The Effects of Early Relational Trauma on Right Brain Development, Affect Regulation, & Infant Mental Health

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Abstract

A primary interest of the field of infant mental health is in the early conditions that place infants at risk for less than optimal development. The fundamental problem of what constitutes normal and abnormal development is now a focus of developmental psychology, infant psychiatry, and developmental neuroscience. In the second part of this sequential work, I present interdisciplinary data to more deeply forge the theoretical links between severe attachment failures, impairments of the early development of the right brainis stress coping systems, and maladaptive infant mental health.

In the following I offer thoughts on the negative impact of traumatic attachments on brain development and infant mental health, the neurobiology of infant trauma, the neuropsychology of a disorganized / disoriented attachment pattern associated with abuse and neglect, trauma-induced impairments of a regulatory system in the orbitofrontal cortex, the links between orbitofrontal dysfunction and a predisposition to posttraumatic stress disorders, the neurobiology of the dissociative defense, the etiology of dissociation and body-mind psychopathology, the effects of early relational trauma on enduring right hemispheric function, and some implications for models of early intervention. These findings suggest direct connections between traumatic attachment, inefficient right brain regulatory functions, and both maladaptive infant and adult mental health.

In the first paper of this serial contribution, I have suggested that an interdisciplinary approach that focuses upon attachment experiences and their effects on regulatory structures and functions can offer us more comprehensive models of normal development. This conception directly evolves from the central tenets of attachment theory. In his groundbreaking volume, Attachment, John Bowlby (1969) argued that developmental processes could best be understood as the product of the interaction of a unique genetic endowment with a particular environment. Integrating then current biology with developmental psychoanalytic concepts, he proposed that the infantis "environment of adaptiveness" has consequences that are "vital to the survival of the species", and that the attachment relationship directly influences the infantis "capacity to cope with stress" by impacting the maturation of a "control system" in the infantis brain that comes to regulate attachment functions. From the very start, Bowlby contended that a deeper understanding of the complexities of normal development could only be reached through an integration of developmental psychology, psychoanalysis, biology, and neuroscience (Schore, 2000a).

Over the course of (and since) the "decade of the brain" the amount of scientific information concerning the unique psychological, psychobiological, and neurobiological phenomena that occur in the early stages of human life has rapidly expanded (Schore, 1996, 1997a, 1998a, b, 1999a, 2000b). With an eye to these data, at the end of the very same decade Mary Main proclaimed:

We are now, or will soon be, in a position to begin mapping the relations between individual differences in early attachment experiences and changes in neurochemistry and brain organization. In addition, investigation of physiological "regulators" associated

with infant-caregiver interactions could have far-reaching implications for both clinical assessment and intervention (1999, pp. 881-882).

This current confluence of attachment theory, psychobiology, and neurobiology, the one that Bowlby predicted, offers us a real possibility of creating more complex interdisciplinary conceptions of attachment and social and emotional development. The field of infant mental health specifically focuses upon social emotional development, and so more detailed psychoneurobiological understandings of attachment can generate a more overarching model of the normal development of the human mind/brain/body at the earliest stage of the lifespan and therefore more precise definitions of adaptive infant mental health.

With that goal in mind, in the preceding article I have argued that in attachment transactions of affective synchrony, the psychobiologically attuned caregiver interactively regulates the infantis positive and negative states, thereby co-constructing a growth facilitating environment for the experience-dependent maturation of a control system in the infantis right brain. The efficent functioning of this coping system is central to the infantis expanding capacity for self regulation, the ability to flexibly regulate stressful emotional states through interactions with other humans - interactive regulation in interconnected contexts, and without other humans - autoregulation in autonomous contexts. The adaptive capacity to shift between these dual regulatory modes, depending upon the social context, is an indicator of normal social emotional development. In this manner a secure attachment relationship facilitates right brain development, promotes efficient affect regulation, and fosters adaptive infant mental health.

But from the beginning, attachment theory has also had a parallel interest in the etiology of abnormal development. In applying the theory to the links between stress coping failures and psychopathology, Bowlby (1978) proposed:

In the fields of etiology and psychopathology [attachment theory] can be used to frame specific hypotheses which relate different family experiences to different forms of psychiatric disorder and also, possibly, to the neurophysiological changes that accompany them.

These germinal ideas have lead to the field of developmental psychopathology, an interdisciplinary approach that conceptualizes normal and aberrant development in terms of common underlying mechanisms (Cicchetti, 1994). This field is also now incorporating current data from neuroscience into more complex models of psychopathogensis.

This is because contemporary neuroscience is now producing more studies of not just the pathology of the mature brain, but the early developmental failures of the brain. And so neurobiology is currently exploring "early beginnings for adult brain pathology" (Altman, 1997) and describing "alteration[s] in the functional organization of the human brain which can be correlated with the absence of early learning experiences" (Castro-Caldas et al., 1998). These data are also relevant to the field of infant mental health, with its interest in all early conditions that place infants and/or their families at risk for less than optimal development.

These trends indicate that an integration of current attachment theory, neuroscience, and infant psychiatry can offer more complex models of psychopathogenesis (Schore, 1994; 1997d; 1998d). Toward that end, in this second part of this sequential work, I will offer interdisciplinary data in order to strenghthen the theoretical connections between attachment failures, impairments of the early development of the brainis stress coping systems, and maladaptive infant mental health. And so I will present ideas on the effects of traumatic attachment experiences on the maturation of brain regulatory systems, the neurobiology of relational trauma, the neuropsychology of a disorganized/disoriented attachment pattern, the inhibitory effects of early trauma on the development of control

systems involved in affect regulation, the links between early relational trauma and a predisposition to postraumatic stress disorder, a neurobiological model of dissociation, the connections between traumatic attachment and enduring right hemisphere dysfunction, and implications for early intervention.

In the course of this work I will use the disorganized/disoriented ("type D") attachment pattern as a model system of maladaptive infant mental health. This attachment category is found predominantly in infants who are abused or neglected (Carlson, Cicchetti, Barnett, & Braunwald, 1989; Lyons-Ruth, Repacholi, McLeod, & Silva, 1991) and is associated with severe difficulties in stress management and dissociative behavior in later life (van Ijzendoorn, Schuengel, & Bakersman-Kranenburg, 1999). In the broadest sense, this work utilizes a psychoneurobiological perspective to attempt to explicate "how external events may impact on intrapsychic structure and development for infants and children already burdened by high psycho-social risk" (Osofsky, Cohen, & Drell, 1995, p. 596). These models are offered as heuristic proposals that can be evaluated by experimental and clinical research.

An Overview of Traumatic Attachments and Brain Development

Development may be conceptualized as the transformation of external into internal regulation. This progression represents an increase of complexity of the maturing brain systems that adaptively regulate the interaction between the developing organism and the social environment. The experiences necessary for this experience-dependent maturation are created within the attachment context, the dyadic regulation of emotions. More specifically, as outlined in the previous paper, the primary caregiver of the securely attached infant affords emotional access to the child and responds appropriately and promptly to his or her positive and negative states. She allows for the interactive generation of high levels of positive affect in co-shared play states, and low levels of negative affect in the interactive repair of social stress, i.e., attachment ruptures.

Because stable attachment bonds are vitally important for the infant's continuing neurobiological development, these dyadically regulated events scaffold an expansion of the childis coping capacities, and therefore adaptive infant and later adult mental health. In psychobiological research on mother-infant affiliative processes, Kalin, Shelton, and Lynn describe the long-enduring effects of such transactions (1995, pp. 740-741):

The quality of early attachment is known to affect social relationships later in life. Therefore, it is conceivable that the level of opiate activity in a mother and her infant may not only affect behaviors during infancy, but may also affect the development of an individualis style of engaging and seeking out supportive relationships later in life.

In contrast to this scenario, the abusive caregiver not only shows less play with her infant, she also induces traumatic states of enduring negative affect. Because her attachment is weak, she provides little protection against other potential abusers of the infant, such as the father. This caregiver is inaccessible and reacts to her infant's expressions of emotions and stress inappropriately and/or rejectingly, and shows minimal or unpredictable participation in the various types of arousal regulating processes. Instead of modulating she induces extreme levels of stimulation and arousal, either too high in abuse or too low in neglect, and because she provides no interactive repair the infantis intense negative emotional states last for long periods of time. Such states are accompanied by severe alterations in the biochemistry of the immature brain, especially in areas associated with the development of the childis coping capacities (Schore, 1996; 1997a).

There is now agreement that repetitive, sustained emotional abuse is at the core of childhood trauma (Oi Hagan, 1995), and that prenatal maltreatment or neglect compromises cognitive development (Trickett & McBride-Chang, 1995). In line with the

established general principle that childhood abuse is a major threat to childrenis mental health (Hart & Brassard, 1987), a context of very early relational trauma serves as a matrix for maladaptive infant (and later adult) mental health. Current developmental research is now delving into the most severe forms of attachment disturbances, reactive (Boris & Zeanah, 1999) and disorganized (Lyons-Ruth & Jacobvitz, 1999; Solomon & George, 1999) attachment disorders, and are offering neurobiological models that underlie these early appearing psychopathologies (Hinshaw-Fuselier, Boris, & Zeanah, 1999). Such massive attachment dysfunctions are clearly prime examples of maladaptive infant mental health.

It has been said that "sexual trauma and childhood abuse may simply be the most commonly encountered severely aversive events inherent in our culture" (Sirven & Glasser, 1998, p. 232). Trauma in the first two years, as at any point in the lifespan, can be inflicted upon the individual from the physical or interpersonal environment. It is now established, however, that social stressors are "far more detrimental" than non-social aversive stimuli (Sgoifo et al., 1999). For this reason I will use the term "relational trauma" throughout this work. Because such trauma is typically "ambient," the stress embedded in ongoing relational trauma is therefore not "single-event" but "cumulative." Because attachment status is the product of the infantis genetically-encoded psychobiological predisposition and the caregiver experience, and attachment mechanisms are expressed throughout later stages of life, early relational trauma has both immediate and long-term effects, including the generation of risk for later-forming psychiatric disorders.

Within the biopsychosocial model of infant psychiatry, the diathesis-stress concept prescribes that psychiatric disorders are caused by a combination of a genetic-constitutional predisposition and environmental or psychosocial stressors that activate the inborn neurophysiological vulnerability. In light of the fact that the brain growth spurt begins in the third trimester in utero (Dobbing & Smart, 1974), genetic-constitutional factors can be negatively impacted during this period by adverse conditions within the uterine maternal-infant environment. For example, very recent research shows that maternal hormones regulate the expression of genes in the fetal brain, and that acute changes in maternal hormone induce changes in gene expression in the fetal brain that are retained when it reaches adulthood (Dowling et al., 2000). Other sudies reveal that high levels of maternal corticotropin-releasing hormone during pregnancy negatively affects fetal brain development (Glynn, Wadhwa, & Sandman, 2000) and reduces later postnatal capacities to respond to stressful challenge (Williams, Hennessey, & Davis, 1995).

These and other data indicate that certain maternal stimuli that impinge upon the fetus negatively impact the hypothalamo-pituitary-adrenocortical (HPA) axis (Sandman et al., 1994; Glover, 1997; Weinstock, 1997) and thereby produce an enduring neurophysiological vulnerability. There is now convincing evidence of the enduring detrimental effects of maternal alcohol (Streissguth et al., 1994), drug (Jacobson et al., 1996; Espy, Kaufman, & Glisky, 1999), and tobacco (Fergusson, Woodward, & Horwood, 1998) use during pregnancy on the childis development. These risk factors in part reflect a delay in postnatal brain development (Huppi et al., 1996) which is expressed not only in prematurity and low birth weight, but also in poor infant interactive capacities (Aitken & Trevarthen, 1997). These limitations in social responsiveness may be aligned with parental avoidance or rejection (Field, 1977), and even physical abuse of the premature infant (Hunter et al., 1978).

Various maternal behaviors may severely dysregulate the homeostasis and even future development of the developing fetus, yet these are not usually considered to be instances of trauma. On the other hand, caregiver abuse and neglect of the postnatal infant are viewed as clear examples of relational trauma. Again, in neonatal phases, both genetic factors that influence stress responsivity and detrimental environmental effects interact to contribute to a behavioral outcome, that is stress exaggerates the effects of a

developmental lesion (Lipska & Weinberger, 1995). This biopsychosocial model suggests that high risk infants born with delayed brain development and poor interactive capacites, and thereby a vulnerable predisposition, would experience even low levels of relational stress as traumatic, while an infant with a more durable constitution would tolerate higher levels of dyadic misattunement before shifting into dysregulation. There is no one objective threshhold at which all infants initiate a stress response, rather this is subjectively determined and created within a unique organismic-environmental history. Even so, the severe levels of stress associated with infant abuse and neglect are pathogenic to all immature human brains, and the latter maybe even more detrimental to development than the former.

These principles suggest that caregiver-induced trauma is qualitatively and quantitatively more potentially psychopathogenic than any other social or physical stressor (aside from those that directly target the developing brain). In an immature organism with undeveloped and restricted coping capacities, the primary caregiver is the source of the infantis stress regulation, and therefore sense of safety. When not safety but danger emanates from the attachment relationship, the homeostatic assaults have significant short- and long-term consequences on the maturing psyche and soma. The stress regulating systems that integrate mind and body are a product of developing limbic-autonomic circuits (Rinaman, Levitt, & Card, 2000), and since their maturation is experience-dependent, during their critical period of organization they are vulnerable to relational trauma. Very recent basic research is revealing that perinatal distress leads to a blunting of the stress response in the right (and not left) prefrontal cortex that is manifest in adulthood (Brake, Sullivan, & Gratton, 2000), and that interruptions of early cortical development specifically affect limbic association areas and social behavior (Talamini et al., 1999).

The nascent psychobiological systems that support the primordial motive systems to attach are located in subcortical components of the limbic system. These brainstem neuromodulatory and hypothalamic neuroendocrine systems that regulate the HPA axis are in a critical period of growth pre- and postnatally, and they regulate the maturation of the later-developing cerebral cortex (Bear & Singer, 1986; Schore, 1994; Osterheld-Haas, Van der Loos, & Hornung, 1994; Aitken & Trevarthen, 1997; Durig & Hornung, 2000). Severe attachment problems with the caregiver negatively impact the postnatal development of these biogenic amine systems (Kraemer & Clarke, 1996).

In human infancy, relational trauma, like exposure to inadequate nutrition during the brain growth spurt (Levitsky & Strupp, 1995; Mendez & Adair, 1999), to biological pathogens or chemical agents that target developing brain tissue (Connally & Kvalsvig, 1993), and to physical trauma to the babyis brain (Anderson et al., 1999), interferes with the experience-dependent maturation of the brainis coping systems, and therefore have a long-enduring negative impact on the trajectory of developmental processes.

