, and this architecture may not support control by plans.

Could the dorsal and ventral modes of motor control be dependent on differing motivational biases? Are these biases unmasked by the personality deficits resulting from dorsal versus orbital frontal lesions? If the patient with a dorsomedial lesion is apathetic and lacks behavioral initiative, this may suggest that the projectional mode of action in the dorsal pathway has a characteristic motivational basis—a bias toward initiation of action that results in holistic motor plans being projected into the environmental context. If the patient with ventrolateral lesions is impulsive and inappropriate, this may reflect an unbalanced exaggeration of the impulsiveness of the dorsal stream. If so, the normal contribution of the ventral pathway would be to restrict and monitor motivational impulses, perhaps in a manner analogous to the feedback guidance of the action plan by perceptual data on Goldberg's model of the ventral trend.

Learning Mechanisms and Working Memory

If there are inherent relations between the motivational biases suggested by clinical observations and the modes of motor control in Goldberg's analysis, we should expect to find these biases of motivational control integral to the cognitive operations of dorsal and ventral regions of frontal cortex. In a general sense, goal-

directed behavior must be organized over time, and therefore it must be guided by the working memory capacities of frontal networks. Given the essential role of corticolimbic interaction in memory consolidation,[60] we can assume that frontal connections with limbic networks will be necessary to consolidate the cognitive representations that support extended motor planning. Although there is substantial evidence that limbic networks are integral to memory and that there are unique memory capacities for dorsal and ventral pathways in the frontal lobe, it is an unanswered question how these memory capacities relate specifically to differing methods of motor control.

Several lines of evidence suggest that the archicortical and paleocortical moieties support functionally as well as anatomically differentiated memory circuits. The ventral memory system appears to be dependent upon rhinal sulcus, the mediodorsal thalamus, and the orbital cortex.[32,61] In contrast, the dorsal circuit appears to be centered on the hippocampus, anterior nucleus of the thalamus, and the cingulate lobe.[61] Lesions to both the orbitoventral and cingulate cortices result in memory deficits,[62] and similar memory impairments are observed after lesions to the mediodorsal and anterior thalamic nuclei.[63]

Although the functional differentiation of these two memory circuits remains to be clarified, a strong hypothesis is that they are differentially involved in object and spatial memory. The dorsal memory circuit centered on the hippocampus may be involved in spatial memory.[64] One speculation is that the dorsal pathway is important to contextual memory, which may be analogous to spatial relations.[42] The ventral trend, on the other hand, may be especially involved in object memory and the fine-tuning of the neocortical representation of objects, whether the objects are conceptual or perceptual.[42]

A similar framework for cognition was suggested by Kleist in 1934 in a remarkable anticipation of today's cognitive neuroscience model of dorsal and ventral memory systems.[65] The studies of perceptual memory by Ungerleider and Mishkin[31] have led to the realization that objects, the "what" of perception, are represented in the ventral processing stream, whereas spatial relations, the "where" of perception, are represented in the dorsal processing stream. Kleist proposed that "what" is represented in orbital frontal cortex, whereas the "how" of organizing actions is organized in dorsal regions of frontal cortex.[65]

Although generalizations to complex cognitive processes remain speculative, there is substantial experimental evidence with monkeys to differentiate between object and spatial memory capacities of ventral and dorsal frontal regions. In the monkey, the principal sulcus is the boundary dividing the two cortical trends.[36] It has long been known that lesions to the dorsal areas above the principal sulcus result in poor performance on spatial delay tasks, and that lesions to areas ventral to the principal sulcus result in poor performance on object alternation tasks.[27] Current work with single-cell recording in monkeys has provided support for these observations.[30,49]

Studies by Goldman-Rakic and associates have provided convincing evidence that the dorsal and ventral pathways of the posterior cortex, with their respective archicortical and paleocortical targets, are continuous with the dorsal and ventral pathways of the frontal lobe. Neurons below the principal sulcus are responsive to foveal visual stimulation and to recognition of objects in the perceptual field.

