The scientific study of emotion, begun by Charles Darwin, Sigmund Freud, and William James in the last century, has been neglected for much of this one. Over-shadowed by behavior and cognition, the formal study of internal emotional states, events that are central to the understanding of the human condition, is only recently receiving the attention of a growing number of experimental researchers and clinical authors. Within the entire spectrum of emotional states, perhaps the last to become a focus of observation of scientific scrutiny has been the “hidden” emotion of shame. A rapid expansion of knowledge about this emotion has occurred only within the last ten years, as contributions from a variety of disciplines are now actively attempting to understand more deeply the unique psychological and biological properties of shame.

A primary purpose of this chapter is to place shame within the context of very recent developmental psychological studies that describe the emergence of emotional states over the course of infancy, as well as of developmental neurobiological research which suggests that these early emotional experiences are required for maturation of the brain in the first two years of life. In pioneering work, H. B. Lewis (1980) referred to shame as “attachment emotion,” a theme I will elaborate on by offering a developmental model of shame that integrates current attachment research. In light of the long-established principle that attachment is more than overt behavior and is “built into the nervous system, in the course and as a result of the infant's experience of his transactions with the mother” (Ainsworth, 1967, p. 429) and the recently demonstrated finding that parenting directly influences the developing patterns of neuronal connectivity in the infant’s brain (Dawson, 1994), I shall present multidisciplinary evidence which suggests that primordial shame experiences play a central role in not only psychological but in neurobiological human development.
To this end I will focus on emotional development in early and then late infancy to demonstrate the tight coupling between the psychobiological processes that underlie attachment and shame dynamics. Attachment transactions in the first year occur within attuned face-to-face interactions that generate increasingly higher levels of positive affects, whereas socialization transactions in the second year involve misattuned face-to-face interactions that generate shame and inhibit these same positive states. I will also outline how, at the end of the first year, dyadic nonverbal communications of internal, positive affective states directly influence the experience-dependent development of a corticolimbic structure involved in attachment functions that begins a critical period of growth at this time. Socialization experiences in the second year that generate stressful levels of shame allow for the further maturation of this frontal system, which is expanded in the early maturing right brain. The right hemisphere plays a dominant role in the processing of socioemotional information, autonomic activities, and the expression and regulation of primitive emotions, including shame. In the final section, I will discuss the adaptive psychobiological role of cortically regulated shame states.

The developmental models presented are not fixed statements or established principles but heuristic proposals that can be evaluated by experimental and clinical research. A more complete referencing of the interdisciplinary studies presented here can be found in Schore (1991, 1994, 1997a,b,c). For conveniences, the terms primary caregiver and mother are used interchangeably throughout this chapter.

Emotional Development in Early Infancy

The last decade has seen a rapid, indeed an explosive, expansion of knowledge about emotional development in the first year of human life. Over the course of this year, the primary caregiver-infant relationship co-constructs an increasingly complex dynamic system of mutual reciprocal influences that mediates the formation of an attachment bond within the dyad. This interactive mechanism regulates the infant's psychobiological states, thereby allowing the child to tolerate more intense and longer lasting levels of heightened, yet modulated, arousal. This ontogenetic achievement, central to human development, enables the infant to experience very high levels of the positive affects of interest-excitement and enjoyment-joy by the end of the first year.

Dyadic Visuoaffective Transactions and the Development of Attachment Dynamics

Early socioemotional development is closely tied into the maturation of sensory systems, and it now appears that, in particular, visual experiences play a paramount role in social and emotional development (Hobson 1993; Preisler, 1995; Wright, 1991). In fact, the mother's emotionally expressive face is, by far, the most potent visual stimulus in the infant's environment, and the child's intense interest in her face, especially in her eyes, leads him or her to track it in space, and to engage in periods of intense mutual gaze. The infant's gaze, in turn, reliably evokes...
the mother's gaze, thereby acting as a potent interpersonal channel for the transmission of reciprocal mutual influences. These face-to-face transactions are quite common and can be of very long duration, and they mediate the dialogue between mother and child.

With the onset of increasing myelination of the visual areas of the infant's occipital cortex over the second and third quarter of the first year, mutual gaze interactions increase significantly. These interactions occur within the context of social play between mother and infant, and they are expressed in synchronous rapid movements and fast changes in affective expressions within the dyad. An organized dialogue occurs within milliseconds, and it acts as an interactive matrix in which both partners match states and then simultaneously adjust their social attention, stimulation, and accelerating arousal in response to the partner's signals. This microregulation continues, as soon after the "heightened affective moment" of an intensely joyful full-gape smile the baby will gaze avert in order to regulate the potentially disorganizing effect of this intensifying emotion (see figure 3.1).