Negative Impact of Relational Trauma on Infant Mental Health

The neuropsychobiological literature underscores a central finding of developmental science - that the maturation of the infantis brain is experience-dependent, and that these experiences are embedded in the attachment relationship (Schore, 1994, 2000b; Siegel, 1999). If there is truth to the dictum that security of the attachment bond is a primary defense against trauma-induced psychopathology, then what about the infant who doesnit have such an experience but its antithesis? And since attachment transactions occur in a period in which the brain is massively developing, what is the future course of the brain/mind/body of an infant who does not have the good fortune of engaging with a caregiver who co-creates the childis internal sense of emotional security? What if the brain is evolving in an environment of not interpersonal security, but danger? Is this a context for the intergenerational transmission of psychopathology, and the origins of maladaptive infant mental health? Will early trauma have lasting consequences

for future mental health, in that the trajectory of the developmental process will be altered?

This portrait of infancy is usually not presented by the media, or even in current books on the effects of early experience on brain development (e.g., Bruer, 1999; Gopnik, Meltzoff, & Kuhl, 1999). This is not the image of a "scientist in the crib," but rather of "ghosts in the nursery" (Fraiberg, Adelson, & Shapiro, 1975). In fact, this infant is depicted in Karr-Morse and Wileyis (1997) Ghosts From the Nursery: Tracing the Roots of Violence. These authors ask, what is the effect of early trauma, abuse and/or neglect, on developing brain anatomy? And how does this effect the future emotional functioning of the individual as he or she passes into the next stages of the lifespan?

In his last work Freud (1940) observed that trauma in early life effects all vulnerable humans because "the ego...is feeble, immature and incapable of resistance." In recent thinking, this dictum translates to the principle that the infantis immature brain is in a state of rapid development, and is therefore exquisitely vulnerable to early adverse experiences, including adverse social experiences. An entire recent issue of the journal Biological Psychiatry is devoted to development and vulnerability (Foote, 1999), and in it De Bellis et al. present two papers on developmental traumatology and conclude, "the overwhelming stress of maltreatment in childhood is associated with adverse influences on brain development" (1999, p. 1281).

A number of scientific and clinical disciplines are now focusing on not only the interactional aspects of early trauma, but also on the untoward effects of abuse and deprivational neglect on the development of the infant brain. In a major advance of our knowledge, discoveries in the developmental sciences now clearly show that the primary caregiver acts as an external psychobiological regulator of the "experience-dependent" growth of the infant's nervous system (Schore, 1994, 1996, 1997a, 2000c). These early social events are imprinted into the neurobiological structures that are maturing during the brain growth spurt of the first two years of life, and therefore have far-reaching effects. Eisenberg (1995) refers to "the social construction of the human brain," and argues that the cytoarchitectonics of the cerebral cortex are sculpted by input from the social environment. The social environment can positively or negatively modulate the developing brain.

Early relational trauma, which is usually not a singular event but "ambient" and "cumulative," is of course a prime example of the latter. These events may not be so uncommon. In 1995 over 3 million children in this country were reported to have been abused or neglected (Barnet & Barnet, 1998), and the Los Angeles Times reported that in California, in 1997, there were 81,583 reported cases of neglect and 54, 491 reported cases of physical abuse. Although these sources did not specify how many infants were in these categories, other evidence indicates that in the United States the most serious maltreatment occurs to infants under 2 years of age (National Center of Child Abuse and Neglect, 1981). Homicide (Karr-Morse and Wiley, 1997) and traumatic head injury (Colombani et al., 1985) are the leading causes of death for children under 4.

A 1997 issue of Pediatrics contains a study of covert videorecordings of infants hospitalized for life-threatening events, and it documents, in a most careful and disturbing manner, the various forms of child abuse that are inflicted by caregivers on infants as young as 3 months while they are in the hospital (Southall et al., 1997). These experiences are recorded and stored in the infant. Terr (1988, p. 103) has written that "literal mirroring of traumatic events by behavioral memory [can be] established at any age, including infancy." According to Luu and Tucker, "To understand neuropsychological development is to confront the fact that the brain is mutable, such that its structural organization reflects the history of the organism" (1996, p. 297).

Because early abuse negatively impacts the developing brain of these infants, it has enduring effects. There is extensive evidence that trauma in early life impairs the development of the capacities of maintaining interpersonal relationships, coping with stressful stimuli, and regulating emotion. A body of interdisciplinary research demonstrates that the essential experiences that shape the individualis patterns of coping responses are forged in the emotion-transacting caregiver-infant relationship (Schore, 1994; 2000b). We are now beginning to understand, at a psychobiological level, specifically how beneficial early experiences enhance and detrimental early histories inhibit the development of the brainis active and passive stress coping mechanisms.

The current explosion of developmental studies are highly relevant to the problem of how early trauma uniquely alters the ongoing maturation of the brain/mind/body. As Gaensbauer and Siegel have written, prolonged and frequent episodes of intense and unregulated interactive stress in infants and toddlers have devastating effects on "the establishment of psychophysiological regulation and the development of stable and trusting attachment relationships in the first year of life" (1995, p. 294). Perhaps even more revealing is the fact that these early dysregulating experiences lead to more than an insecure attachment, they trigger a chaotic alteration of the emotion processing limbic system that is in a critical period of growth in infancy. The limbic system has been suggested to be the site of developmental changes associated with the rise of attachment behaviors (Anders & Zeanah, 1984) and to be centrally involved in the capacity "to adapt to a rapidly changing environment" and in "the organization of new learning" (Mesulam, 1998, p. 1028). These limbic circuits are particularly expressed in the right hemisphere (Tucker, 1992; Joseph, 1996), which is in a growth spurt in the first two years of life (Schore, 1994).

There is now agreement that, in general, the enduring effects of traumatic abuse are due to deviations in the development of patterns of social information processing. I suggest that, in particular, early trauma alters the development of the right brain, the hemisphere that is specialized for the processing of socioemotional information and bodily states. The early maturing right cerebral cortex is dominant for attachment functions (Henry, 1993; Schore, 1994, 2000b, c; Siegel, 1999) and stores an internal working model of the attachment relationship. An enduring developmental impairment of this system would be expressed as a severe limitation of the essential activity of the right hemisphere - the control of vital functions supporting survival and enabling the organism to cope actively and passively with stressors (Wittling & Schweiger, 1993).

Davies and Frawley (1994) describe the immediate effects of parent-inflicted trauma on attachment:

The continued survival of the child is felt to be at risk, because the actuality of the abuse jeopardizes (the) primary object bond and challenges the childis capacity to trust and, therefore, to securely depend (p. 62).

In contexts of relational trauma the caregiver, in addition to dysregulating the infant, withdraws any repair functions, leaving the infant for long periods in an intensely disruptive psychobiological state that is beyond her immature coping strategies. In studies of a neglect paradigm, Tronick and Weinberg describe:

When infants are not in homeostatic balance or are emotionally dysregulated (e.g., they are distressed), they are at the mercy of these states. Until these states are brought under control, infants must devote all their regulatory resources to reorganizing them. While infants are doing that, they can do nothing else (1997, p. 56).

In other words, infants who experience chronic relational trauma too frequently forfeit potential opportunities for socioemotional learning during critical periods of right brain development.

But there is also a pernicious long-term consequence of relational trauma - an enduring deficit at later points of the life span in the individualis capacity to assimilate novel (and thus stressful) emotional experiences. At the end of the nineteenth century Janet (1889) speculated:

All [traumatized] patients seem to have the evolution of their lives checked; they are attached to an unsurmountable object. Unable to integrate traumatic memories, they seem to have lost their capacity to assimilate new experiences as well. It is...as if their personality development has stopped at a certain point, and cannot enlarge any more by the addition of new elements.

The functional limitations of such a system are described by Hopkins and Butterworth (1990): "Undifferentiated levels of development show relatively rigid but unstable modes of organization in which the organism cannot adapt responses to marked changes coming from within or without" (p. 9). From a psychoanalytic perspective, Emde (1988) defines pathology as a lack of adaptive capacity, an incapacity to shift strategies in the face of environmental demands. In psychiatric writings, van der Kolk (1996) asserts that under ordinary conditions traumatized individuals adapt fairly well, but they do not repond to stress the way others do, and Bramsen, Dirkzwager, and van der Ploeg observe that in the aftermath of trauma, certain personality traits predispose individuals to engage in less successful coping strategies. All of these all descriptions characterize an immature right brain, the locus of the human stress response (Wittling, 1997).

This structural limitation of the right brain is responsible for the individualis inability to regulate affect. As van der Kolk and Fisler (1994) have argued, the loss of the ability to regulate the intensity of feelings is the most far-reaching effect of early trauma and neglect. I further suggest that significantly altered early right brain development is reflected in a "type D" (Main & Solomon, 1986) the disorganized/disoriented attachment seen in abused and neglected infants (Carlson et al., 1989; Lyons-Ruth et al., 1991). This severe right brain attachment pathology is involved in the etiologies of a high risk for both posttraumatic stress disorder (Schore, 1997a; 1998c, e; 1999c, d; 2000d) and a predisposition to relational violence (Lyons-Ruth & Jacobvitz, 1999; Schore, 1999b). In discussing the characteristics of toddlers and preschoolers exhibiting severe psychiatric disturbance, Causey, Robertson, and Elam (1998) report that a large number of these young patients were neglected and/or physically or sexually abused. Main (1996) argues that "disorganized" and "organized" forms of insecure attachment are primary risk factors for the development of mental disorders.

The Neurobiology of Infant Trauma

Although the body of studies on childhood trauma is growing, to this date there is still hardly any research on infant trauma. A noteworthy example is the work of Perry and his colleagues, which is extremely valuable because it includes not just behavioral but also developmental neurobiological and psychobiological data. Perry et al. (1995) demonstrate that the human infantis psychobiological response to trauma is comprised of two separate response patterns, hyperarousal and dissociation. In the initial stage of threat, a startle or alarm reaction is initiated, in which the sympathetic component of the autonomic nervous system (ANS) is suddenly and significantly activated, resulting in increased heart rate, blood pressure, respiration, and muscle tone, as well as hypervigilance. Distress is expressed in crying and then screaming.

In very recent work, this dyadic transaction is described by Beebe as "mutually escalating overarousal" of a disorganized attachment pair:

Each one escalates the ante, as the infant builds to a frantic distress, may scream, and, in this example, finally throws up. In an escalating overarousal pattern, even after

extreme distress signals from the infant, such as ninety-degree head aversion, arching away...or screamimg, the mother keeps going (2000, p. 436).

The infantis state of "frantic distress," or what Perry terms fear-terror is mediated by sympathetic hyperarousal, known as ergotropic arousal (Gellhorn, 1967. It reflects excessive levels of the major stress hormone corticotropin releasing factor (CRF) which regulates catecholamine activity in the sympathetic nervous system (Brown et al., 1982). Noradrenaline is also released from the locus coeruleus (Svensson, 1987; Butler et al., 1990; Aston-Jones et al, 1996). The result is rapid and intensely elevated noradrenaline and adrenaline levels which trigger a hypermetabolic state within the brain. In such "kindling" states (Adamec, 1990; Post et al., 1997), very large amounts of CRF and glutamate, the major excitatory neurotransmitter in the brain (Chambers et al., 1999), are expressed in the limbic system (Schore, 1997a). Harkness and Tucker (2000) state that early traumatic experiences, such as childhood abuse, literally kindle limbic areas.

But Perry's group describes a second, later-forming reaction to infant trauma, dissociation, in which the child disengages from stimuli in the external world and an attends to an "internal" world. The childis dissociation in the midst of terror involves numbing, avoidance, compliance and restricted affect. Traumatized infants are observed to be staring off into space with a glazed look. This behavioral strategy is described by Tronick and Weinberg:

When infants' attempts to fail to repair the interaction infants often lose postural control, withdraw, and self-comfort. The disengagement is profound even with this short disruption of the mutual regulatory process and break in intersubjectivity. The infantis reaction is reminiscent of the withdrawal of Harlowis isolated monkey or of the infants in institutions observed by Bowlby and Spitz (1997, p. 66).

The state of conservation-withdrawal (Kaufman & Rosenblum, 1967, 1969; Schore, 1994) is a parsympathetic regulatory strategy that occurs in helpless and hopeless stressful situations in which the individual becomes inhibited and strives to avoid attention in order to become "unseen." This state is a primary hypometabolic regulatory process, used throughout the lifespan, in which the stressed individual passively disengages in order "to conserve energies...to foster survival by the risky posture of feigning death, to allow healing of wounds and restitution of depleted resources by immobility" (Powles, 1992, p. 213). It is this parasympathetic mechanism that mediates the "profound detachment" (Barach, 1991) of dissociation. If early trauma is experienced as "psychic catastrophe" (Bion, 1962), dissociation represents "detachment from an unbearable situation" (Mollon, 1996), "the escape when there is no escape" (Putnam, 1997), and "a last resort defensive strategy" (Dixon, 1998).

Most importantly, the neurobiology of the later-forming dissociative reaction is different than the initial hyperarousal response. In this passive state pain numbing and blunting endogenous opiates are elevated. These opioids, especially enkephalins, instantly trigger pain-reducing analgesia and immobility (Fanselow, 1986) and inhibition of cries for help (Kalin, 1993). In addition, the behavior-inhibiting steroid, cortisol is elevated. The inhibition produced by cortisol results from the rapid modulation of gamma-aminobutyric acid (GABA) receptors by cortisol metabolites (Majewska et al., 1986; Orchinik, Murray, & Moore, 1994). GABA is the principal inhibitory neurotransmitter in the brain.

Furthermore, vagal tone increases dramatically, decreasing blood pressure and heart rate, despite increases in circulating adrenaline. This increased parasympathetic trophotropic hypoarousal (Gellhorn, 1967) allows the infant to maintain homeostasis in the face of the internal state of sympathetic ergotropic hyperarousal. In the traumatic state, and it may be long-lasting, both the sympathetic energy-expending and parasympathetic energy-conserving components of the infantis developing ANS are hyperactivated.

In the developing brain states organize neural systems, resulting in enduring traits. That is, traumatic states in infancy trigger psychobiological alterations that effect state-dependent affect, cognition, and behavior. But since they are occurring in a critical period of growth of the emotion regulating limbic system, they negatively impact the experience-dependent maturation of the structural systems that regulate affect, thereby inducing characterological styles of coping that act as traits for regulating stress. In light of the principle that "Critical periods for pathogenic influences might be prolonged in these more slowly maturing systems, of which the prefrontal cortex is exemplary" (Goldberg & Bilder, p. 177), prefrontolimbic areas would be particularly vulnerable. What psychoneurobiological mechanism could account for this?