Both of these functions are linked to the posterior ventral pathway of the visual system proceeding from occipital to inferior temporal areas.[51] Neurons above the principal sulcus are responsive to peripheral visual stimulation and to spatial aspects of the perceptual task, consistent with the posterior dorsal visual pathway through parietal lobe to cingulate cortex.[51] The working memory operations of frontal cortex appear to maintain the functional continuity with the dorsal and ventral processing streams of posterior cortex.[30]

Fuster[49,66] proposes that the memory capacities of the frontal lobe provide the primate brain with an extended time frame within which more complex patterns of behavior may be organized. Many human cognitive processes can be said to be motor plans that are rehearsed, and evaluated for their adaptive significance, covertly. The most complex forms of cognition require the capacity to evaluate events after they have occurred and to anticipate action before it is required. These are skills that draw explicitly on the temporal span of experience that Fuster describes.

If the dorsal and ventral pathways represent integrated networks for higher cognitive functions, we might expect there would be general principles that could relate the specialized forms of spatial and object working memory to the projectional and reactive modes of motor control, respectively. Do peripheral vision and spatial memory provide a holistic context to support the ballistic, projectional mode of action in the dorsal pathway? Do foveal vision and object identification provide a parsing of the sensory stream in a way that supports a differentiated feedback-monitoring of sequential actions?

If there are coherent systems of working memory that are integral to dorsal and ventral motor control pathways, these may provide clues to the initiative versus inhibitory motivational biases suggested for the dorsal and ventral pathways by clinical neurology. Substantial evidence indicates that the memory operations of the limbic circuitry are closely linked with motivational mechanisms. Clues to the unique adaptive controls inherent to the archicortical and paleocortical substrates of the neocortex may be present in this evidence.

Motivational Bias of the Ventral Pathway

The ventrolateral motor system, with its limbic cortical base in the orbital frontal lobe, may derive its affective influences through extensive connections with the amygdala, insular, and temporal pole cortices.[25,57] The temporal and insula regions provide the ventrolateral system with data from the auditory, visual, and somesthetic modalities for evaluation. In addition, the interconnections of the ventral trend with the insula may be important for linking visceroautonomic associations to perceptual events and to the organization of action plans.[25,39] Based upon their review of the literature, Buchanan and Powell[67] emphasized the importance of sympathetic autonomic responses to ventral limbic cortex. By integrating sensory information with autonomic responses, the ventral limbic complex is well suited to evaluate stimuli for their motivational significance in relation to internal states. Given the evidence linking the ventral trend to the flight/fight response,[42] sympathetic regulation may be particularly important for dealing with threat.

The amygdala appears important to integrating the sensory data that lead to fear responses in rats.[68] In primates, Pribram and his associates have observed effects of amygdala lesions that may suggest ways that memory consolidation in the ventral trend is associated with a specific motivational bias. This bias may be consistent with a role of the ventral limbic networks in anxiety[42] and in the inhibition of impulses suggested by classical and by more recent[69] clinical observations.

Pribram[70] suggests that the amygdala integrates visceroautonomic information with ongoing perception in a process of memory consolidation—familiarization—that marks an episode in time. This parsing of an episode from the flow of experience may be relevant for the learning deficits of amygdalectomized monkeys. In addition, the behavioral abnormalities of these monkeys are consistent with the classical Kluver-Bucy syndrome:[71] they show inappropriate approach behavior to previously feared objects, and they appear hypersexual and hyperoral. These examples of disinhibited behavior may be consistent with the loss of the normal inhibition of hedonic impulses that would stem from the anxiety and threat-monitoring operations of the ventral trend. This tight, inhibitory control in the affective domain may represent the motivational counterpart to the reactive, feedback mode of control of the ventrolateral system in the motor domain.[46]

Motivational Bias of the Dorsal Pathway

In considering the control of learning by the hippocampus, Pribram[70] has suggested that it may support a representation of the context in which behavior occurs. The mechanism for doing this is an interesting one. Several findings suggest the hippocampus may code information about nonreinforced stimuli. This form of discrimination may be important to the extinction of ineffective attention and behavior, and it may be important in relegating nonreinforced stimuli to the background or context of the current behavior.