In order to maintain the infant's positive emotion, the psychobiologically attuned mother takes her cue, backs off to reduce her stimulation, and waits for the
baby’s signals for reengagement. In this way, not only the tempo of their engagement but also of their disengagement and reengagement is coordinated. In this process of contingent responsivity, the more the mother tunes her activity level to the infant during periods of social engagement, the more she allows him or her to recover quietly in periods of disengagement; and the more she attends to his or her reinitiating cues for reengagement, the more synchronized their interaction. In this manner, the caregiver facilitates the infant’s information processing by adjusting the mode, amount, variability, and timing of stimulation to the infant’s actual integrative capacities. These early experiences of “interaffectivity” (Stern, 1985) are thus organized by ongoing regulations, and the development of such mutually attuned synchronized interactions is fundamental to the ongoing affective development of the infant (Feldman & Greenbaum, 1997; Schore, 1994).

But facial mirroring exchanges generate much more than overt facial changes in the dyad; they represent a transformation of inner events. To enter into this communication, the mother must be psychobiologically attuned not so much to the child’s overt behavior as to the reflections of his or her internal state. The human face is a unique stimulus whose features display biologically significant information, and as the mother and infant synchronize with each other’s temporal and affective patterns, each recreates an inner psychophysiological state similar to the partner’s. In this dynamic system, the crescendos and decrescendos of the infant’s psychobiological state are in resonance with similar states of crescendos and decrescendos, cross-modally, of the mother. In physics, a property of resonance is sympathetic vibration, the tendency of one resonance system to enlarge and augment through matching the resonance frequency pattern of another resonance system. Stern (1985) refers to the delight the infant displays in reacting to the augmenting effects of his or her mother’s playful, empathically attuned behavior. The caregiver’s attuned resonance with the child’s affect thereby allows for a multimodal sensory amplification of the child’s state. Consequently, both experience a state transition as they move together from a state of neural affect and arousal to one of heightened positive emotion and high arousal (see figure 3.1). It is now thought that the attachment relationship is essentially a regulator of arousal (van der Kolk & Fisler, 1994), that regulatory processes are the precursors of psychological attachment and its associated emotions (Hofer, 1994), and that psychobiological attunement is the mechanism that mediates attachment bond formation (Field, 1985).

According to Bowlby (1969), vision is central to the establishment of a primary attachment to the mother, and imprinting is the learning mechanism that underlies attachment bond formation. Imprinting involves a state of mutually entrained central nervous system (CNS) propensities and a synchrony between sequential infant-maternal stimuli and behavior (Petrovich & Gewirtz, 1985). Emde (1988) suggests that the infant is biologically prepared to engage in visual stimulation to stimulate its brain. This brings us to another level of analysis—the neurobiological level. In this “transfer of affect between mother and infant,” how are developing systems of the organizing brain influenced by these interactions with the social environment?

This question is directly addressed by Trevarthen’s (1993) studies of maternal-infant “protoconversations,” which he describes as an interactive mechanism by which older brains engage with mental states of awareness, emotion, and interest.
in younger brains (figure 3.2). Coordinated with eye-to-eye messages as channels of communication are auditory vocalizations and tactile and body gestures. A traffic of visual and prosodic (variations in pitch, rhythm, and auditory stress that convey affect) signals induce instant emotional effects; excitement and pleasure are amplified within the dyad. Indeed, the resonance of the dyad ultimately permits the intercoordination of positive affective brain states. Trevarthen's studies underscore the fundamental principle that the baby's brain is not only affected by these transactions, but also that its growth literally requires brain-brain interaction and occurs in the context of a positive affective relationship between mother and infant. Trevarthen concludes that "the affective regulations of brain growth" are embedded in the context of an intimate relationship and that they promote the development of cerebral circuits.

The psychoneurobiological mechanism underlying these events is revealed in Hofer's (1990) research, which demonstrates that the mother influences the neural substrates for emotion by directly regulating the levels of the catecholamines dopamine and noradrenaline in the infant's brain. Dopamine is centrally involved in arousal, elation (joy), and the anticipation of reward. Amplified levels of interest-excitement in the mother's face are also accompanied by elevated levels of corti-
cotropin-releasing factor, a neuropeptide produced in paraventricular hypothalamic centers that activates the energy-mobilizing sympathetic division of the autonomic nervous system (ANS) and increases in plasma concentrations of noradrenaline (Brown et al., 1982), thereby intensifying levels of ergotropic (sympathetic-dominant) arousal (Gellhorn, 1970) associated with heightened infant activity levels. The mother's face also triggers high levels of endogenous opiates (endorphins) in the child's growing brain (Hoffman, 1987; Kalin, Shelton, Lynn, 1997) that biochemically mediate the pleasurable qualities of social interaction, social affect, and attachment via activation of the ventral tegmental dopamine system (Bozarth, 1986). Both catecholamines and opioids act as trophic regulators of neural development.