The brain of an infant who experiences frequent intense attachment disruptions is chronically exposed to states of impaired autonomic homeostasis which he/she shifts into in order to maintain basic metabolic processes for survival. If the caregiver does not participate in stress-reparative functions that reestablish psychobiological equilibrium, the limbic connections in the process of developing are exposed to high levels of excitotoxic neurotransmitters, such as glutamate (Choi, 1992; Moghaddam, 1993) as well as cortisol (Moghaddam et al., 1994; Schore, 1997a) for long periods of time. The neurotoxic effects of glucocorticoids are synergistcally amplified by simultaneous activation of the excitotoxic N-methyl-D-aspartate (NMDA)-sensitive glutamate receptor, a critical site of neurotoxicity and synapse elimination in early development (McDonald, Silverstein, & Johnston, 1988; Guilarte, 1998).

It is known that stress-induced increases of glucocorticoids in postnatal periods selectively induce neuronal cell death in "affective centers" in the limbic system (Kathol et al., 1989), imprint an abnormal limbic circuitry (Benes, 1994), and produce permanent functional impairments of the directing of emotion into adaptive channels (DeKosky, Nonneman, & Scheff, 1982). The interaction between corticosteroids and excitatory transmitters is now thought to mediate programmed cell death and to represent a primary etiological mechanism for the pathophysiology of neuropsychiatric disorders (Margolis, Chuang, & Post, 1994). Here is a template for impaired limbic morphogenesis, a structural alteration which will reduce future adaptive coping functions. This is a context for psychopathogenesis.

The major environmental influence on the development of the limbic structures involved in organismic coping is the attachment relationship. Severe disruption of attachment bonds in infancy leads to a regulatory failure expressed in disturbances in limbic activity, hypothalamic dysfunction, and impaired autonomic homeostasis (Reite & Capitanio, 1985). The dysregulating events of abuse and neglect produce extreme and rapid alterations of ANS sympathetic ergotropic hyperarousal and parasympathetic trophotropic hypoarousal that create chaotic biochemical alterations, a toxic neurochemistry in the developing brain.

The neurochemistry of brain growth is essentially regulated by the monoaminergic neuromodulators, especially the biogenic amines dopamine, noradrenaline, and serotonin, and the neuropeptide and steroid neurohormones. In critical periods, increased production of these agents, many of which are trophic, are matched by increased production of the receptors of such agents. Prenatal stress is known to alter biogenic amine levels on a long-lasting basis (Schneider et al., 1998). Postnatal traumatic stress also induces excessive levels of dopamine, activating excitatory NMDA receptor binding of glutamate (Knapp, Schmidt, & Dowling, 1990). Excitatory neurotransmitters regulate postsynaptic calcium influx in developing neocortex (Yuste & Katz, 1991) and glutamate acting at NMDA receptors increases intracellular calcium in neurons (Burgoyne, Pearce, & Cambray-Deakin, 1988), which, if uncontrolled, leads to intracelluar damage or cell death (Garthwaite & Garthwaite, 1986).

In other words, intense relational stress alters calcium metabolism in the infantis brain, a critical mechanism of cell death (Farber, 1981). Dopamine (Filloux & Townsend, 1993;

McLaughlin et al., 1998) and glutamate (Tan et al., 1998) can be neurotoxic, by generating superoxide free radicals associated with oxidative stress (Lafon-Cazal, Pietri, Culcas, & Bockaert, 1993), especially hydroxyl radicals which destroy cell membranes (Lohr, 1991). These events greatly enhance "apoptotic" or "programmed cell death" (Margolis et al., 1994; Schore, 1997a). During a critical period of growth of a particular brain region, DNA production is highly increased, and so excitotoxic stress, which is known to cause oxidative damage to DNA, lipid membrane, and protein (Liu et al., 1996), also negatively impacts the genetic systems within evolving limbic areas.

Indeed, there is now evidence to show that adverse social experiences during early critical periods result in permanent alterations in opiate, corticosteroid, corticotropin releasing factor, dopamine, noradrenaline, and serotonin receptors (Coplan et al., 1996; Ladd et al., 1996; Lewis et al., 1990; Martin et al., 1991; Rosenblum et al., 1994; van der Kolk, 1987). Such receptor alterations are a central mechanism by which "early adverse developmental experiences may leave behind a permanent physiological reactivity in limbic areas of the brain" (Post, Weiss, & Leverich, 1994, p. 800).

It is now established that "dissociation at the time of exposure to extreme stress appears to signal the invocation of neural mechanisms that result in long-term alterations in brain functioning" (Chambers et al., 1999, p. 274). In other words, infants who experience states of terror and dissociation and little interactive repair, especially those with a genetic-constitutional predisposition and an inborn neurophysiological vulnerability, are high risk for developing severe psychopathologies at later stages of life. Bowlby asserted,

since much of the development and organization of [attachment] behavioral systems takes place whilst the individual is immature, there are plenty of occasions when an atypical environment can divert them from developing on an adaptive course (1969, p. 130).

Recall, attachment involves limbic imprinting, and so infant trauma will interfere with the critical period organization of the limbic system, and therefore impair the individualis future capacity to adapt to a rapidly changing environment and to organize new learning (Mesulam, 1998). Maladaptive infant mental health is therefore highly correlated with maladaptive adult mental health.

The infant posttraumatic stress disorder of hyperarousal and dissociation thus sets the template for later childhood, adolescent, and adult posttraumatic stress disorders (PTSD), all of which show disturbances of autonomic arousal (Prins, Kaloupek, & Keane, 1995) and abnormal catecholaminergic function (Southwick et al., 1993). In each, "chronic, inescapable or uncontrollable stress may lead to impairment of the normal counter-regulatory mechanisms producing hyperactivity of the hypothalamic-pituitary-adrenal and sympathetic nervous systems, which could lead to excessive anxiety, feelings of hopelessness and defeat, and depression" (Weinstock, 1997, p. 1). The latter symptomatic triad represents unregulated parasympathetic activity that is associated with dissociation. At any point of the lifespan, dissociative defensive reactions are elicited almost instantaneously.

This continuity in infant and adult coping deficits is described by Nijenhuis, Vanderlinden, and Spinhoven (1998):

The stress responses exhibited by infants are the product of an immature brain processing threat stimuli and producing appropriate responses, while the adult who exhibits infantile responses has a mature brain that, barring stress-related abnormalities in brain development, is capable of exhibiting adult response patterns. However, there is evidence that the adult brain may regress to an infantile state when it is confronted with severe stress (pp. 253).

But, as we have seen, developmental neurobiological studies now demonstrate that "the overwhelming stress of maltreatment in childhood is associated with adverse influences on brain development" (De Bellis et al. 1999, p. 1281), and that "early adverse experiences result in an increased sensitivity to the effects of stress later in life and render an individual vulnerable to stress-related psychiatric disorders" (Graham et al., 1999, p. 545).

The Neuropsychology and Neuropsychoanalysis of a Disorganized / Disorriented Attachment Pattern

The next question is, how would the trauma-induced psychobiological and neurobiological alterations of the developing brain be expressed in the behavior of an early traumatized toddler? We have the data. In a classic study, Main and Solomon (1986) studied the attachment patterns of infantis who had suffered trauma in the first year of life. This lead to the discovery of a new attachment category, "type D", an insecure-disorganized / disoriented pattern. [This work is updated and summarized by Solomon and George (1999) in a recent volume, Attachment Disorganization].

The "type D" pattern is found in over 80% of maltreated infants (Carlson et al., 1989). Indeed Spangler and Grossman (1999) demonstrate that this group of toddlers exhibits the highest heart rate activation and most intense alarm reaction in the strange situation procedure (see Figure 1). They also show higher cortisol levels than all other attachment classifications and are at greatest risk for impaired hypothalamo-pituitary-adrenocortical axis stress responding (Hertsgaard et al., 1995). Main and Solomon conclude that "these infants are experiencing low stress tolerance" (1986, p. 107). These authors contend that the disorganization and disorientation reflect the fact that the infant, instead of finding a haven of safety in the relationship, is alarmed by the parent. They note that because the infant inevitably seeks the parent when alarmed, any parental behavior that directly alarms an infant should place it in an irresolvable paradox in which it can neither approach, shift its attention, or flee. At the most basic level, these infants are unable to generate a coherent behavioral coping strategy to deal with this emotional challenge.

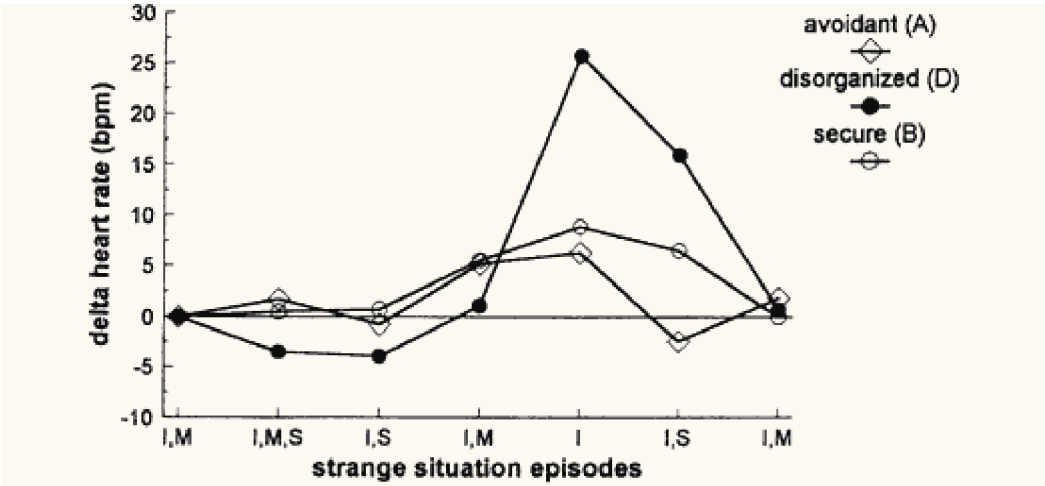

FIGURE 1. Changes in heart rate during strange situation episodes for different attachment groups (M, mother, I, infant, S, stranger) (from Spangler & Grossman, 1999, and used with the permission of the Society for Research in Child Development).

Main and Solomon documented, in some detail, the uniquely disturbing behaviors these 12-month-old infants show in Strange Situation reunion transactions. These episodes of interruptions of organized behavior and low stress tolerance are often brief, frequently lasting 10-30 seconds, yet they are highly significant. For example, they show a simultaneous display of contradictory behavior patterns, such as "backing" towards the parent rather than approaching face-to-face.

The impression in each case was that approach movements were continually being inhibited and held back through simultaneous activation of avoidant tendencies. In most cases, however, proximity-seeking sufficiently "over-rode" avoidance to permit the increase in physical proximity. Thus, contradictory patterns were activated but were not mutually inhibited (Main & Solomon, 1986, p. 117).

Notice the simultaneous activation of the energy expending sympathetic and energy conserving parasympathetic components of the ANS.

Maltreated infants also show evidence of apprehension and confusion, as well as very rapid shits of state during the stress inducing Strange Situation.

One infant hunched her upper body and shoulders at hearing her mother's call, then broke into extravagent laugh-like screeches with an excited forward movement. Her braying laughter became a cry and distress-face without a new intake of breath as the infant hunched forward. Then suddenly she became silent, blank and dazed (Main & Solomon, 1986, p. 119).

A dictionary definition of apprehension is distrust or dread with regard to the future. These apprehensive behaviors generalize beyond just interactions with the mother. The intensity of the babyis dysregulated affective state is often heightened when the infant is exposed to the added stress of an unfamiliar person. At a strangeris entrance, two infants moved away from both mother and stranger to face the wall, and another "leaned forehead against the wall for several seconds, looking back in apparent terror" (Main & Solomon, 1986).

These maltreated infants also showed "behavioral stilling" - that is, "dazed" behavior and depressed affect (again a hyperactivation of the PNS). One infant "became for a moment excessively still, staring into space as though completely out of contact with self, environment, and parent" (p. 120) Another showed "a dazed facial appearance...accompanied by a stilling of all body movement, and sometimes a freezing of limbs which had been in motion". And yet another "fell face-down on the floor in a depressed posture prior to separation, stilling all body movements".

Furthermore, Main and Solomon point out that the type "D" behaviors take the form of stereotypies that are found in neurologically impaired infants. It should be emphasized that these behaviors are overt manifestations of an obviously impaired regulatory system, one that rapidly disorganizes under stress. Notice that these observations are taking place at 12 to 18 months, a critical period of corticolimbic maturation, and they reflect a severe structural impairment of the orbitofrontal control system that is involved in attachment behavior and state regulation. The orbitofrontal areas, like other limbic structures in the anterior temporal areas and the amygdala, contains neurons that fire to emotionally expressive faces. The motheris face is the most potent visual stimulus in the childis world, and it is well known that direct gaze can mediate powerful aggressive messages.

During the trauma, the infant is presented with an aggressive expression on the motheris face. The image of this aggressive face, as well as the chaotic alterations in the infantis bodily state that are associated with it, is indelibly imprinted into subcortical limbic circuits as a "flashbulb memory" (Brown & Kulik, 1977) and thereby stored in implicit-procedural memory in the visuospatial right hemisphere. These are stored memories of what Lieberman (1997) calls "negative maternal attributions" that contain an intensely negative affective charge, and therefore rapidly dysregulate the infant.

In the course of the traumatic interaction, the infant is presented with another affectively overwhelming facial expression, a maternal expression of fear-terror. Main and Solomon note that this occurs when the mother withdraws from the infant as though the infant

were the source of the alarm, and they report that dissociated, trancelike, and fearful behavior is observed in parents of type "D" infants. Current studies show a link between frightening maternal behavior and disorganized infant attachment (Schuengel, Bakersmans-Kranenburg, & Van Ijzendoorn, 1999). I suggest that during these episodes the infant is matching the rhythmic structures of the motheris dysregulated states, and that this synchronization is registered in the firing patterns of the stress-sensitive corticolimbic regions of the infantis brain that are in a critical period of growth. This is the context of the down-loading of programs of psychopathogenesis.

Mothers of children with disorganized attachment describe themselves as unable to care for or protect their infants, inflicting harsh punishments, feeling depressed, and being out of control. In general, high risk and physically abusive mothers, relative to comparison mothers, differ in the types of perceptions, attributions, evaluations, and expectations of their childrenis behavior, engage in fewer interactions and communicate less with their children, use fewer positive parenting behaviors, and use more aversive disciplinary techniques, (Nayak & Milner, 1998). Role reversal (Mayseless, 1998) and a subjective feeling of helplessness (George & Solomon, 1996) are commonly found mothers of disorganized infants. In light of the fact that many of these mothers have suffered from unresolved trauma themselves (Famularo, Kinscherff, & Fenton, 1992), this spatiotemporal imprinting of the chaotic alterations of the motheris dysregulated state may be a central mechanism for the "intergenerational transmission child abuse" (Kaufman & Zigler, 1989).