This framing of the context may be related to the emergence of spatial attention and memory skills of the archicortical pathway[31,72] and perhaps to the notion that the dorsal cortical regions represent contextual information in more general semantic cognition in humans.[42] There is also the suggestion that the learning and memory mechanisms of the hippocampus are associated with a particular motivational bias. Monkeys with hippocampectomy become more conservative and take fewer risks in task performance.[73] This appears to be an opposite bias to the fearless impulsivity of the amygdalectomized monkeys.[70] These differential effects of amygdala versus hippocampal lesions may provide clues to the limbic substrate of the apathy and loss of initiative with dorsal frontal lesions versus the disinhibition of impulses observed with ventral frontal lesions.[5,44,53,74]

Some researchers have argued that the motivational and emotional processes of the limbic circuitry are centered on the amygdala and that the hippocampus and associated dorsal limbic cortex are more relevant to cognition than emotion.[75] However, this view ignores the substantial evidence of the importance of the cingulate cortex to emotion in both animals and humans.[76] The cingulate cortex runs along the superior surface of the corpus callosum and is separated from it by

the callosal sulcus. Recent studies suggest the cingulate is a highly heterogeneous structure. In addition to being divided in the rostral/caudal dimension, it is also differentiated in the dorsal/ventral dimension.[77] The anterior cingulate (areas 24, 25, and 32) can be differentiated from the posterior cingulate based upon cytoarchitecture and patterns of projections, as well as function.[78] Most notably, the anterior cingulate receives afferents from the amygdala, whereas the posterior cingulate does not.[79] The posterior cingulate does not have direct projections to the premotor areas of the frontal lobe, whereas the anterior cingulate does.[80] Consistent with a general rostral/caudal motor/sensory distinction, the anterior cingulate is characterized as "executive" in function, whereas the posterior is characterized as "evaluative."[78]

One way of interpreting the apathy and loss of initiative resulting from dorsomedial frontal lesions would be to attribute these effects to impairment of the dorsal limbic contribution to integrating hedonic value with potential action plans. Patients with cingulate lesions are found to lose interest in formerly important activities, such as hobbies.[81] This evidence is consistent with the view that the cingulate cortex contributes to attention by monitoring the motivational significance of stimuli.[82]

MacLean has emphasized that the dorsal limbic structures have become enlarged in mammalian evolution in parallel with the appearance of complex social and emotional behavior, including care for the young, emotional vocalization, and play.[76,83] Research examining this hypothesis for emotional vocalization has supported the importance of cingulate cortex. Ploog and associates[54,84] have used a combination of lesion and stimulation studies to show the control hierarchy for emotional vocalization in the monkey, with fragmentary motor features represented in brain-stem motor nuclei, patterned species-specific calls represented in the midbrain, emotional coloration of calls deriving from limbic influences, and voluntary call initiation being controlled by cingulate cortex.[54,84] In contrast, lateral motor cortex appears to control "voluntary call formation" through articulated actions mediated by direct pyramidal pathways from motor cortex to brain-stem motor nuclei.

The motivated initiation of holistic patterns of vocalization by the cingulate region bears interesting similarities to both the motivational initiative[44] and the projectional mode of motor control[46] ascribed to dorsomedial frontal cortex. In humans, there are suggestions that the cingulate region may be important in attaching motivational significance, and self-relevance, to the organization of actions in the dorsal limbic-frontal pathway. The decrease in agitation following cingulate lesions for chronic anxiety or for intractable pain[4] may be interpreted as a loss of caring about the condition. The fact that patients lose interest in formerly valued activities[81] suggests that the cingulate contribution to motivational significance is not limited to aversive initiation of action, but that it may involve hedonic value as well. The incorporation of motivational significance, visceral tone, and kinesthetic sensation within the organization of an action may be integral to perceiving the action as part of the self. Goldberg[46] describes the "alien hand" syndrome resulting from dorsomedial frontal lesions, in which an action of the hand contralateral to the lesion, apparently arising within the ventrolateral motor system, is perceived as belonging to someone else.