Infant studies thus reveal that the primary function of early synchronized gaze interactions is the generation of pleasurable states and that the baby becomes attached to the modulating caregiver, who expands opportunities for positive affect and minimizes negative affect. In other words, the affective state underlies and motivates attachment, and the central adaptive function of dyadic attachment dynamics is to interactively amplify and maintain optimal levels of the pleasurable states of what Tomkins (1962) referred to as enjoyment-joy and interest-excitement.

The Onset of a Critical Period for the Maturation of the Orbitofrontal Cortex at the End of the First Year

Attachment functions involve highly visual mechanisms and generate positive affect, and they mature near the end of the first year of life. Mary Main, perhaps the most influential attachment researcher, now concludes that "The formation of an attachment to a specified individual signals a quantitative change in infant behavioral (and no doubt also brain) organization" (1995, p. 214; italics added). Gilbert (1989) proposes that the mother-child relationship has important and very specific effects on the maturation of the infant's limbic system. So the question is: What specific limbic areas of the brain are beginning a critical period of growth at 10 to 12 months and are involved in attachment functions?

In earlier work (see Schore, 1994, 1996), I proposed that dyadic communications which generate intense positive affective states and high levels of dopamine and endogenous opiates represent a growth-promoting environment for the prefrontal cortex, an area that undergoes a major maturational change at 10 to 12 months (Diamond & Doar, 1989). The maturation of the prefrontal areas, which represent 30% of the total surface of the human cortex, is completely postnatal, and the limbic orbital prefrontal areas are known to mature before the nonlimbic dorsolateral prefrontal areas (figure 3.3). Attachment experiences, face-to-face transactions between caregiver and infant, directly influence imprinting, the final circuit wiring of this system. Indeed, there is now evidence to show that activity of the orbitofrontal cortex, an area which contains neurons that specifically respond to the emotional expressions of faces (Scalaidhe, Wilson & Goldman-Rakic, 1997), is directly associated with attachment functions (Steklis & Kling, 1985). This cortical region plays an essential role in the processing of social signals necessary for the initiation of affiliative behaviors and in the pleasurable qualities of social interaction.
The orbital area of the prefrontal cortex (so called because of its relation to the orbit of the eye) is “hidden” in the ventral and medial surfaces of the prefrontal lobe. This unique cerebral region is so intimately interconnected limbic areas that it has been conceived of as an “association cortex” for the limbic forebrain. In addition to receiving multimodal input from all sensory association areas of the posterior cortex, it uniquely projects extensive pathways to limbic areas in the temporal pole and the amygdala, to dopamine neurons in reward centers of the ventral tegmental areas of the anterior reticular formation, and to drive centers in the paraventricular hypothalamus that are associated with the sympathetic branch of the autonomic nervous system. This excitatory limbic circuit, the ventral tegmental limbic forebrain-midbrain circuit (Nauta & Domesick, 1982), is involved
with the generation of positively valenced states associated with motivational re-
ward, approach behavior, and active coping strategies.

This area is especially expanded in the right cortex (Falk et al., 1990), which 
matures before the left (Chiron et al., 1997), is known to be specifically affected by 
early social experiences, and is centrally involved in attachment experiences 
(Schore, 1994). Indeed, it is now thought that “The emotional experience of the 
infant develops through the sounds, images, and pictures that constitute much of 
an infant’s early learning experience, and are disproportionately stored or proc-
essed in the ‘visuospatial’ right hemisphere during the formative stages of brain 
ontogeny” (Somrud-Clíkeman & Hynd, 1990, p. 198). As the first year draws to a 
close, the initial phases of the anatomical maturation of the orbitofrontal cortex, a 
system that subserves cognitive-emotional interactions (Barbas, 1995), allows for 
developmental advances. This system can now generate interactive representations 
that encode expectations of being matched by, and being able to match, the 
partner, as well as “participating in the state of the other” (Beebe & Lachmann, 
1988). By the end of the first year, a period when the infant begins to toddle and 
explore the world, the child can access internal working models of the infant’s 
transactions with the primary attachment figure in order to appraise self and other 
in encounters with the social and physical environments.