Current research on the neurobiology of attachment is revealing that the early experiences of female infants with their mothers (or absence of these experiences) influence how they respond to their own infants when they later become mothers, and that this provides a psychobiological mechanism for the intergenerational transmission of adaptive and maladaptive parenting styles and responsiveness (Fleming, OiDay, & Kraemer, 1999). This psychobiological principle is advanced in the very recent clinical writings of Silverman and Lieberman, who conclude that although the motheris caregiving system has an instinctual basis, it is expressed through the filter of her own representational templates, "which derive from her sense of being cared and protected in her relationship with her own parents" (1999, p. 172). This experience did not occur in the abusive motheris early attachment.

In the latest biosocial model of the determinants of motherhood, Pryce (1995) views parenting as varying on a continuum between the extremes of maximal care and infant/abuse and neglect. Expanding upon these ideas, Maestripieri (1999) asserts that although models of parenting are often presented in terms of social and cognitive processes, recent biological studies in primates of the neurobiological regulation of parental responsivess and the determinants of infant abuse indicate that human parenting is much more sensitive to neuroendocrine mechanisms than previously thought. He portrays maximal parental care, as represented in a mother

...with a genotype for a secure and sensitive personality, a developmental environment that included a secure attachment to an adequate caregiver and experience of eplay-mothering,i a stress-free pregnancy and postpartum period, optimal neurobiological priming and control, and considerable social support. Such a female will be highly attracted to her infant and made anxious by its crying, but will not averse to her infant or its novelty per se (Maestripieri, 1999, p. 417).

In contrast, the mother characterized as expressing minimal parental care and maximal neglect presents

...with a genotype for a insecure...personality, a developmental environment that included a insecure attachment to a caregiver and no experience with infants, a stressful pregnancy and postpartum period, a suboptimal neurobiological priming and control, and

little or no social support. Such a female will be weakly attracted to her infant and will be averse to the infant, including its crying, its physical burden, and its novelty (Maestripieri, 1999, p. 417).

Maestripieri also suggests that high vulnerability to stress and emotional disorders are common among abusive parents.

Indeed, a vulnerability to dissociation in the postpartum period has been reported by Moleman, van der Hart, and van der Kolk (1992). In a number of cases they describe women panic-striken with the anticipation of losing their babies:

Panic ceased when they dissociated from both their subjective physical experience and from contact with their surroundings. They all continued to experience dissociative phenomena, intrusive recollections about some aspects of the delivery, and amnesia about others, and they all failed to attach to their children (1992, p. 271, my italics).

These symptoms lasted months after the delivery. Notice the authors contention that maternal dissociation blocks infant attachment.

What would be the effect if the motheris dissociative episodes continued as a clinical depression well through the first year of the infantis life? I suggest that in certain critical stressful dyadic moments, this same individual will show a vulnerability for a suboptimal neurobiological priming in the form of dissociation. In light of the fact that infant cries produce elevated physiological reactivity and high levels of negative affect in abusing mothers (Frodi & Lamb, 1980), episodes of "persistent crying" (Papousek & von Hofacker, 1998) may be a potent trigger of dissociation. The caregiveris entrance into a dissociative state represents the real-time manifestation of neglect. Such a context of an emotionally unavailable, dissociating, unresolved/disorganized mother and a disorganized/disoriented infant is evocatively captured by Fraiberg, who provides a painfully vivid description of a dissociative mother and her childis detachment:

The mother had been grudgingly parented by relatives after her motheris postpartum attempted suicide and had been sexually abused by her father and cousin. During a testing session, her baby begins to cry. It is a hoarse, eerie cry...On tape, we see the baby in the motheris arms screaming hopelessly; she does not turn to her mother for comfort. The mother looks distant, self-absorbed. She makes an absent gesture to comfort the baby, then gives up. She looks away. The sceaming continues for five dreadful minutes. In the background we hear Mrs. Adelsonis voice, gently encoraging the mother. eWhat do you do to comfort Mary when she cries like this?i (The mother) murmurs something inaudible...As we watched this tape later...we said to each other incredulously, eItis as if this mother doesnit hear her babyis cries (Fraiberg, cited in Barach, 1991, p. 119).

Ultimately, the child will transition out of hyperexcitation-protest into hyperinhibition-detachment, and with the termination of protest (screaming), sheill become silent. She will shift out of the hyperarousal, and sheill dissociate and match the motheris state. This regulatory failure is experienced as a discontinuity in what Kestenberg (1985) refers to as dead spots in the infant's subjective experience, an operational definition of the restriction of consciousness of dissociation. Winnicott (1958) holds that a particular failure of the maternal holding environment causes a discontinuity in the babyis need for going-on-being, and that this is a central factor in psychopathogensis. And so not just trauma but the infantis posttraumatic response to the relational trauma, the parasympathetic regulatory strategy of dissociation, is built into the personality.

There is a long tradition in the classical psychoanalytic literature of the severely detrimental effects of the traumatic effects of a sudden and unexpected influx of massive external stimulation (sympathetic hyperexcitation) that breaches the infantis stimulus

barrier (Freud, 1920) and precludes successful self-regulation (Freud, 1926). This has lead to an emphasis of the role of overstimulation and annihilation anxieties in classical, object relational, and self psychological models of trauma. I suggest that "screaming hopelessly" is the vocal expression of annihilation anxiety, the threat to oneis bodily wholeness and survival, the annihilation of oneis core being.

However, Freud also described the psychic helplessness associated with the egois immaturity in the first years of childhood, and postulated that the passively experienced re-emergence of the trauma is "a recognized, remembered, expected situation of helplessness." (1926, p. 166). In writings on psychic trauma and "emotional surrender" Anna Freud (1951/1968; 1964/1969) also referred to helplessness, defined as a state of "disorientation and powerlessness" that the organism experiences in the traumatic moment. Although almost all psychoanalytic theoreticians have overlooked or undervalued this, Krystal (1988) and Hurvich (1989) emphasize that at the level of psychic survival helplessness constitutes the first basic danger. This helplessness is an early appearing primitive organismic defense against the growth inhibiting effects of maternal over- or understimulation.

What has been undetermined in this literature is how, as Mahler (1958) states, trauma interferes with psychic structure formation. This question can only be answered with reference to current neurobiological models of developing psychic structure. Translating this into developmental neurobiological concepts, evidence now shows that the neurobiological alterations of traumatic sympathetic hyperexcitation and parasympathetic hyperinhibition on the developing limbic system are profound. Perry state that sympathetically-driven early terror states lead to a "sensitized" hyperarousal response. Due to the alterations of maturing catecholamine systems, "critical physiological, cognitive, emotional, and behavioral functions which are mediated by these systems will become sensitized". According to these authors,

Everyday stressors that previously may not have elicited any response now elicit an exaggerated reactivity...This is due to the fact that...the child is in a persisting fear state (which is now a "trait"). Furthermore, this means that the child will very easily be moved from being mildly anxious to feeling threatened to being terrorized (Perry et al., 1995, p. 278).

Thus, not only is the onset of sympathetically driven fear-alarm states more rapid, but their offset is prolonged, and they endure for longer periods of time. This permanent dysregulation of CRF-driven fear states is described by Heim and Nemeroff, who on the basis of a study of adult survivors of childhood abuse suggest that "stress early in life results in a persistent sensitization of these CRF circuits to even mild stress in adulthood, forming the basis for mood and anxiety disoders" (1999, p. 1518).

But, in addition, due to the chaotic parasympathetic alterations that accompany trauma to the early self, this branch of the ANS also is dysregulated. Deprivation of early maternal stress modulation is known to trigger not only an exaggerated release of corticosteroids upon exposure to novel experiences, but, in addition, inhibitory states that persist for longer periods of time. The result is a quicker access into and a longer duration of dissociated states at later points of stress. This represents a deficit, since adaptive coping is reflected by a the termination of a stress response at an appropriate time in order to prevent an excessive reaction (Weinstock, 1997).

Sroufe and his colleagues conclude that early more so than later trauma has a greater impact on the development of dissociation. They write, "The vulnerable self will be more likely to adopt dissociation as a coping mechanism because it does not have either the belief in worthiness gained from a loving and responsive early relationship or the normal level of defenses and integration that such a belief affords" (Ogawa et al., 1997, p. 875).

Critical Period Trauma and Deficient Orbitofrontal Connectivity

In an editorial of a special issue of Biological Psychiatry, Foote writes, "Combining developmental and affective approaches, it may even be possible to test hypotheses regarding the components of stress and affective circuitry that can exhibit dysregulation following traumatic and/or harmful events, especially early in life" (1999, p. 1457). A developmental perspective can tell us when adverse experiences have the greatest disorganizing impact on evolving adaptive functions, and a neurobiological approach can give us clues as to which limbic circuits that mediate these functions are in a critical period of growth, and therefore most vulnerable. Clearly, attachment neurobiology is centrally involved. This leads to the question, specifically what brain systems involved in attachment are negatively impacted by early abuse and neglect?

Relational trauma in the first through third quarters of the first year negatively impacts the experience-dependent maturation of the amygdala and anterior cingulate limbic circuits (see previous article). But by the end of this year and into the second, the higher corticolimbic circuits are in a critical period of growth, and therefore negatively impacted. Referring back to Main and Solomonis studies, these involved infants of 12 to 18 months, a time when internal working models of the attachment relationship are first assessed by the Strange Situation. Research documents that disorganized infant attachment strategies increase in frequency from 12 to 18 months (Lyons-Ruth, Alpern, & Repacholi, 1993). In fact this interval is a critical period for the experience-dependent maturation of the orbitofrontal areas of the cortex (see Figure 2).

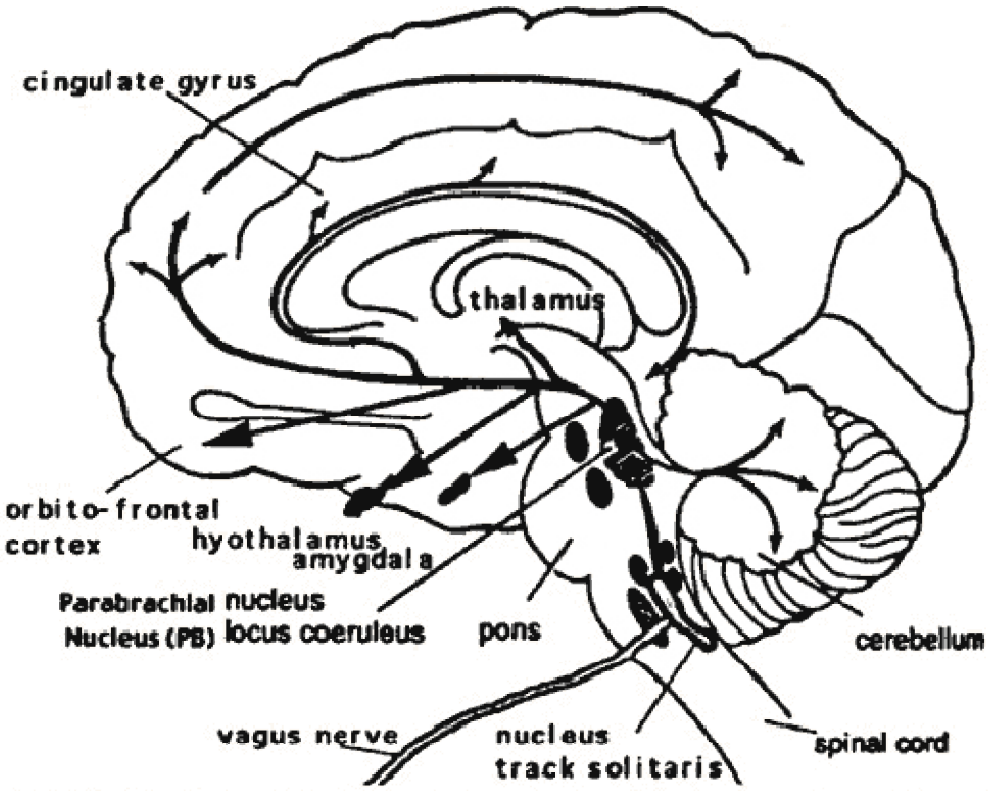

FIGURE 2. Medial view of the right hemisphere, showing limbic structures and connections of the vagus nerve into the medulla. Reprinted by permission of Elsevier Science from "Vagus Nerve Stimulation: A New Tool for Brain Research and Therapy," by George, M.S., et al., Biological Psychiatry, 47, 287–295. Copyright 2000 by the Society of Biological Psychiatry.

Perry et al. (1995) contend that early traumatic environments that induce atypical patterns of neural activity interfere with the organization of cortical-limbic areas and compromise, in particular, such brain-mediated functions as attachment, empathy, and affect regulation. These very same functions are mediated by the frontolimbic areas of the cortex, and because of their dysfunction, affective disturbances are a hallmark of

early trauma. Teicher (1996) reports that children with early physical and sexual abuse show EEG abnormalities in frontotemporal and anterior brain regions. Teicher concludes that stress alters the development of the prefrontal cortex, arrests its development, and prevents it from reaching a full adult capacity. So the next question is, what kind of psychoneurobiological mechanism could account for this prefrontal developmental arrest?

The developing infant is maximally vulnerable to nonoptimal and growth-inhibiting environmental events during the period of most rapid brain growth. During these critical periods of intense synapse production, the organism is sensitive to conditions in the external environment, and if these are outside the normal range a permanent arrest of development occurs. In the previous paper I proposed that the amygdala, anterior cingulate, and insula limbic structures play a role in pre-attachment experiences that onset early in the first year, and thus trauma during each of their critical periods would interfere with the experience-dependent maturation of these limbic structures (see Figure 3).