Redundancy and Habituation Biases as Adaptive Attentional Modes

In theorizing about the motivational basis of the orbital frontal lobe's contribution to the executive functions, Tucker and Derryberry[42] proposed that anxiety may be the affective characteristic of the preparation for fight/flight within the extended amygdala and ventral limbic-frontal pathway. This interpretation would be consistent with the decreases in anxiety with psychosurgery of the orbital region[4] and with the increases in blood flow in ventral frontal cortex seen in clinical anxiety states.[43] This view would not be consistent with Jeffrey Gray's view that anxiety is regulated by the hippocampus. Gray, however, emphasized behavioral inhibition as the key feature of anxiety, whereas Tucker and Derryberry emphasized vigilance and attentional focusing. Although anxiety is often considered to be a pathological state, it may have an integral role in optimal brain function, focusing attention on adaptively important objects.

Tucker and Derryberry[42] argued that the redundancy bias of the dopaminergic tonic activation system[17] may mediate the attentional focusing associated with anxiety. This elementary mode of controlling working memory may have both primitive and sophisticated influences on behavior. In the primitive form, a redundancy bias would facilitate routinized actions, such as in habit formation or in the stereotyped motor sequences of fight/flight responses. In the more sophisticated form, the redundancy bias may focus the representation of plans in working memory on motivationally significant issues, allowing an extended representation that supports the continuity of goal-directed behavior over time. Tucker and Derryberry suggest that the focused attention of the dopaminergic redundancy bias may be especially important to the analytic cognition of the left hemisphere.[42]

In both primitive and sophisticated forms, the redundancy bias may be integral to the feedback modulation of discrete motor sequences in the ventrolateral motor system. The pathological symptoms of exaggerated redundancy in working memory, such as the ruminations and compulsions of the chronically anxious person, may represent the distortion of a neurocybernetic mode that is essential to the normal maintenance of motivated attention.[42]

We speculate that the dorsal motor system may also be regulated by a qualitatively-specific activity control system, the habituation bias of the noradrenergic phasic arousal system. The noradrenergic projections from the brain stem densely innervate cingulate cortex, ascend to the frontal pole, then proceed caudally to innervate the dorsal regions of the neocortex preferentially.[21] Tucker and Williamson[17] theorized that, by decrementing attention to constant features of the environment, a habituation bias would create the positive control of a selection for novelty. They proposed that this novelty selection is integral to the orienting response and that in humans the resulting expansive allocation of working memory is important to the holistic spatial cognitive skills of the right hemisphere. Whereas right hemisphere specialization is an integral aspect of spatial attention in humans, this must be an elaboration of the more fundamental organization of spatial attention and memory within the primate dorsal corticolimbic pathway.[85]

At the limbic root of the archicortical pathway, the hippocampus may regulate learning and memory through mechanisms that are consistent with a habituation bias. The extinction of activity in relation to nonreinforced stimuli theorized by Pribram[70] may be the key limbic mechanism of the habituation bias. The representation of the context for behavioral activity created by this mechanism may be

consistent with the holistic attentional mode attributed to the noradrenergic phasic arousal system by Tucker and Williamson.[17] A critical link in this theorizing, unknown to us at this time, would be between the hippocampus and its associated cortices and the brain-stem noradrenergic and serotonergic projection systems theorized to mediate the habituation bias.

Connections between the parietal lobe (dorsal pathway of the posterior brain) and the brain-stem noradrenergic and serotonergic nuclei have been proposed by Mesulam to be integral to the neglect syndrome.[82] In this disorder, the patient fails to orient to stimuli contralateral to the lesion.[19] Heilman and associates have pointed out that frontal lesions may also produce neglect, and they proposed that the frontal cortex regulates brain-stem reticular and thalamic arousal mechanisms.[86] In rats, right but not left frontal lesions deplete norepinephrine in the locus coeruleus and cortex bilaterally.[87] In humans, the fact that the neglect syndrome is more severe with right-hemisphere lesions suggests that the right hemisphere is particularly important to the higher-order elaboration of the orienting response in attention and working memory.