Emotional Development in Late Infancy

In optimal growth-promoting environments, the interactive mechanism for gen-
erating positive affect becomes so efficient that by the time the infant begins to 
toddle he or she is experiencing very high levels of elation and excitement. The 
socioemotional environment of the caregiver-infant dyad, however, changes dra-
matically from early to late infancy. At 10 months, 90% of maternal behavior con-
sists of affection, play, and caregiving. In sharp contrast, the mother of the 13–17-
month-old toddler expresses a prohibition on the average of every 9 minutes. In 
the second year, the mother’s role now changes from primarily a caregiver to a 
socializing agent, as she must now persuade the child to inhibit unrestricted ex-
ploration, tantrums, bladder and bowel functions; that is, activities that he or she 
enjoys.

Socialization Experiences and the Emergence of 
the Attachment Emotion of Shame

In other words, to socialize the child, the mother must now engage in affect reg-
ulation to reduce the heightened levels of positive affect associated with the plea-
sure of these activities. How does she inhibit and restrict the behaviors that the 
child finds pleasurable? In fact, there is one very specific inhibitor of accelerating 
pleasurable emotional states. Shame, “the primary social emotion” (Scheff, 1988), 
acts as a specific inhibitor of the activated ongoing affects of interest-excitement 
and enjoyment-joy and uniquely reduces self-exposure or exploration powered by 
these positive affects (Tomkins, 1963). In earlier work (Schore, 1991, 1994), I pre-
sented multidisciplinary developmental data which suggests that shame makes its 
initial appearance at 14 to 16 months.
In the second year, the ambulatory infant, now able to physically separate him- or herself from the mother for longer periods of time, is able to explore realms of the physical and social environment that are beyond her watchful eye. Upon return from these forays, the toddler brings the things he or she is exploring and attempting to master to the mother, but now, more than any time previously, these reunions can engender interactive stress. Excitedly expecting her “sparkling-eyed pleasure” (Emde, 1988), the toddler is suddenly and unpreparedly confronted with the “unexpected noncooperation of the mirroring object” (Kohut, 1978). Face-to-face encounters that at one time elicited only joy become the principal context for stressful shame experiences.

The experience of shame is associated with unfulfilled expectations and is triggered by an appraisal of a disturbance in facial recognition, the most salient channel of nonverbal communication. The toddler’s attentive focus on the mother’s eyes and the frustration of the child’s expectation of her participation in eye-to-eye contact and visuoaffective communication may be the key visual cue that triggers the visual, nonverbal affect of shame. Reciprocal gaze behavior, the most intense form of interpersonal communication, acts as a powerful mediator of affect attunement, but it can also transmit misattunement, because “this visual feedback system carries within it the potential of mutual gratification as well as frustration” (Riess, 1978, p. 382). The impediment to anticipated positive affect is specifically a perception of a facial display which conveys not mirroring but, rather, disgust. M. Lewis (1992) points out that a disgusted face is widely used in the socialization of children, though parents are often unaware that they are producing it.

These developmental data describe the rapid state-transforming events that underlie the primordial shame transaction. As a result of earlier dyadic mirroring experiences, when the senior toddler approaches the caregiver in a state of accelerating positive affect, he or she expects this state of rising positive arousal to be amplified. Recall that the child now has access to presymbolic representations that encode the expectation of being matched by, and being able to match, the partner, as well as “participating in the state of the other” (Beebe & Lachmann, 1988). Despite an excited expectation of a psychobiologically attuned shared positive affect state with the mother and a dyadic amplification of the positive affects of excitement and joy, the infant unexpectedly encounters a facially expressed affective misattunement. The ensuing break in an anticipated visual-affective communication triggers a sudden shock-induced deflation of positive affect, and the infant is thus propelled into a state which he or she cannot yet autoregulate. Shame represents this rapid state transition from a preexisting positive state to a negative state.

Psychobiological attunement drives the attachment process by acting as a mechanism that maximizes and expands positive affect and minimizes and diminishes negative affect. The negative affect of shame is thus the infant’s immediate physiological-emotional response to a perceived interruption in the flow of an anticipated maternal regulatory function—psychobiological attunement which generates positive affect—and to the mother’s use of misattunement to mediate the socialization process. In other words, shame, which has been called an “attachment emotion” (Lewis, 1980), is the reaction to an important other’s unexpected refusal to cocreate an attachment bond that allows for the dyadic regulation of emotion (Sroufe, 1996). Thus, in the prototypical object relation of shame, a sep-
aration response is triggered in the presence of and by the mother, who sponta-
neously and unconsciously blockades the child's initial attempt to emotionally
reconnect with her in a positive affective state. It is well established that attach-
ment bond disruptions precipitate an imbalance in the regulation of affect. In the
shame transaction, instead of encountering the synchronizing function of the sig-
nificant other who regulates the child's homeostatic equilibrium, the individual
experiences a traumatic interruption of interpersonal synchronizing processes
(Maunder, 1996). The misattunement in shame, as in other negative affects, rep-
resents a regulatory failure and is phenomenologically experienced as a disconti-
nuity in what Winnicott (1958) calls the child's "going-on-being."