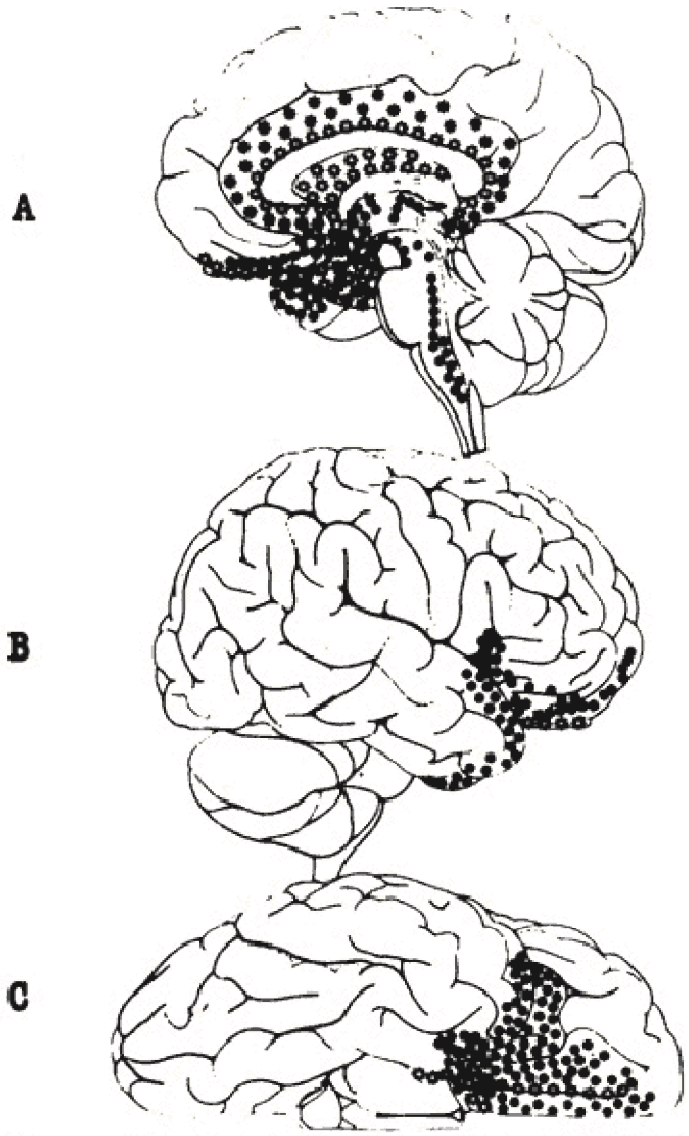

FIGURE 3. Limbic areas of the right brain (from Brothers, 1997). (Top) Medial view. Front of the brain at left. (Middle) Lateral view. Front of the brain is to the right. (Bottom) Ventral view. Front of the brain is to the right. Reprinted from Macchi, G. (1999). Anatomical substance of emotional reactions. In F. Boller & J. Grafman (Eds.), Handbook of Neuropsychology (vol. 3). New York: Elsevier. All possible efforts were made to contact the author by press time.

Indeed, neurobiological studies indicate that damage to the amygdala in early infancy is accompanied by profound changes in the formation of social bonds and emotionality (Bachevalier, 1994). These socioemotional effects are long-lasting and appear even to increase in magnitude over time (Malkova et al., 1997). Abnormalities of the social functions of the amygdala are implicated in autism (Baron-Cohen et al., (2000), and this would include autistic posttraumatic developmental disorder (Reid, 1999), a sub-group of children in which trauma in the first two years of life precipitates autism. Even more specifically to the model outlined here, abnormally large right (and not left) amygdala volumes have been reported in children and adolescents with generalized anxiety disorders (de Bellis et al., 2000a).

Relational traumatic events in the middle of the first year act as a growth inhibiting environment for the anterior cingulate limbic network. This would interfere with the ongoing development of the infantis coping systems, since impairments in anterior cingulate functions are known to lead to prolonged glucocorticoid and ACTH release during stress (Diorio, Viau, & Meaney, 1993) and later deficits in emotional arousal and an impoverished conscious experience of emotion (Lane et al., 1997). Indeed, maltreated children diagnosed with PTSD manifest metabolic abnormaliites of the anterior cingulate (de Bellis, Keshavan, Spencer, & Hall, 2000b). And early relational trauma which interferes with the experience-dependent maturation of the insula negatively impacts its role in generating an image of oneis physical state (body image), a process that underlies the experiencing of basic emotions (Craig et al., 2000).

But in addition, abuse and/or neglect over the first two years negatively impacts the major regulatory system in the human brain, the orbital prefrontolimbic system. In classic basic research, Kling and Steklis (1976) found that orbitofrontal lesions critically disrupt behaviors of "social bonding." More recently, Damasiois group reports that early neurological damage of this prefrontal cortex caused a failure "to acquire complex social knowledge during the regular developmental period" and an enduring impairment of social and moral behavior due to a "disruption of the systems that hold covert, emotionally related knowledge of social situations" (Anderson et al., 1999, p. 1035). Interestingly, a 20 year-old female patient, who at 15 months sustained ventromedial prefrontal damage due to a car accident, was unable to experience empathy, and "her maternal behavior was marked by dangerous insensitivity to (her) infantis needs" (p. 1032).

In these cases damage to the orbitofrontal system is of neurological causation. It should be pointed out that relational trauma may be accompanied by physical trauma to not only the body (Southall et al., 1997) but to the developing brain. In either case, the developmental trajectory of the brainis regulatory systems are negatively altered. In other words failures of structural development occur in relational trauma that includes or does not include physical trauma to the brain. In human infancy, purely "psychological" relational trauma leads to altered brain development, and purely "neurological" trauma negatively impacts relational development. Indeed, "type D" behaviors are found in neurologically impaired infants (Barnett et al., 1999), and infants who experience perinatal complications show orbitofrontal dysfunction in adolescence (Kinney et al., 2000). Sapolsky has pointed out that exposure to acute of chronic stress may be associated with either psychological disorders (such as child abuse) or neurological disorders (Moghaddam et al., 1994).

Earlier, I suggested that physical trauma to the babyis head and brain have a long-enduring negative impact on the trajectory of developmental processes. Infant/toddler abuse in the form of violent shaking of the head or forceful impact to the skull are sources of traumatic brain injury. The potential for such catastrophic outcomes of relational trauma may increase as the toddler becomes ambulatory in the second year, and account for the increase of "type D" attachments at this time. Although direct studies of such relationally induced brain injuries have not been done, information about the deleterious effects on brain function can be extrapolated from two sources, brain

magnetic resonance imaging (MRI) human studies of closed head injuries (Mamelak, 2000), and animal studies of traumatic brain injuries (McIntosh et al., 1989; Gennarelli, 1994). This research may give a model of the metabolic dysregulation occurring within the orbitofrontal areas of a physically traumatized infant/toddler.

Due to the topography of the brain as it sits within the bony skull, orbitofrontal contusions on the ventral surface are particularly common following impact-type closed head injury (Adams et al., 1980). Studies of the biomechanics of traumatic head injury in mature humans demonstrate that the sudden forceful movement of the brain within the skull causes inertial strain and tissue deformation that are greatest at the orbital surfaces of the frontal and temporal lobes, and that these areas are common sites of contusion (Mamelak, 2000). The ensuing anatomical and functional damage occurs whether or not there is impact on the skull or loss of consciousness. The response to orbitofrontal injury is a sudden increase in metabolic energy utilization followed by a prolonged period of metabolic energy depression.

Parallel studies in animal models demonstrate that the neurochemical consequences of closed-head injury occur even without signs of gross morphologic damage. This profile shows an initial significant rise in extracellular levels of excitatory amino acids, glutamate and aspartate, which trigger an initial hypermetabolic response. This in turn elevates intracellular calcium levels, which may last for days, and leads to a prolonged posttraumatic depression. This pattern of initial hypermetabolism followed by hypometabolism (Yoshino et al., 1991) is identical to that described by Perry. The later shut down of cerebral metabolism has been suggested to be due to the action of a sensor in the dorsal medullary region that functions as an energy conservation system which protects the brain against the detrimental consequences of energy depletion (Pazdernik, Cross, & Nelson, 1994).

It has recently been suggested that the symptoms of cerebral physical trauma take the form of a change in emotional functions, personality, and indeed psychiatric disorders, and can be explained as consequences of the impairment of specifically orbitofrontal functions (Mamelak, 2000). As mentioned earlier, Anderson et al. (1999) document the enduring psychosocial deficits that result from orbitofrontal damage (car accident and tumor resection) in the first and second year. These studies of orbitofrontal injury in infancy and adulthood suggest a similar pattern of impairments of energy metabolism in response to both intense physical and psychosocial stressors. The developing brain, which requires large amounts of energy during the brain growth spurt, reacts with massive bioenergetic alterations in response to traumatic assaults of brain and/or body. What would be the common outcome of either physical trauma or relational trauma-induced energy impairments during a critical period of energy-dependent growth of corticolimbic systems?

The postnatal organization of the brain and the progressive postnatal assembly of limbic-autonomic circuits (Rinaman et al., 2000) occurs in a very specific pattern. During a critical period of regional brain growth, genetic factors are expressed in an initial overproduction of synapses. This is followed by a process that is environmentally-driven, the pruning and maintenance of synaptic connections and the organization of functional circuits. This process of genetic-environmental organization of a brain region is energy dependent (Schore, 1994; 1997a; 2000c), and can be altered, especially during its critical period of growth. The construct of developmental instability (Moller & Swaddle, 1997) has been invoked to describe the imprecise expression of the genetic plan for development due to genetic (e.g., mutations) and environmental effects (e.g., toxins). I suggest that the psychotoxic contexts of early relational trauma acts as an inducer of developmental instability, which has been shown to contribute to alterations of cerebral lateralization (Yeo et al., 1997a) and to a vulnerability factor in the etiology of neurodevelopmental disorders (Yeo et al., 1997b).

In a very recent magnetic resonance spectroscopy (^{1}H-MRS) study of the right frontal lobe, Yeo et al. (2000) conclude that developmental instability

...may lead to a greater need for energy-requiring, stabilizing forces in development. Hence, there may be less in the way of metabolic resources left for metabolic growth (p. 155).

These very same conditions are produced in the patterns of initial hypermetabolism followed by enduring hypometabolism of relational trauma described earlier. This disruption of energy resources for the biosynthesis of right lateralized limbic connections would be expressed in a critical period developmental overpruning of the corticolimbic system, especially one that contains a genetically-encoded underproduction of synapses. This psychopathomorphogentic mechanism acts a generator of high risk conditions.

It is now accepted that "psychological" factors "prune" or "sculpt" neural networks in specifically the postnatal frontal, limbic, and temporal cortices (Carlson, Earls, & Todd, 1988). I propose that excessive pruning of cortical-subcortical limbic-autonomic circuits occurs in early histories of trauma and neglect, and that this severe growth impairment represents the mechanism of the genesis of a developmental structural defect. Since this defect is in limbic organization, the resulting functional deficit will specifically be in the individualis maturing stress coping systems. The dysregulating events of abuse and neglect create chaotic biochemical alterations in the infant brain, a condition that intensifies the normal process of apoptotic programmed cell death. Post and his colleagues report a study of infant mammals entitled, "Maternal deprivation induces cell death" (Zhang et al,. 1997). Maternal neglect is the behavioral manifestation of maternal deprivation, and this alone or in combination with paternal physical abuse is devastating to developing limbic subsystems.

In its critical period the orbitofrontal areas are synaptically connecting with other areas of the cerebral cortex, but they are also forging contacts with subcortical areas. And so the orbitofrontal cortex is a "convergence zone" where cortex and subcortex meet. In earlier writings I have proposed that it is the severe parcellation (excessive pruning) of hierarchical cortical-subcortical circuits that is central to the developmental origins of the regulatory deficits that are the sequelae of early trauma (Schore, 1997a). Caregiver-induced trauma exacerbates extensive destruction of synapses in this "Senior Executive" of limbic arousal (Joseph, 1996) which directly connects into the dopaminergic and noradrenergic systems in the anterior and caudal reticular formation. Exposure to fear cues provokes enhanced dopamine metabolism in the ventral tegmental areas (Deutsch et al., 1991) which activates the locus coeruleus (Deutsch, Goldstein, & Roth, 1986) and increases noradrenaline activty (Tanaka et al., 1990; Clarke et al., 1996). Both catecholamines are released in response to stressful disruptions of the attachment bond, and elevated levels of these bioamines result in regression of synapses and programmed cell death (McLaughlin et al., 1998; see Schore 1994 and 1997a for a description of dopaminergic disruptions of mitochonrial energy metabolism).

The mechanism of this parcellation, the activity-dependent winnowing of surplus circuitry, has been previously described in terms of hyperactivation of the dopamine-sensitive excitotoxic NMDA-sensitive glutamate receptor, a critical site of synapse neurotoxicity and elimination during early development. As opposed to this hypermetabolic response, cortisol release triggers hypometabolism, a condition that enhances the toxicity of excitatory neurotransmitters (Novelli , Reilly, Lysko, & Henneberry, 1988). During critical periods, dendritic spines, potential points of connection with other neurons, are particularly vulnerable to long pulses of glutamate (Segal, Korkotian, & Murphy, 2000) that trigger severely altered calcium metabolism and therefore "oxidative stress" and apoptotic damage (Park, Bateman, & Goldberg, 1996; Schore, 1997a). It is known that stress causes oxidative damage to brain lipid membranes, protein, and DNA (Liu et al., 1996), including mitochondrial DNA (Bowling et al., 1993; Schinder, Olson, & Montal, 1996; Schore, 1997a), that stress increases levels

of excitatory amino acids such as glutamate in the prefrontal cortex (Moghaddam, 1993), and that excitotoxins can destroy orbitofrontal neurons (Dias et al., 1996).

Developmental parcellation, experience-dependent circuit pruning, is associated with a selective loss of connections and redistribution of inputs via an elimination of long axon collaterals and dendritic processes, and such regressive events are essential mechanisms of brain maturation. But excessive parcellation during a critical period of synaptogenesis leads to a stress-induced shrinkage of dendritic fields, which are exquisitely vulnerable to oxidative damage (Schore, 1997a). Due to the massive bioenergetic alterations that accompany relational trauma, orbitofrontal dendritic fields are virulently over-pruned and extremely retracted, thus reducing potential sites of synaptic connectivity with distant cortical and subcortical inputs into this major convergence zone of the brain. Such smaller dendritic surface areas would lead to reduced current flow through orbitofrontal regulatory circuits.

In writings on brain plasticity and behavior Kolb and Whishaw (1998, p. 59) articulate the general principle:

[Individuals] with extensive dendritic growth...show facilitated performance on many types of behavioral measures...In contrast, individuals with atrophy in dendritic arborization show a decline in behavioral capacity. Similar factors that enhance dendritic growth...facilitate behavioral outcome, whereas factors that block dendritic growth (e.g., brain injury at birth) retard functional outcomes

Relational trauma not only blocks critical period dendritic growth, but also astrocyte proliferation that occurs in these intervals. These glial cells surround the most active regions of neurons, and thereby regulate the metabolic activity and connectional plasticity of all synapses in the brain (Laming et al., 2000). The postnatal proliferation and surface area of astrocytic processes that surround synapses is directly influenced by events in early the social environment (Jones & Greenough, 1996), including traumatic events.