The neural mechanisms of phasic arousal and the habituation bias appear to have inherent affective qualities, reflecting the depression of mood at low levels of function and mania at high levels.[17] A critical role of the phasic arousal system in the dorsomedial frontal lobe may be relevant to the pseudodepression seen with dorsomedial frontal lesions.[44,88] In some patients, treatment of depression with tricyclics reverses both spatial memory deficits and left neglect.[89,90] In normal subjects, a depressed mood produces a mild attentional neglect that is lateralized to the left visual field.[91] The psychomotor retardation of severe psychiatric depression may be seen as a form of motor initiative deficit that is not unlike the akinetic mutism seen with dorsomedial frontal lesions.

If this line of reasoning is correct, a specific motivational bias may be integral to the cognition and motor organization in the dorsomedial frontal lobe. Closely linked to the individual's mood state, the projectional motor system would be highly charged by motivational directives in manic or euphoric mood states, leading to an impulsive mode of behavior. Although the pathological extreme is instructive, the motivational control of the habituation bias may be integral to the optimal function of the dorsal frontal lobe as well, leading to a bias toward initiating hedonically charged thoughts and actions in the mild elation associated with successful coping, and a specific attenuation of cognitive and behavioral hedonic initiative under conditions of failure.

HEMISPHERIC SPECIALIZATION FOR EMOTION

In addition to the inherent asymmetries of arousal, attention, and memory systems, it has become apparent over the last two decades that important aspects of emotional experience and behavior are asymmetrically distributed in the human brain.[92] The majority of the evidence pertains to emotional communication, the understanding and expression of emotion that is accomplished largely through nonverbal means. In right-handers, the right hemisphere plays the major role for both the comprehension and the expression of emotion. Understanding facial expressions of emotion, for example, is particularly impaired by right-hemisphere

damage.[92] Normal subjects show greater expressivity of emotion on the left side of the face,[93] and they show greater attention to the speaker's emotional tone of voice when passages are presented to the left ear.[94]

This evidence of the importance of the right hemisphere to emotion may be consistent with certain clinical observations suggesting that right frontal lobe lesions are particularly likely to produce the personality disinhibition of the frontal lobe syndrome.[10] However, other evidence has suggested that the left hemisphere also plays an important role in emotional experience and behavior, and a number of recent findings point to the importance of left-frontal lobe function in particular. For both right and left frontal lobes, a key question is how the frontal cortex relates to the emotional processes mediated by subcortical circuits.

Lateralization of Positive and Negative Emotions

Altered emotional and personality processes with right-hemisphere lesions had been recognized since Babinski's observation that anosognosia, denial of illness, was most common with left hemiplegia. However, the "depressive-catastrophic response" to stroke or other brain damage was recognized by Goldstein[95] to occur more frequently with left-hemisphere lesions. The obvious interpretation of this association was that the loss of language is more devastating than loss of nonverbal intelligence. However, a number of studies have failed to correlate the degree of depressive response with the degree of language or cognitive impairment,[96] suggesting that a more fundamental relation may exist between hemispheric specialization and the balance between positive and negative emotional orientations.

An important milestone in this literature was Gainotti's confirmation that catastrophic responses are more likely with left-hemisphere lesions, whereas indifference denial of problems may be more likely with right-hemisphere lesions.[97] Assuming that these findings provide an insight into human emotional balance, the interpretive question became whether damage to a hemisphere results in a release of the contralateral hemisphere's normal emotional orientation,[98] or a release of the damaged hemisphere's subcortical circuits.[99]

In support of the contralateral release interpretation, Sackeim *et al.*[98] reviewed several forms of evidence from the neurological literature. A strong association between the laterality of the lesion and emotional valence was found for cases of pathological laughing (more common with right-hemisphere lesions) and crying (more common with left-hemisphere lesions). The classical interpretation of such cases of "pseudobulbar palsy" is a release of brain-stem emotional mechanisms.[100–102] Rinn points out that whereas corticobulbar projections to brain-stem motor nuclei are contralateral, the pathways disrupted in pseudobulbar palsy involve the reticular formation and, therefore, are bilateral.