In direct contrast to the psychobiologically energized state, shame, an acutely
painful stress-associated affect, triggers a rapid de-energizing state in the infant in
which the deflated self, depleted of energy, withdraws, recoils, and attempts to
disappear from the view of significant objects. As opposed to processes that pro-
mote and prolong contact and facilitate "merging with sources of satisfaction" in
order to generate euphoric emotions and pleasurable activity, shame induces "end-
ing contact and halting arousal" (Knapp, 1967). The conscious subjective experi-
ence of shame, a sharp gradient of change in emotion, represents a rapid, unex-
pected, uncontrollable transition from a "crescendo" to a "decrescendo," an
"animate" to an "inanimate" feeling state (Stern, 1985), a switch from an attach-
ment-affiliation or exploratory-assertive to an aversive motivational functional sys-
tem. Shame stress thus precipitates a rapid and unexpected contraction of the self.

This state is mediated by a different psychobiological pattern than positive
states—corticosteroids are produced in a stress response, and these reduce opioid
and corticotropin-releasing factor in the brain. Even short-lived elevations of cor-
ticosteroids induce inhibition and withdrawal (Stansbury & Gunnar, 1994). The
psychobiological components of the shame response involve an influx of auto-
nomic proprioceptive and kinesthetic feedback into awareness, reflecting a neu-
robiological activation of medullary reticular formation activity in the brain stem.
In contrast to the attuned state, shame elicits a painful infant distress state, mani-
fested in a sudden decrement in mounting pleasure, a rapid inhibition of excite-
ment, and cardiac deceleration by means of vagal impulses in the medulla oblon-
gata (Knapp, 1967). This shift reflects the reduced activation of the excitatory
ventral tegmental limbic forebrain-midbrain circuit and increased activation of the
inhibitory lateral tegmental limbic forebrain-midbrain circuit.

The onset of the interactively triggered shame state thus represents a sudden
shift from energy-mobilizing sympathetic to energy-conserving parasympathetic
dominant ANS activity, a rapid transition from a hyperaroused to a hypoaroused
state, and a sudden switch from ergotropic (sympathetically driven) to tropho-
tropic (parasympathetically driven) arousal. Indeed, Buss (1979) has demonstrated
that shame represents parasympathetic arousal. In such a psychobiological state
transition, sympathetically powered elation, heightened arousal, and elevated ac-
tivity level instantly evaporate. This represents a shift into a low-keyed inhibitory
state of parasympathetic conservation-withdrawal that occurs in helpless and
hopeless stressful situations (Engel & Schmale, 1972). How long the child remains
in this stress state is an important factor.

In fact, active parental participation in regulating the child's shame state is
critical to enabling the child to shift from the negative affective state of deflation
and distress to a reestablished state of positive affect. In early development, parents provide much of the necessary modulation of states, especially after a state disruption and across a transition between states, and this allows for the development of self-regulation. This transition involves and highlights the central role of stress recovery mechanisms in affect regulation. Stress has been defined as the occurrence of an asynchrony in an interactional sequence; further, “a period of synchrony, following the period of stress, provides a ‘recovery’ period” (Chappie, 1970 p. 631). The child’s facial display, postural collapse, and gaze aversion act as nonverbal signals of his or her internal distress state. If the caregiver is sensitive, responsive, and emotionally approachable, especially if she reinitiates and reenters into mutual gaze visual affect regulating transaction, the dyad is psychobiologically reattuned, shame is metabolized and regulated, and the attachment bond is reestablished. The key to this is the caregiver’s capacity to monitor and regulate her own affect.

In this essential pattern of what Beebe and Lachmann (1994) call “disruption and repair,” the “good-enough” caregiver who induces a stress response in her infant through a misattunement reinvokes in a timely fashion her psychobiologically attuned regulation of the infant’s negative affect state that she has triggered. This reattunement is mediated by the mother’s reengagement in dyadic visuoaffective transactions that regenerate positive affect in the child. Her shame-stress-regulating interventions allow for a state transition in the infant—the parasympathetic-dominant trophotropic arousal of the shame state is supplanted by the reignition of sympathetic-dominant ergotropic arousal that supports increased activity and positive affect. Shame transactions are carried out continually throughout the early period of socialization, and a characteristic pattern of regulating and thereby coping (or not coping) with misattuned states and distressing affects develops within the dyad. These events are stored within an internal working model of a secure attachment.