Under conditions of continual traumatic assaults, more than dendritic fields may be lost, rather the neuronal components of one or both of the limbic circuits may undergo extensive programmed cell death. In describing the mechanism of neurotoxicity, Fornai et al. (1997, p. 402) state:

When acute insults are repeated, the neuronal loss progresses downstream to synaptically linked neurons. This trans-synaptic progression of neuronal death...resembles...neurodegenerative diseases in which the "systemic degeneration" consists of spreading cell loss to neurons interconnected in functional circuits.

An overly extensive developmental pruning of particularly, vertically-organized limbic circuits would result in an inefficent regulation of subcortical systems by cortical inputs. Excessive parcellation of the lateral orbitofrontal areas and the excitatory ventral tegmental forebrain-midbrain limbic circuit would severely alter the capacity to experience positive states, and underlie a vulnerability to hypoarousal, that is anhedonia and depression. On the other hand, a severe parcellation of the medial orbitofrontal areas and the inhibitory medial tegmental forebrain-midbrain circuit would result in a limited capacity to inhibit stressful hyperaroused states, such as terror and rage (Schore, 1996, 1997a).

It is important to point out that these disorganized insecure attachment psychopathologies are defined by an impairment of both limbic circuits. In less severe dysfunctions, such as those in the organized insecure attachments, only one circuit of the dual circuit limbic system is developmentally structurally immature and functionally inefficent (see Schore, 1994, 1996). But in these most severe attachment disturbances

the regulatory failures are manifest in the individualis limited capacity to modulate, either by autoregulation or interactive regulation, the intensity and duration of biologically primitive sympathetic-dominant affects like terror, rage, excitement, and elation, or parasympathetic-dominant affects like shame, disgust, and hopeless despair. Notice that intense positive affect, excitement and joy, is also a stressor to these personalities (Litz, Orsillo, Kaloupek, & Wathers, 2000).

This early-appearing adaptive dysfunction of internal reparative coping mechanisms endures in later developmental stages, and is most obvious under stressful and challenging conditions that call for behavioral flexibility. In this manner the coping deficits of maladaptive infant mental health endure as inefficient stress regulating deficts of maladaptive adult mental health.

Relational Trauma, Orbitofrontal Dysfunction, & a Predisposition to Posttraumatic Stress Disorders

In fact, this hierarchical apex of the limbic system manifests a "preferential vulnerability" to psychiatric disorders (Barbas, 1995). The limbic system is described as "the border zone where psychiatry meets neurology" (Mega & Cummings, 1994, p. 315). In updated psychiatric models, not severity of the trauma but characteristics of the individual, including his or her reactions to a trauma, are viewed as the essential factors that contribute to PTSD (American Psychiatric Association, 1994). An individualis repertoire of stress coping strategies is directly affected by the attachment relationship, and a disorganized/disoriented attachment interferes with this ontogenetic achievement. In this manner, there are direct connections between infant posttraumatic stress disorder and child, adolescent, and adult stress disorders. Indeed, traumatic chldhood events are commonly reported by adult PTSD patients with neurologic soft signs (Gurvitz et al., 2000).

Since the loss of the ability to regulate the intensity of affect is the most far-reaching effect of early trauma and neglect, this deficit involves a developmentally impaired inefficient orbitofrontal regulatory system. Current neurobiological research on PTSD reveals dysfunctional frontal-subcortical systems (Sutker, 1995; Uddo, 1993), and altered orbitofrontal (Bremner et al., 1997; Shin et al., 1999), anterior cingulate (Hamner, Lorberbaum, & George, 1999), and amygdala (Rauch et al., 1996) functions. So the next question is, how would a severe developmental pruning of the connections between the higher and lower levels of vertical limbic circuits be expressed in the functional deficits of PTSD?

Of special importance are the connections between the orbitofrontal areas and the hypothalamus, the head ganglion of the ANS and control system of visceral-somatic emotional reactions, and the amygdala, the major fear center in the brain. The right amygdala is known to process frightening faces and to mediate the "nonconscious processing" (Whalen et al., 1998) of "unseen fear" (Morris, Ohman, & Dolan, 1999), but when adequately functioning the right frontotemporal cortex exerts inhibitory control over intense emotional arousal (Kinsbourne and Bemporad, 1984). According to Rolls "although the amygdala is concerned with some of the same functions as the orbitofrontal cortex, and receives similar inputs, there is evidence that it may function less effectively in...very rapid learning" (1996, p. 1443). In optimal contexts the orbitofrontal cortex takes over amygdala functions.

The connections between the orbitofrontal areas and the amygdala form postnatally, and are negatively impacted by the adverse environmental events of relational trauma (see Figure 4). A severe experientially-driven pruning of these interconnections would allow for amygdala-driven states, such as fear-flight states to be later expressed without cortical inhibition. It is now established that a pathological response to stress reflects the functions of a hyper-excitable amygdala (Halgren, 1992) and that the memory processes

of the amygdala are amplified by extreme stress (Corodimas, et al., 1994). Even subliminally-processed low intensity interpersonal stressors could activate unmodulated terrifying and painful emotional experiences of the individualis early history that are imprinted into amygdalar-hypothalamic circuits. These fear-freeze responses would be intense, because they are totally unregulated by the orbitofrontal areas that are unavailable for the correction and adjustment of emotional responses.

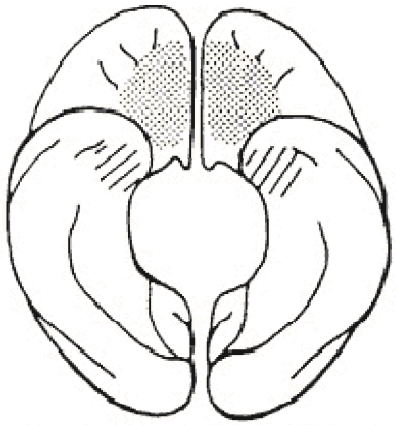

FIGURE 4. Undersurface of the brain. Front of the brain at top. Dotted area: the orbitofrontal cortex Hatched area: the amygdala (from Baron-Cohen, 1995, and used with permission of the MIT Press).

In optimal contexts, both the amygdala and the orbital prefrontal cortex have direct connections with the lateral hypothalamus (Kita & Oomura, 1981), an area known to activate parasympathetic responses through interconnections with the vagus nerve in the medulla (Brownstein, 1989). The anterior regions of the lateral hypothalamus are involved in "tonic immobility," defined as an inborn behavioral inhibition and terminal defense characterized by profound physical inactivity and lack of responsiveness to the environment that is triggered by fear generated during prey-predator confrontation (de Oliveira, Hoffmann, & Menescal-de-Oliveira, 1997). Notice the similarity of this to the immobility of the infantis conservation-withdrawal response, to dissociation, the escape when there is no escape, the detachment from an unbearable situation, and to Bowlbyis terminal separation response, "profound detachment."

The lateral hypothalamus develops postnatally (Fisher & Almli, 1984). Relational trauma in this period could result in weaker orbitofrontal and stronger amygdala connections into this structure, leading to amygdala-dominant behavioral inhibition. The higher corticolimbic areas would inefficiently regulate the immobility response, that is, there would be a tendency to dissociate under stress, and this response would be long-lasting. Morgan and Le Doux describe such a condition:

...while the amygdala determines the emotional significance of threatening stimuli, the ventromedial prefrontal cortex uses this information to monitor and give feedback about the internal state of the [organism] and to update response outputs dependent on this internal state. Without the internal feedback as to the level of threat posed by the stimulus at any given time, the [organism] might, for adaptive purposes, remain in the defensive response state longer than necessary (1995, p. 687).

In other work LeDoux suggests that a defective orbitofrontal system results in an inability to shift cognitive strategies and a reduction in behavioral flexibility, and that this "emotional perseveration" would lead to an increased resistance to extinction of fear behaviors, such as found in "anxiety, phobic, panic, and posttraumatic stress disorders." (Morgan, Romanski, & LeDoux, 1993, p. 112).

An inefficient orbitofrontal reparative function is expressed in a poor capacity for the state regulation that is necessary for self-comforting in times of stress. In such unstable

systems, small disruptions associated with interpersonal stresses too easily become rapidly amplified into intense distress states. This is subjectively experienced as a sudden transition into rapidly shifting negative affective states. A failure of orbitofrontal modulation of limbic arousal and an uncoupling of both the ventral tegmental and lateral tegmental forebrain-midbrain limbic circuits results in a cycling between intrusive hypersympathetically-driven terrifying flashbacks and traumatic images and parasympathetically-driven dissociation, avoidance, and numbing. Recent models of PTSD refer to stressor-induced oscillations between traumatic and avoidant states, and cycling between the bidirectional symptoms of emotional reexperiencing and emotional constrictedness (Antelman, 1997).

In very recent functional magnetic imaging (fMRI) research, Hariri, Bookheimer, & Mazziotta (2000) suggest an inability of the orbitofrontal areas, specifically in the right hemisphere, to modulate the amygdalais fear response to emotionally valent stimuli would underlie the emotional disturbances of posttraumatic stress disorder. These authors emphasize the adaptive importance of this network by which higher right frontal brain regions attenuate emotional stimuli mediated by more primitive brain regions. It is exactly this higher network that is rendered dysfunctional by early relational trauma and disorganized/disoriented attachments.

It is important to note that "type D" attachments are associated with another form of psychopathology, one of hostile-aggressive behavior (Lyons-Ruth et al., 1993). PTSD patients also show dysregulation of aggression, and so in addition to unmodulated hypothalamic hypersympathetic fear-freeze states, these individuals also manifest dysregulated hypothalamic hypersympathetic fight states. Basic neurobiological studies indicate that the orbitofrontal cortex exerts an inhibitory control over hypothalamic sites from which aggression can be elicited by electrical stimulation (Kruk, Van der Poel, & De Vos-Freichs, 1979), and that this cortex is implicated in the suppression of aggression in dyadic encounters (de Bruin, 1990).

A substantial body of neurological studies also indicate that aggression dysregulation is associated with specifically altered orbitofrontal function (Fornazzari et al., 1992; Grafman et al., 1996; Miller et al., 1997; Starkstein & Robinson, 1997; Raine et al., 1998a). Davidson, Putnam, and Larson (2000) implicate a dysregulation of an orbitofrontal-anterior cingulate-amygdala circuit of emotion regulation in a risk for violence and aggression. Right orbitofrontal impairment is associated with difficulties in emotional recognition of angry and disgusted facial expressions, autonomic responding, and social cognition, as well as with high levels of aggression (Blair & Cipolotti, 2000).

There is now evidence that there are two types of aggression, predatory or "stalking" attack and defensive or "affective" rage (Panksepp, 1998; Siegel et al., 1999). Positron emission tomography (PET) studies reveal that both predatory and affective murderers show reduced prefrontal and increased subcortical activity (Raine et al., 1998b). Increased metabolic rate in the right hemisphere is also seen in affective, impulsive muderers. Affective rage is mediated by the hypothalamic ventromedial nucleus, a structure associated with elevated sympatho-adrenal and cardiovascular activity (Stoddard-Apter, Levin, & Siegel, 1983), increases in anxiety (Adamec & McKay, 1993), and parasympathetic vagal suppression (Colpaert, 1975). This system is also involved in maternal rage that is part of maternal protectiveness, and so, in line with the finding that maternal aggression shares similarities with hypothalamic attack (Siegel et al., 1999), it is tempting to speculate that a functional dysregulation of this system occurs in maternal abuse.

Sympathetic ventromedial hypothalamic neurons continue to develop in a postnatal critical period (Almli & Fisher, 1985), a time when their dendrites receive frontolimbic axonal projections. These neurons also receive input from the amygdala (Adamec, 1998), and thus an excessive developmental parcellation of the orbitofrontal (and cingulate-insular) inhibitory pathway to the sympathetic ventromedial hypothalamic nucleus (Ohta

& Oomura, 1979) would seriously interfere with the ability of higher limbic inputs to regulate amygdala-driven affective rage. This deficit represents the outcome of early relational trauma, and it mediates the intermittent states of relationally triggered uncontrolled aggression seen in certain traumatized populations.

Furthermore, a large body of studies indicates disrupted early attachments and early trauma and abuse in the histories of children and adults diagnosed as borderline personality disorder (Lyons-Ruth & Jacobvitz, 1999), and thus there is a high correlation of PTSD and borderline diagnoses (Famularo et al., 1992; Herman, Perry, & van der Kolk, 1989; van der Kolk et al., 1994). Zanarini et al. (1997) report that 91% of borderline patients report childhood abuse, and 92% report some type of childhood neglect. In an overview of the literature Paris summarizes the developmetal data and asserts "the weight of the research evidence supports the hypothesis that abuse during childhood is an important risk factor for borderline personality disorder" (1995, p. 15). Herman and van der Kolk (1987) assert that PTSD and borderline personality disorders both share massive disturbances in affect regulation, impulse control, interpersonal difficulties, self-integration, and a bias to use dissociation when under stress. Neurobiological studies reveal altered amygdala (Carrigan, Davidson, & Heard, 2000) and orbitofrontal function (Goyer, Konicki, & Schulz, 1994) in borderline personality disorder.

Brain imaging research also demonstrates decreased orbitofrontal metabolism in another class of psychiatric patients with a history of violent behavior (Raine et al., 1998a), that is, sociopathic personality disorder. These authors implicate a defective orbitofrontal system in a "predisposition to violence", a finding of numerous studies (see previous references). These personalities show the second type of aggression, predatory or "stalking" attack (Panksepp, 1998; Siegel et al., 1999). This type of aggression is also associated with hypothalamic activity, but from different areas in the parasympathetic lateral hypothalamus, an area also innervated by amygdala inputs. In natural settings, predatory attacks are released when modulatory brain mechanisms, such as those in the amygdala and prefrontal cortex, are suppressed (Siegel et al., 1999). These authors report that stimulation of the medial prefrontal cortex blocks predatory attack elicited from the lateral hypothalamus.

The orbital prefrontal cortex has direct connections with lateral hypothalamus, and thereby regulates its activity (Kita & Oomura, 1981). Since this hypothalamic system also continues to develop in a postnatal critical period (Fisher & Almli, 1984), an excessive experience-dependent parcellation of prefrontal-lateral hypothalamic connections would result in an inefficient higher cortical regulation of not just the immobility response, but under extremely intense stressful levels, predatory attack. This predisposition to relational violence, which may result from a toxic growth-inhibiting combination of early maternal neglect and later paternal abuse, is expressed in "type D" attachments (Lyons-Ruth et al., 1993; Lyons-Ruth & Jacobvitz, 1999). This model clearly suggests that a sociopathic personality organization is frequently another manifestation of developmental posttraumatic stress disorder.