In their review, Sackeim *et al.*[98] also observed that outbursts of laughter were frequently associated with left-hemisphere seizures. Reasoning that seizures represent an exaggeration of hemispheric function, Sackeim *et al.* concluded that this evidence implicates a positive emotional bias for the left hemisphere. This conclusion would fit with a contralateral release view of the effects of lesions, assuming that the left hemisphere normally tends toward positive emotion and

the right hemisphere toward negative emotion. Particularly important to this issue was the evidence on chronic changes in emotional outlook in temporal lobe epilepsy.[103] In this research, patients with left-hemisphere pathology showed a negative, critical orientation in self-report measures, whereas those with right-hemisphere pathology showed an inappropriately positive approach to self-evaluation. A form of ipsilateral release was suggested by the cognitive styles of these patient groups. The patients with a left focus were highly intellectualized as well as self-critical, suggesting exaggerated if degraded left-hemisphere cognitive function. The patients with a right focus were emotionally expressive as well as inappropriately positive, suggesting exaggerated right-hemisphere function.

Asymmetric Frontal Lobe Contributions

Although temporal lobe mechanisms are obviously critical to this controversy, the frontal lobe has been found to have an integral role as well. Some of the initial evidence came from EEG studies with normal emotion, which have found asymmetries in frontal lobe alpha activity in a number of paradigms. The initial report[104] observed greater EEG activation (alpha suppression) over the right frontal lobe in response to negative emotional material, in contrast to EEG activation over the left frontal lobe in response to positive emotional material. Independently, Tucker *et al.* found a consistent pattern of results: normal subjects in an induced depressed mood showed alpha suppression (EEG activation) over the right frontal lobe.[105]

Although the findings of frontal lobe EEG asymmetry were consistent in these initial studies, the functional interpretations were not. In line with Sackeim *et al.*'s reasoning, Davidson and associates proposed that the left hemisphere contributes to positive emotion and to approach behavior generally,[106] whereas the right hemisphere is responsible for negative emotion and behavioral withdrawal. In contrast, Tucker *et al.*[105] interpreted their frontal EEG results in line with the ipsilateral release interpretation of the neurological evidence, proposing that the frontal activity may be inhibitory in nature. In their mood induction study, Tucker *et al.* had observed that the depressed mood was associated not only with right frontal EEG activation, but impaired visuospatial performance suggestive of decreased right-hemisphere cognitive functioning.

The evidence of poor visuospatial perception in depression is now quite substantial, and a number of findings implicate impaired right-hemisphere function specifically.[91] In addition to replicating and extending their findings of frontal-lobe alpha asymmetries in a number of experiments, Davidson and associates have also observed poor right-hemisphere cognitive function in depression.[107] The interpretation that the role of the frontal lobe could be inhibitory is consistent with the many findings of disinhibition with frontal lesions. Knight and associates,[108] for example, observed increased auditory ERP responses over the ipsilateral hemi-

sphere in frontal-lesioned patients, suggesting the normal attentional control of frontal cortex may be inhibitory.

An important recent addition to this line of evidence has been the finding that, in normal subjects, left-frontal alpha suppression is related less to the subject's current emotional state than it is to the tendency to deny negative characteristics.[109] In this research it was repressors, subjects who present themselves in a favorable light, who showed the greatest left-frontal alpha suppression in the normal sample of university students. This finding, coupled with an inverse relation between left-frontal blood flow and bilateral amygdala activity in depressed patients, has led Davidson[110] to propose that an important role of left-frontal activity may be the inhibition of negative affect.