If, on the other hand, an attachment figure frequently humiliates, ridicules, and rejects the child’s requests for comfort in stressful situations, the child develops not only an internal working model of the parent as rejecting but also one of him-or herself as unworthy of help and comfort. I suggest that, as opposed to the elevated parasympathetic autonomic component which always accompanies shame, humiliation, a common accompaniment of early physical trauma, involves an extremely dysregulated state of elevated parasympathetic plus heightened sympathetic reactivity. Psychophysiologically this may represent, respectively, a state of activated trophotropic arousal versus an intensely stressful state of trophotropic combined with ergotropic arousal, a state of “shame-rage” (H. B. Lewis, 1987). Clinical observers note that failures of early attachment invariably become sources of shame (Kaufman, 1989), that impairments in the parent-child relationship lead to pathology through an enduring disposition to shame (M. Lewis, 1992), and that early abuse engenders intense bodily shame (Andrews, 1995). These data imply that it is not shame itself but rather an early developing inefficient capacity to autoregulate or interactively regulate this potent affect that is psychopathogenic.

Specific emotions are now understood to involve a distinctive “core relational theme” which describes an essential person-environment relationship (Lazarus, 1991) and to be elicited by an appraisal of actual or expected changes that are important to the individual (Frijda, 1988). In light of the fact that shame is directly
related to visual phenomena, the core relational shame transaction becomes internalized in implicit, procedural memory as a visual stored image. Nathanson describes shame as "a biological system by which the organism controls its affective output so that it will not remain interested or content when it may not be safe to do so, or so that it will not remain in affective resonance with an organism that fails to match its patterns stored in memory" (1992, p. 140). Although the origin of shame is dyadic and external, "the experience of being looked at by the Other" (Wright, 1991), it eventually becomes internalized as "the eye of the self gazing inward" (Morrison, 1987). By the end of late infancy, the elicitation of this affect does not require the presence of an external person—it can be activated by an internal image. Morrison (1989) asserts that turning the potential control of shame inward is an important developmental step. Most important, because shame generally inhibits the expression of emotion per se, the capacity to internally regulate shame allows for an ability to experience a broad range of positive and negative affects.

The Maturation of the Orbitofrontal Cortex

These advances in emotional functions reflect structural progressions in the limbic system, specifically a reorganization and maturation of the orbitofrontal cortex, a corticolimbic area that is centrally involved in affect regulation (Schore, 1994, in 1996; 1997a, b; in press). A period of structural development in this cortex occurs in the first year of infancy, and a second period marked by further anatomical changes occurs in the second year of human life. This reorganization of the prefrontal region is “open to interactions with the external world” (Kostovic, 1990). In the second year, such interactions are expressed in dyadic shame and interactive repair transactions that are part of the socialization process. These experiences trigger specific psychobiological patterns of hormones and neurotransmitters, and the resultant biochemical alterations of brain biochemistry influence the experience-dependent maturation of the orbitofrontal cortex.

Specifically, the interactive misattunements generated in socialization transactions induce shame, an interruption of interpersonal synchronizing processes. Developmental psychobiological research indicates that interruptions of the attachment bond are correlated with increased cortisol and decreased endogenous opioid levels (Trad, 1986). The sudden triggering of shame reflects an alteration of the infant’s psychobiological state and the onset of a stress reaction, manifested in elevated levels of corticosteroids in the infant’s brain. Corticosteroids suppress production of corticotropin-releasing factor and thereby reduce endorphin levels. But during critical periods of cortical maturation these neurohormones do more than just transiently perturb states—in fact, they directly influence brain growth (Meyer, 1985). Developmental shame experiences thus induce a neurobiological reorganization of evolving brain circuitries.

In optimal socialization experiences in the second year, the child is exposed not only to interpersonal transactions that induce negatively valenced shame and inhibition and withdrawal behaviors but also to interactive regulations that repair this state and resume positively valanced excitatory activities and approach behaviors. These distinct types of socioemotional experiences allow for the critical-period experience-dependent structural maturation of the orbitofrontal cortex. (In
contrast, misattuned relational environments that generate high and long-enduring levels of negative affect act as growth-inhibiting environments for developing frontal-limbic systems (Schore, 1996). This organization includes the fine-tuning of descending projections from the prefrontal cortex to subcortical structures that are known to mature during infancy. Of particular importance is the growth of prefrontal axons back down to subcortical targets on noradrenergic neurons in the nucleus of the solitary tract of the brain stem caudal reticular formation and the vagal complex in the medulla (Yasui, Itoh, Kaneko, Shigemoto, & Mizuno, 1991) and in parasympathetic autonomic areas of the hypothalamus (Kita & Oomura, 1981). By this process the organization of the lateral tegmental forebrain-midbrain limbic circuit that braces arousal and activates the onset of an inhibitory state is completed. Along with the earlier developing ventral tegmental limbic forebrain-midbrain circuit, the orbitofrontal system now connects into both the excitatory and the inhibitory limbic circuits. Its direct connections with the hypothalamus enable it to act as a major center of central nervous system control over the energy-mobilizing sympathetic and energy-conversing parasympathetic branches of the autonomic nervous system (Neafsey, 1990).