In an earlier work I presented ideas on the regulation of infantile rage reactions and on how structural impairments associated with attachment failures can be incorporated into models of primitive personality disorders (Schore, 1994). Continuing this, in an upcoming publication (Schore, work in progress) I offer data to show that "type D" disorganized/disoriented attachments and severe orbitofrontal pruning and apoptotic programmed cell death represent the developmental origins of both the affective aggression of various levels of borderline personality disorders and the predatory or stalking type of aggression of sociopathic personality disorders.

The Neurobiology of the Dissociative Defense

In severe attachment pathologies the developing infant/toddler is repeatedly exposed to the ambient cumulative trauma that emanates from an interactive dysregulating context with a misattuning caregiver. Since this growth-inhibiting context generates dense and prolonged levels of negative affect in the infant, for self-protective purposes it severely restricts itis overt expressions of an attachment need for dyadic regulation. The child thus significantly reduces the output of its emotion-processing, limbic-centered attachment system. And so for defensive functions it shifts from interactive regulatory modes into long-enduring, less complex autoregulatory modes. These subcortical-limbic organizational patterns are primitive strategies for survival, and therefore they become self-organizing attractor states. This sets the stage for primitive autoregulation, for the habitual use of dissociation. Indeed the type D attachment classification utilizes dissociative behaviors in later life (van Ijzendoorn et al., 1999).

The principle that severe attachment psychopathologies frequently access more primitive modes of autoregulation can be translated into the clinical tenet that more severe psychiatric disorders use dissociation as a characterological defense. In the parasympathetic-dominant state of dissociation, the individual is cut off (dis-associated) from both the external and the internal environment. This clearly implies a dysfunction of the orbitofrontal cortex, a site at which cortically processed exteroceptive information concerning the external environment is integrated with subcortically processed interoceptive information regarding the internal visceral environment.

The orbitofrontal system directly connects into the body via its direct connections into the ANS (Neafsey, 1990), and its modulation of the ANS is achieved via descending axons that synapse on dendritic fields of the hypothalamus, the head ganglion of the ANS, and vagal areas of the medulla. An extensive parcellation or thinning of these synaptic connections would lead to an inefficient regulation of the ANS by higher centers in the CNS. This loss means that under stress there would be not be a counterbalancing mechanism between the sympathetic-excitatory and parasympathetic-inhibitory components of the ANS, a loss of a coupled reciprocal mode of autonomic control, in which increases in activity in one ANS division are associated with a decrease in the other (Berntson, Cacioppo, and Quigley, 1991).

Under stress a developmentally immature orbitofrontal regulatory system would give way to a coupled nonreciprocal mode of autonomic control (Berntson et al., 1991). The result is an intensely high state of sympathetic ergotropic plus parasympathetic trophotropic arousal, the same pattern of Perryis infant trauma response. Although right vagal and sympathetic innervation of the heart elicits, respectively, decreased and increased cardiac activity, simultaneous stimulation produces an even greater cardiac output and aortic blood flow (Koizumi et al, 1982). Behaviorally this is like "riding the gas and the brake at the same time," and the simultaneous activation of hyperexcitation and hyperinhibition results in the "freeze response".

In classic neurological primate research, Ruch and Shenkin (1943) lesioned the orbitofrontal cortex (area 13) and observed a "definite reduction in emotional expression," and an elimination of "fear" and aggressive" behaviors that were replaced by "gazing into the distance with a blank expression." Such behavior was interpreted as an "over-reactive" response to the presence of the experimenter-observer. This is identical to the blank, dazed behavior of the "freeze" or "surrender" reaction of "type D" infants, to the "frozen watchfulness" observed in the abused child who waits warily for parental demands, responds quickly and compliantly, and then returns to her previous vigilant state, and to the "frozen state" of speechless terror seen in adult PTSD patients. Primate studies by Kalin et al. (1998) show that freezing in infants, which is elicited by eye contact, correlates with extreme right frontal EEG activity and high basal cortisol levels. This pattern, first measured in late infancy, endures for the rest of the lifespan as a fearful temperament. Extremely inhibited fearful children show heightened sympathetic activity as well as increased cortisol levels (Kagan, Reznick, & Snidman, 1987).

But in addition due to a loss of a counterbalancing functions of the ANS, severe attachment pathologies also show an inefficient orbitofrontal capacity in coordinating the two branches of the ANS and therefore in regulating affective shifts. Henry et al. (1992) point out that a simultaneous activation of the sympathetic-adrenal medullary and hypothalamic-pituitary adrenal axes typically occurs in the initial phases of overwhelming stress, but these systems can operate also independently. In PTSD, they note, these two systems can undergo an "increasing separation," and this "dissociation" is the basis for the emotional psychopathology of this disorder. This represents what Berntson et al. (1991) call an uncoupled nonreciprocal mode of autonomic control, in which responses in one division of the ANS occur in absence of change in the other.

A resultant rapid uncoupling of the two frontolimbic circuits would occur in response to even low levels of interactive stress, and be expressed in emotional lability and rapid state shifts. Putnam (1997) describes pathological "dissociative switches" between states, which occur rapidly, and are manifest in "Inexpliable shifts in affect", changes in facial appearance, mannerisms and speech, and discontinuities in train of thought. Recall, in trauma sympathetic hyperarousal is suddenly followed by hyperparasympathetic dissociation. Meares also concludes that "dissociation, at its first occurrence, is a consequence of a epsychological shocki or high arousal" (1999, p. 1853).

A habitual tendency to shift into primitive parasympathetic states is a characterstic of a developmentally immature regulatory system with weak connections between the highest level of the limbic system and the ANS. Of particular importance is the experience-dependent maturation of orbital areas that regulate the parasympathetic system, a development that is slower and later than the sympathetic (Schore, 1994). The orbitofrontal areas, like the amygdala, have direct inputs into the medulla (Mizuno, Sauerland, & Clemente, 1968; Yasui et al., 1991), including the medullary reticular formation (Travers, Dinardo, & Karimnamazi, 1997) and medullary noradrenergic neurons in the nucleus of the solitary tract (see Figure 2).

These are the sites of the medullary vagal system, but it is now known that there are two parasympathetic vagal systems, a late developing "mammalian" or "smart" system in the nucleus ambiguus which allows for the ability to communicate via facial expressions, vocalizations, and gestures via contingent social interactions, and a more primitive early developing "reptillian" or "vegetative" system in the dorsal motor nucleus of the vagus that acts to shutdown metabolic activity during immobilization, death feigning, and hiding behaviors (Porges, 1997). Both of these vagal systems are right laterlized (Porges, Doussard-Roosevelt, & Mati, 1994). The central nucleus of the amygdala has extensive connections into the dorsal motor vagal nucleus (Schwaber et al., 1982) and is involved in passive coping, immobile behavior, and parasympathetic activity (Roozendaal et al., 1997).

In an earlier discussion of traumatic brain injury I referred to this same dorsal medullary region that functions as an energy conservation system. I posit that in growth-facilitating socioemotional environments, the orbitofrontal system enhances its inputs into the nucleus ambiguus vagal system and therefore expands its affect regulatory capacities, but in traumatic growth inhibiting environments, this "smart" system never optimally develops, and the "vegetative" system dominates. I suggest that the longer enduring "developmental vegetative state" (Multi-Society Task Force on Persistent Vegetative State, 1994) of trauma-induced widespread hypometabolism would interfere with the growth of the developing brain, which requires massive amounts of energy for the biosynthetic processes of the brain growth spurt (Schore, 1994; Schore, 1997a; 2000c).

In neurological patients the vegetative state is characterized as a "complete loss of attention to the external world" (Laureys et al., 2000), a description that echoes the psychiatric concept of dissociation. I propose that the massive inhibition of the dorsal motor vegetative vagal system mediates dissociation, a primitive defensive mechanism which has long been implicated in trauma-induced psychopathogenesis (Janet, 1889; Chu

& Dill, 1990). Porges states that the dorsal motor nucleus of the vagus "contributes to severe emotional states and may be related to emotional states of eimmobilizationi such as extreme terror" (1997, p. 75). Perryis description of the traumatized infantis sudden state switch from sympathetic hyperarousal into parasympathetic dissociation is reflected in Porgesi characterization of

the sudden and rapid transition from an unsuccessful strategy of struggling requiring massive sympathetic activation to the metabolically conservative immobilized state mimicking death associated with the dorsal vagal complex (1997, p. 75).

Clinically, dissociation is described as "a submission and resignation to the inevitability of overwhelming, even psychically deadening danger" (Davies & Frawley, 1994, p. 65).

Dissociation is a primitive defense, and in early traumatized developmental psychopathologies more complex defenses never organize. The inhibitory vagal brake in such systems is predominantly provided by the rigid, fixed "vegetative" dorsal motor vagus, and not the more evolved and flexible "smart" nucleus ambiguus that allows for social communication. The vagal brake must be withdrawn when the individual shifts from a state of low to high metabolic demand, an operation that is adaptive for engaging and disengaging with the dynamically changing environment (Porges, 1997). This precludes involvement in dyadic play states and loss of a context for interactively creating high levels of arousal and metabolic energy for brain biosynthesis (see earlier section on developmental instability). The lack of the ability to engage in interactive play is an indicator of maladaptive infant health.

Dissociation and Body-Mind Psychopathology

Dissociation is a common symptom of a spectrum of severe psychopathologies, from reactive attachment disorder of infants (Hinshaw-Fuselier et al., 1999), to dissociative identity disorders (Putnam, 1989), psychotic experiences (Allen & Coyne, 1995), borderline personality disorders (Golynkina & Ryle, 1999), and posttraumatic stress disorders of adults (van der kolk, McFarlane, & Weisaeth, 1996). The DSM-IV lists five dissociative disorders: dissociative amnesia, dissociative fugue, depersonaliztion disorder, dissociative identity disorder, and dissociative disorder not otherwise identified (American Psychiatric Association, 1994).

Since dissociation appears in the earliest life stage, a developmental psychopathology perspective is being utilized to understand its etiology (Putnam, 1997), and disorganization of attachment is now proposed as a model system to understand dissociative psychopathology (Liotti, 1992, 1999). However, these models are purely psychological, and do not refer to the neurobiological mechanisms that underlie the phenomena. An integration of neuroscience and clinical data can offer such a model.

It is important to emphasize that in traumatic abuse the individual dissociates not only from the external world, from processing external stimuli associated with terror, but also from the internal world, that is painful stimuli originating within the body. It is sometimes difficult to keep in mind the fact that the body of an abused infant is physically assaulted, and therefore in pain. Darwin, in the work that began the scientific study of emotion, asserted that "Pain, if severe, soon induces extreme depression or prostration but it is first a stimulant and excites to action...Fear again is the most depressing of all emotions, and it soon induces utter helpless prostration" (1872, p. 31). Krystal, in a classic text on trauma, also describes the state switch from sympathetic hyperaroused-terror into parasympathetic hypoaroused conservation-withdrawal hopelessness and helplessness:

The switch from anxiety to the catatonoid response is the subjective evaluation of the impending danger as one that cannot be avoided or modified. With the perception of fatal helplessness in the face of destructive danger, one surrenders to it (1988, p. 114-115).

Using interdisciplinary data, Krystal further explains how the catatonoid reaction is the affective response to unavoidable danger, a pattern of surrender, and equates it with the "freeze" response and state of cataleptic immobility. "[I]n the state of surrender and catatonoid reaction, all pain is stilled and a soothing numbness ensues" (Krystal, 1988, p. 117). As previously described, this numbness is due to a sudden massive elevation of endogenous opioids in stress-induced catalepsy or immobility (Fanselow, 1986). A clinical description of the traumatized child state is offered by Nijenhuis et al.:

Individuals tend to hide in dark places, freeze there, and prefer to physically disappear when they feel threatened. Adopting a fetal position, they seem to be unresponsive to external stimuli (1998, p. 114-115).

Bodily stiffening frequently accompanies these incidents, and the passive defense of dissociation increases with the severity of abuse.

The long-term effect of infantile psychic trauma is the arrest of affect development and the process of desomatization (Krystal, 1997). The ultimate endpoint of experiencing catastrophic states of relational-induced trauma in early life is a progressive impairment of the ability to adjust, take defensive action, or act on oneis own behalf, and, most importantly, a blocking of the capacity to register affect and pain. Lane et al. assert that "traumatic stress in childhood could lead to self-modulation of painful affect by directing attention away from internal emotional states" (1997, p. 840), a principle consonant with the well-documented association between traumatic childhood events and proneness to dissociation (Irwin, 1994; Ogawa et al., 1997).

In an earlier section of this paper I offered a psychoneurobiological model of the developmental events that lead to such maladaptive coping strategies. The pattern of cataleptic immobility described by Krystal is normally seen in the first two months of life of human infants: "[In] dangerous situations a sudden behavioral change in the infant may occur...the infant lies motionless with non-converging, staring eyes and sleep-like respiration" (Papousek & Papousek, 1975, p. 251). The right lateralized dorsal motor "vegetative vagus" is involved with respiration (Porges et al., 1994). Recall traumatized infants are observed to be staring off into space with a glazed look, and the childis dissociation and immobility in the midst of terror result from elevated levels of cortisol and vagal tone, while opiates induce pain numbing and blunting. The state of conservation-withdrawal occurs in hopeless and helpless contexts, and is behaviorally manifest as feigning death (Powles, 1992). Krystal (1988) notes that in German catalepsy is called Totstell-reflex, or "death-posturereflex."

The purpose of this primitive defensive reaction is to protect the developing organism against the overwhelming psychobiological pain of the attachment disruptions induced by early relational trauma. MacLean points out that "nature appears to have ensured that maternal-offspring separation in mammals results in distress comparable to pain" (1987, p. 136). This implies that maternal regulation of pain-distress occurs as a normal attachment function. But what if the caregiver is a source of intense noxious and painful stimulation? There is now an increasing consensus that both the childis reactivity and the parenting context contribute to changes in the infantis pain response (Sweet, McGrath, & Symons, 1999). According to Grunau et al:

Non-optimal parenting may contribute to the development of inappropriate strategies for coping with common pains in childhood, or of chronic pain patterns, in some children who have experienced prolonged or repeated pain as neonates (1994, p. 353).

Since these are attachment experiences occurring in a critical period of limbic ontogeny, they alter the organization of the brain circuits that process pain. What do we know of these circuits?