Frontal lobe function has been found to be critical to the interpretation of hemispheric contributions to emotion in lesioned patients as well as in normal subjects. In studies of acute depression in stroke patients, Robinson and associates confirmed the previous reports of greater depression with left-hemisphere lesions, and they found a striking trend for greater depression with lesions of more anterior regions of the left hemisphere.[90] Tucker and Frederick[111] interpreted these observations in line with an inhibitory role for the left-frontal region, specifically inhibiting the left-hemisphere limbic and subcortical contributions to anxiety and negative affect. However, at least for striatal contributions to emotional responsivity, this reasoning does not seem to hold. Starkstein *et al.*[112] examined emotional responses in patients with lesions of the caudate nucleus as well as frontal cortex; depression was common in patients with left-caudate lesions as well as left frontal cortex.

If the left frontal lobe is important in inhibiting negative affect, the subcortical structures that are inhibited may be the limbic structures of the left hemisphere. Recent PET blood flow findings have suggested that increased functioning of the amygdala of the left hemisphere may accompany negative affect in both normal subjects and depressed patients. Coupled with previous reports of increased activity of left frontal cortex in negative emotion, these findings raise interesting questions about hemispheric frontal-limbic interactions in emotional self-regulation.

A number of studies have observed increased blood flow and metabolism of regions of left frontal cortex in negative affect. With the Xenon surface rCBF (regional cerebral blood flow) method, Johanson and associates examined anxiety disorder patients as they considered the source of their anxiety. Increased blood flow was observed in inferior regions of the left hemisphere.[113] Using positron emission tomography (PET) measures of rCBF with normal volunteers, Pardo *et al.*[114] found increased orbitofrontal-blood flow bilaterally for women in their sample, but only on the left for the men.

An important question for mood induction research is the uncontrolled cognition, such as self-verbalization, that may be induced by the instructions in addition to the affect. Extending their study of anxious patients with a high-resolution rCBF scanner, Johanson *et al.* also included a control condition of a neutral mood induction that served to equate the possible demands for self-verbalization.[43] They again found high flow over inferior (orbital) left-frontal areas in anxiety. Given the chronic high anxiety of most obsessive-compulsive patients, a consistent find-

ing may be that of Baxter *et al.*, who found increased metabolism in both caudate nuclei and the left orbital frontal cortex in a PET study of obsessive-compulsives.[115]

The improved anatomical precision of PET rCBF, particularly with registration with magnetic resonance anatomical images, has allowed estimates of activity in the amygdala. Examining unipolar depressed patients, Drevets and associates found increased blood flow in the left amygdala and in left frontal cortex.[116] Converging findings with normal emotion have come from the PET rCBF study of Schneider, Gur and associates,[117] who used viewing of emotional faces as a mood induction procedure. In the negative-emotion condition of this experiment, the subjects showed increased flow of the left amygdala. In the positive-emotion condition, they showed increased blood flow in the right amygdala. Interestingly, in a manner similar to that described by Davidson,[110] several measures of frontal cortical flow were found to be inversely correlated with amygdala blood flow.

These several findings may be consistent with the unilateral release interpretation of the effect of hemispheric lesions on emotional orientation.[99] The inherent negative affect (anxiety and hostility) of the left hemisphere may be seen to be modulated by cortical control in the normal brain, such that the depressive catastrophic response to left frontal damage reflects a release of the ipsilateral limbic emotionality. Similarly, the inherently positive emotional tone of right limbic regions may be normally modulated by the right frontal lobe, such that right frontal damage leads to personality disinhibition and denial of problems. Within this framework, the exaggerated corticolimbic interconnection in temporal lobe epilepsy[103] reflects the inherent relations between a self-critical emotional tone and the intellectual ideation of the left hemisphere, compared to the optimistic, self-aggrandizing emotional tone and emotional expressivity of the right hemisphere. These inherent relations between hemispheric cognitive styles and hemispheric affective styles are important clues to the structure of both normal personality and the personality disorders that include avoidant, schizoid, and anxious personalities on the one hand, and histrionic, antisocial, and narcissistic personalities on the other.[118]