Because of the organization of its dense connections, with sites in both the cortex and subcortex, this corticolimbic system plays an essential adaptive regulatory role. At the orbitofrontal level cortically processed exteroceptive information concerning the external environment (such as visual and prosodic information emanating from an emotional face) is integrated with subcortically processed exteroceptive information regarding the internal visceral environment (such as concurrent changes in the emotional or bodily state). This adaptive function allows for incoming social information to be associated with emotional and motivated states. Orbitofrontal areas are involved in the generation of high-level psychological representations of other individuals (Brothers & Ring, 1992) and in the self-regulation of bodily states (Luria, 1980). According to Hofer (1984), internal representations of external human relationships serve an important intrapsychic role as "biological regulators" that control physiological processes. The essential activity of this ventromedial system in emotional behavior (Damasio, 1994; Price, Carmichael, & Drevets, 1996) and in mediating processes relevant to the organism’s participation in social groups (Zald & Kim, 1996) is thus the adaptive switching of internal bodily states in response to changes in the external social environment that are appraised to be personally meaningful (Schore, in press, c). This includes the onset of a parasympathetically driven inhibitory shame state in response to the appraisal of a facially (visually and prosodically) expressed stressful state of the interpersonal environment.

Over 60 years ago, MacCurdy (1930) proposed that the immobility seen in shame is due to a shift in balance in the autonomic nervous system, with an offset of sympathetic activity and an onset of vagal activity, leading to cardiac deceleration and a fall in blood pressure. The shame state is manifested in a rapid inhibition of excitement, a sudden decrement in mounting pleasure, and cardiac deceleration by means of vagal impulses (Knapp, 1967). Activation of the orbitofrontal cortex, a cortical area that receives direct vagal inputs (Hardstaff, Jagadeesh, & Newman, 1973) and acts as a "nonspecific system governing internal inhibition" (Velasco & Lindsley, 1965), psychobiologically mediates the onset of socially-induced stressful shame states, since direct projections from frontal cor-
ticolimbic areas down to subcortical autonomic sites allows for an orbital role in "vagal restraint" and in the energy-conserving inhibition of autonomic function (Kaada, Pribram, & Epstein, 1949). Stimulation of particular orbitofrontal sites triggers an almost instantaneous inhibition of gastrointestinal motility, respiratory movements of inspiration, and somatic locomotor activity and a dramatic precipitous fall in blood pressure, thereby accounting for the influx of autonomic feedback into awareness that accompanies shame. The involvement of orbitofrontal-vagal connections in shame is suggested by the "active restraining quality" of this affect, which brakes arousal and triggers a "partial paralysis of outer activity" (Knapp, 1967), since orbital activity inhibits muscle tone and brain-stem-regulated somatic reflexes (Sauerland, Knauss, Nakamura, & Clemente, 1967). The central role played by frontolimbic areas in shame states is also expressed in its neurohormonal regulatory operations—orbitofrontal activation triggers increases of hypothalamico-pituitary-adrenocortical corticosteroid levels (Hall & Marr, 1975), a function mediated by the parasympathetic branch of the ANS (Henry & Stephens, 1977).

Indeed, this prefrontolimbic region comes to act in the capacity of an executive control function for the entire right cortex (see figure 3.4). The right hemisphere is particularly well connected reciprocally with limbic (Tucker, 1992) and autonomic (Spence, Shapiro, & Zaidel, 1996) areas, and is therefore dominant in controlling fundamental physiological and endocrinological functions whose primary control centers are located in subcortical regions of the brain (Wittling & Pfluger, 1990). These authors point out that the right cortex is also centrally involved in regulating corticosteroid activity. Furthermore, a right hemispheric vagal circuit of emotion regulation, hierarchically dominated by the right orbitofrontal cortex, has been recently described by Forges, Doussard-Roosevelt, and Maiti (1994). I suggest that the activity of this right brain cortical-subcortical circuit, identical to the arousal-braking, inhibitory lateral tegmental forebrain-midbrain limbic circuit, mediates all forms of shame-related emotional behaviors. Neurobiological studies show that the orbitofrontal system plays a major role in the adjustment or correction of emotional responses (Rolls, 1986; Schore, 1994), that is, affect regulation. It acts as a recovery mechanism that efficiently monitors and autoregulates the duration, frequency, and intensity of not only positive but also negative affect states, including stressful shame states.