The implications of this line of reasoning for frontal lobe function in emotional self-regulation are interesting and somewhat complex. The frontal lobe has been described as inhibitory for hemispheric emotionality;[105] this would be consistent with a release (disinhibition) of emotional behavior following a frontal lesion. Yet, at least for orbitofrontal regions, the rCBF findings suggest that the emotional state is associated with increased frontal activity. The interesting question is whether that increased activity during the emotional state reflects frontal inhibitory modulation of the limbic emotional response, or whether it reflects an elaboration of the emotional process itself.

ASYMMETRIES OF CORTICOLIMBIC ARCHITECTURE

Very likely, the frontal lobe contribution to emotional experience and behavior involves both excitatory and inhibitory influences from both left and right frontal regions. As neuroimaging methods provide increasingly detailed views of human

view is not necessarily wrong, it is incomplete. The higher "association" areas of posterior and frontal cortex represent intermediate networks between sensory and motor isocortex and the densely interconnected paralimbic networks.[34] For the posterior brain, memory consolidation seems to involve recruitment and organization of the processing in neocortical networks under motivational control from paralimbic networks. For the frontal lobes, the process is reversed, with the organization of action emerging from paralimbic cortices—where the representation is inextricably bound with its motivational significance—and then progressively articulated into discrete actions in the multilevel network recursion culminating in motor cortex.[37]

This general limbic-frontal progression in behavioral organization appears to take different forms in the dorsal and ventral corticolimbic pathways. For the dorsal pathway, the "projectional" or feedforward mode of motor control appears to be based on a motivational mode that readily spawns behavioral impulses. The exaggerated case may be the hypomanic or sociopathic personality whose actions are readily generated by hedonic impulses with inadequate feedback from critical self-monitoring. For the ventral pathway, the tight sensorimotor links suggest a high degree of monitoring feedback in the generation of each action from the initial global paralimbic representation. Dominated by this control mode, the chronically anxious or obsessive-compulsive personality may show highly constrained, articulated actions that are seldom tainted by the hedonic impulse. These exaggerated personality styles may be understood in terms of exaggerated neurophysiological mechanisms, providing clues to the motivational biases that must be integrated to balance effective frontal lobe contributions to social and emotional self-regulation.

SUMMARY

In humans, frontal lesions result in deficits of social and emotional behavior that are often surprising in the presence of intact language and other cognitive skills. The connections between the motivation and memory functions of limbic cortex and the motor planning functions of frontal neocortex must be fundamental to meeting the daily challenges of self-regulation. The connectional architecture of limbic and neocortical networks suggests a model of function. The densely interconnected paralimbic cortices may serve to maintain a global motivational context within which specific actions are articulated and sequenced within frontal neocortical networks. The paralimbic networks represent the visceral and kinesthetic information that is integral to the representation of the bodily self. In a general sense, the implicit self-representation within paralimbic networks may shape the significance of perceptions and the motivational context for developing actions. The network architecture of the frontal lobe reflects the dual limbic origins of frontal cortex, in the dorsal archicortical and ventral paleocortical structures. In this paper, we speculated that these two limbic-cortical pathways apply different motivational biases to direct the frontal lobe representation of working memory. The dorsal limbic mechanisms projecting through the cingulate gyrus may be influenced by hedonic evaluations, social attachments, and they may initiate a mode of motor control that is holistic and impulsive. In contrast, the ventral limbic

pathway from the amygdala to orbital frontal cortex may implement a tight, restricted mode of motor control that reflects adaptive constraints of self-preservation. In the human brain, hemispheric specialization appears to have led to asymmetric elaborations of the dorsal and ventral pathways. Understanding the inherent asymmetries of corticolimbic architecture may be important in interpreting the increasing evidence that the left and right frontal lobes contribute differently to normal and pathological forms of self-regulation.

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