The Adaptive Psychobiological Role of Cortically Regulated Shame States

The emergent capacity of experiencing and autoregulating the intensely negative affect of shame thus reflects the experience-dependent maturation of a right frontolimbic system that can access cortically processed social information in order to modulate subcortical autonomic functions that underlie various emotional states. There is now a large body of evidence which indicates that negative emotions activate and are modulated by the right (not left) hemisphere (e.g., Davidson, Ekman, Saron, Senulis, & Friesen, 1990; Heilman & Bowers, 1990; Heller, 1993, Schore, 1997b). This hemisphere, which contains a "primitive affect system" (Gazzaniga, 1985) and a "nonverbal affect lexicon" of facial expressions (Bowers,
Bauer, & Heilman, 1993), is dominant for the regulation of the autonomic correlates of emotional arousal (Heilman, Schwartz, & Watson, 1977). These data strongly suggest that "primitive, biologically based" shame (Broucek, 1982), perhaps the most painful and least tolerable negative affect, is a right-brain phenomenon. Indeed, a neuropsychological study shows that the words "shame" and "humiliation," when presented tachistoscopically, specifically activate and prime the right hemisphere (Van Strien & Morpugo, 1992).

Almost 50 years ago, Erikson (1950) offered the classical conception of shame as a feeling of being visible and exposed to the eyes of an Other, which leads to an urge to hide and cover one's face. The importance of regulating and thereby being able to tolerate shame from toddlerhood through adulthood lies in the adaptive capacity of conservation-withdrawal. Powles characterizes this state, in which the inhibited organism passively disengages in order to attempt to become "unseen," as the organismic strategy "to conserve energies and strive to avoid attention, to foster survival by the risky posture of feigning death, to allow healing of wounds and restitution of depleted resources by immobility" (1992, p. 213). This primary regulatory process for organismic homeostasis allows for a passive coping mechanism that improves survival efficiency through inactive disengagement and unresponsiveness to environmental input in order to conserve resources and to assure organismic autonomy until environmental conditions are once again more com-

Figure 3.4. Relationships of brain stem structures to the orbital surface of the right hemisphere. From Smith (1981). Copyright 1981 by Urban & Schwarzenberg. Reprinted by permission.
compatible (Engel & Schmale, 1972). As opposed to sympathetically driven “fight-flight” active coping strategies, parasympathetically mediated passive coping mechanisms expressed in immobility and withdrawal associated with “giving up” (Benus, Bohus, Koolhaas, & Van Oortmerssen, 1991) and submission (Gilbert, 1992) and in seeking a physical environment of “refuge,” a place to hide without being seen (Mealey & Theis, 1995), represent an alternative but equivalent strategy for effectively regulating social interactional stress. In contrast to “problem focused coping,” which entails direct action on the self or on the environment to remove the source of stress, this “emotion focused coping” is directed toward the reduction of the emotional impact of stress through psychological processes (Folkman & Lazarus, 1980).

The physiological expression of emotion is dependent upon the coordinated responses of both the sympathetic and parasympathetic components of the ANS, and this allows for parasympathetically dominant shame states to combine with sympathetically driven states of, for example, fear, anger, and joy. It is known that parasympathetic inhibitory systems, such as in the frontal lobes, develop more slowly than sympathetic excitatory processes (Thompson, 1990), that emotion expression changes developmentally as a function of the experience-dependent maturation of neural inhibitory mechanisms (Izard, Hembree, & Huebner, 1987), and that the maturation of the frontal region in the second year is responsible for affect regulation and the development of complex emotions such as shame (Fox, 1991). In light of the fact that affects in general serve the critical adaptive function of informing the individual who is tracking biologically relevant goals (Gilbert, 1992) and that specific emotions help to prepare for and sustain the person-environment relationship (Lazarus, 1991), successful emotional adaptation requires the capacity to tolerate both positive and negative affects of formidable intensity. Internal working models are now thought to essentially encode coping strategies of affect regulation which are unconsciously used to regulate distress in situations that normally elicit attachment behaviors. The attachment dynamic continues in adult life, and the ability to cope with the stressful ruptures of attachment that elicit shame is essential to ongoing development, because this affect, which is generated by virtually constant monitoring of the self in relationship to others, comes to play a central role in the regulation of all emotional expression and therefore all human interaction.

References