Recent basic research reveals that persistent pain experiences during the early neonatal period, a critical period for the organization of nociceptive neuronal circuits, rewires immature pain circuits, and leads to lasting and potentially detrimental alterations in the individualis response to pain in adulthood (Ruda et al., 2000). These studies track the long term effects of physical pain, but the effects of pain associated with relational trauma may lead to even more adverse consequences. It is now established that a traumatic painful event, in contrast to nontraumatic pain, triggers an intense emotional experience with concomitant autonomic / somatic outflow, and activates increased sympathetic activity and prominent responses in the limbic system, i.e., hypothalamus, periaqueductal gray, anterior cingulate, insula, posterior parietal and prefrontal cortex (Hsieh et al., 1995a; Hutchison, Harfa, & Dostrovsky, 1996). Positron emission tomography (PET) studies show that the right anterior cingulate plays a central role in the sensorial / affective aspect of pain (Hsieh et al., 1995b; Price, 2000) and that the orbitofrontal regions modulate distant processing of pain and therefore coping with a painful stimulus (Petrovic et al., 2000).

Neurobiological studies indicate that through its hierarchical connections with the pain processing areas in the periaqueductal gray, hypothalamus, anterior cingulate, and insula, the orbitofrontal areas are involved in both the perception (Zhang et al., 1997) and the regulation (Gyulai et al., 1997) and therefore coping with pain, especially the affective-motivational aspects of pain (Petrovic et al., 2000). The latter authors conclude that increased orbitofrontal activation is necessary for coping "during pain with a relevant threat to the organism" (p. 28). Such a context occurs in relational traumatic abuse.

An efficient mature orbitofrontal system can adaptively regulate both sympathoadrenomedullary catecholamine (Euler & Folkow, 1958) and corticosteroid levels (Hall & Marr, 1975), and therefore hyper- and hypoarousal. It can also facilitate or inhibit the defense reactions of the amygdala (Timms, 1977). But stress may also take the prefrontal areas "off-line", allowing the "more habitual" responses mediated by the subcortical structures to regulate behavior (Arnsten, & Goldman-Rakic, 1998). This occurs all-to-frequently in a severely developmentally compromised immature frontolimbic system, especially one with an inefficient medial orbitofrontal area involved in processing and regulating negative emotional states (Northoff et al., 2000).

When optimally functioning, the orbitofrontal cortex is "one of the few brain regions that is "privy to signals about virtually any activity taking place in our beingsi mind or body at any given time" (Damasio, 1994, p. 181). This implies that an inefficient frontolimbic system will not process pain signals that come from the body, an adaptive loss. Indeed, inactivation of the medial orbitofrontal cortex produces an analgesic effect (Cooper, 1975), and its removal elicits a suppression of pain-related behaviors and an increased threshold of pain associated with affect (Reshetniak & Kukushkin, 1989). Patients with neurological damage in this cortex report that they know a stimulus is pain-producing, but that the pain does not feel very bad (Melzack & Wall, 1996).

These studies suggest that an inefficient orbitofrontal-cingulate higher limbic circuit (Dostrovsky et al., 1995) would be unable to adaptively sense and regulate pain, and a lower amygdala limbic level-driven dissociation would dominate. Cutting, a common form of self-destructive behavior associated with early trauma (van der Kolk et al., 1991; Russ et al., 1992), may be an attempt to autoregulate out of the altered pain sensitivity associated with the elevated opioid activity of the dissociative state.

Dissociation is a common symptom in PTSD patients, and its occurrence at the time of a trauma is a strong predictor of this disorder (Koopman, Classen, & Spiegel, 1994; Shalev et al., 1996). At the moment of feeling threatened, individuals who characterlogically dissociate switch into a trance-like state, freeze, become analgesic, and later report out-of-body experiences and dissociative amnesia. Total amnesia for traumatic events is now increasingly documented (van der Hart & Nijjenhuis, 1995; Elliott, 1997).

In very recent work Markowitsch et al. (2000) report a case of "dissociative amnesia" triggered by re-exposure to a traumatic scene, a fire in the patientis house. For the next two months he exhibited a severe memory impairment, barely recognized his partner, and failed to remember any friends. He showed flat affect and lack of interest, and his mood was sad and helpless. After 3 weeks of psychotherapy he recalled an early memory of childhood, a car crash in which he witnessed the driveris screams and death in flames. PET studies at 2 months after the trauma showed an "unusually drastic" hypometabolism in memory-sensitive regions, which improved upon recovery at 12 months. The authors conclude that early emotionally negative childhood events and prolonged stress lead to a dissociative (functional) amnesia, that acute stress can trigger posttraumatic stress disorder, and that "even sporadic environmental stress can apparently induce long-lasting brain dysfunction with subsequent cognitive deterioration" (p. 65).

In considering possible factors for generating the hypometabolic state these authors point to the memory-influencing role of dopamine under stress conditions and alterations within the hypothalamo-hypophysal-adrenocortical axis, specifically excessive release of glucocorticoids. I suggest the finding of these researchers that psychic trauma can grossly reduce brain metabolism and thereby cognitive deterioration describes the hypometabolic mechanism of vagal-induced conservation-withdrawal and the mechanism of dissociation in response to trauma.

The characterological use of dissociation by certain personalities underlies the description offered by Allen and Coyne:

Although initially they may have used dissociation to cope with traumatic events, they subsequently dissociate to defend against a broad range of daily stressors, including their own posttraumatic symptoms, pervasively undermining the continuity of their experience (1995, p. 620).

These "initial traumatic events" are embeded in infant relational trauma, the first context in which dissociation is used to autoregulate massive stress.

Dissociation represents a disruption of the monitoring and controlling functions of consciousness. Fonagy et al. describe:

...victims of childhood abuse who coped by refusing to conceive of the contents of their caregiveris mind and thus successfully avoided having to think about their caregiveris wish to harm them. This initially defensive disruption of the capacity to depict feelings and thoughts in themselves and others becomes a characteristic response to all subsequent intimate relationships. It also drastically limits their capacity to come to terms with these abusive experiences in later life and creates a vulnerability to interpersonal stress (1996, p. 384).

Furthermore,

Trauma victims who lack the cognitive and emotional structures to immediately assimilate the experience use the state of consciousness known as dissociation to escape from the full psychological impact of the event (Classen, Koopman, & Spiegel, 1993, p. 179).

It should be pointed out that dissociation may be a more common phenomenon of the psychopathology of everyday life than previously thought. On the Adult Attachment Interview a classification of unresolved (the adult disoriented/disorganized analog) is made when an individualis narrative of his or her early experiences shows lapses in monitioring, prolonged silences of 20 seconds, and "micro-dissociative processes" (Schuengel et al., 1999). Under a series of dyadic emotional stressors these

characterolgical microstates, however, become stabilized attractor macrostates of dissociation.

Such clinical descriptions describe impaired activity of the orbitofrontal system, which acts in the highest level of control of behavior, especially in relation to emotion (Price, Carmichael, & Drevets, 1996), plays a fundamental role in monitoring relevant past and current experiences (Cavada et al., 2000) and in controlling the allocation of attention to possible contents of consciousness (Goldenberg et al., 1989), and allows for choosing appropriate actions in a flexible and purposeful manner in stressful contexts of uncertainty (Elliott, Dolan, & Frith, 2000). These and the above data clearly suggest that a developmentally immature and metabolically inefficient orbitofrontal regulatory system is found in immature personalities who characterolgically use dissociation.

In 1893 Breuer and Freud, citing the recent work of Janet (1889), described dissociation as the major mechanism for "strangulations of affect," but by 1900 and The Interpretation of Dreams Freud discarded this notion and favored repression as the major force of the unconscious. In later writings Freud again hinted of its existence in asserting:

Unconscious ideas continue to exist after repression as actual structures in the system Ucs, whereas all that corresponds in that system to unconscious affects is a potential beginning which is prevented from developing (Freud, 1915, p. 178).

With an eye to Freud's ideas on the negative effects of early truama, Winnicott postulated:

If maternal care is not good enough, then the infant does not really come into existence, since there is no continuity in being; instead, the personality becomes built on the basis of reactions to environmental impingement (1960, p. 54).

Tustin (1981) described this impingement as a "psychological catastrophe," which is responded to by "autistic withdrawal" or "encapsulation," an innate defensive measure against bodily hurt that involves a "shutting out of mind" what can not be handled at the moment. This is an operational definition of the growth inhibiting defense of dissociation, the generator of unconscious affects and the block against potential affective development and the ongoing continuity of existence.

What is maladaptive about this psychic-deadening defense is not only that the individual shifts into dissociation at lower levels of stress, but that it finds difficulty in exiting the state of conservation-withdrawal. Once dissociated it stays in this massive autoregulatory mode for long periods of time. During these intervals it is shut-down to the external environment, and thus totally closed and impermeable (encapsulated) to attachment communications and interactive regulation. If this becomes a basal state, the avoidance of emotional contexts, especially those containing novel and more complex affective information, prevents emotional learning, which in turn precludes any advances of right brain emotional intelligence or what Janet (1889) calls an "enlargement" of personality development. The habitual use of this primitive defense against affect is thus another manifestation of maladaptive infant (and adult) mental health.

Early Relational Trauma and Enduring Right Hemispheric Dysfunction

The orbitofrontal system, which is expanded in the right hemisphere (Falk et al., 1990), acts as an executive control function for the entire right brain. The right prefrontal cortex is critical to the processing and regulation of self functions (Schore, 1994; Keenan, Wheeler, Gallup, & Pascual-Leone, 2000). During its critical period of maturation in the first two years, prolonged episodes of intense and unregulated interactive traumatic stress induce not only heightened negative affect, but chaotic biochemical alterations that produce a developmentally immature, structurally defective right brain. Although

very few neuropsychobiological studies on traumatized human infants have yet been done, basic research on trauma in infant mammals and adult humans strongly implicates dysfunction in the right hemisphere, the hemisphere that is dominant in human infancy (Chiron et al., 1997).

And yet compelling theoretical, research, and clinical links have been made between right hemisphere functions and attachment behaviors (Henry, 1993; Schore, 1994), attachment transactions and the regulation of the right brain (Schore, 2000b, c), traumatic stress, attachment and right brain function (Wang, 1997), and the role of impaired right hemispheric activity in very early-forming reactive attachment disorders (Hinshaw-Fuselier, Boris, & Zeanah, 1999), personality disorders (Horton, 1985) and various psychiatric syndromes (Cutting, 1992; Cummings, 1997). The development of attachment, the interactive regulation of biological synchronicity between organisms (Schore, 2000b), allows for the development of emotions, the highest order direct expression of bioregulation in complex organisms (Damasio, 1998).

At the beginning of this two-paper series I cited Damasiois (1994) description of the fundamental adaptive function of "the brain" - to be be well informed about its own activities, the rest of the body and the environment so that suitable survivable accommodations can be achieved between the organism and the environment. Although the existence of not one but two brains, a left brain and a right brain, was discovered at the dawn of neurology (Harrington, 1985), current neuroscience, armed with neuroimaging technologies, is now detailing the unique functions of the right brain and its critical roles. These systems that contribute to the forementioned adaptive functions are maturing before the advent of language, and are influenced by the attachment relationship.

The right hemisphere, more so than the left, is deeply connected into the limbic system and the sympathetic and parasympathetic components of the ANS, and therefore it plays a predominant role in the physiological and cognitive components of emotional processing (Spence et al., 1996). This "nondominant" (!) hemisphere is specialized for neuroendocrine and autonomic activation (Sullivan & Gratton, 1999), for the secretion of the stress hormones, CRF (Kalogeras et al., 1996) and cortisol (Wittling & Pfluger, 1990), for the human stress response (Wittling, 1997), and for controlling the vital functions supporting survival and enabling the organism to cope with stresses and challenges (Wittling & Schweiger, 1993). Severe developmental impairments of these right brain structure-function relationships are manifest in inefficient and vulnerable coping mechanisms, and they occur in the attachment pathology of disorganized infant and toddlers. I would amend Mainis (1996) assertion that "disorganized" attachment is a primary risk factor for the development of mental disorders to specifically posttraumatic stress, borderline, and sociopathic personality disorders.

Throughout these two papers I have offered interdisciplinary data indicating a developmental right brain etiology of these severe regulatory disorders. Continuing this theme, a juxtaposition of trauma-associated functional deficits in coping strategies and stress tolerance of "type D" infants found in attachment research, right lateralized structural defects from developmental psychobiological studies, and findings on the right brain of normal and abnormal adults from neuroscience can offer more powerful models of the mechanisms by which early relational trauma in the first two years alters the experience-dependent maturation of the right brain and thereby induces a high risk for psychopathogenesis.

Evidence of this lateralization effect is provided by human studies showing that conditioned fear acquisition and extinction are associated with amygdala function, and that this activation is right hemisphere dominant (La Bar, Gore, LeDoux, & Phelps, 1998). In a series of basic psychobological studies Adamec reports that partial kindling of the right and not left amygdala induces long-lasting increases in anxiety-like behavior (1997, 1999), that NMDA receptors mediate transmission from the right amygdala to the

ventromedial hypothalamus (Adamec, 1998), and that right amygdala kindling also induces elevated production of the stress hormone CRF (Adamec & McKay, 1993). In parallel research, "type D" infants show "direct indices of apprehension regarding the parent," as manifest in fearful facial expressions (Solomon & George, 1999). These authors document that such infants show asymmetries of facial expression, as in "an extremely swift etici which lifts only the left side of the facial musculature," indicating right hemispheric dysfunction. They also describe freezing lasting 20 seconds or more, accompanied by dazed or trance-like facial expressions in "type D" infants. Recall freezing in primate infants is associated with high basal cortisol levels and extreme right frontal EEG activity (Kalin et al., 1998).

In fact, EEG studies of one-month-old (Jones et al., 1997) and 3-to 6-month-old (Field, Fox, Pickens, & Nawrocki, 1995) infants of depressed (and therefore potentially neglectful) mothers show this same right frontal EEG asymmetry, a finding that has been interpreted as reflecting a subcortical asymmetry in the amygdala (Calkins & Fox, 1994). At 10 months, infants who express more intense distress to maternal separation display a greater right than left frontal activation (Davidson & Fox, 1989), and this asymmetry is related to emotional reactivity and vulnerability to psychopathology in both infants and adults (Davidson et al., 1990). At 3 to 6 years, children of depressed mothers show a right frontal EEG asymmetry and lack of empathy (Jones, Field, & Davalos, 2000).

Individuals with extreme right frontal activation are thought to exhibit a negative affective response to a very low intensity negative affect elicitor, and to be impaired in the ability to terminate a negative emotion once it has begun (Wheeler, Davidson, & Tomarken, 1993). Fox et al. (1996) report that young children with internalizing and externalizing problems show greater right than left frontal EEG activation, and suggest that this pattern reflects difficulties with affect regulation, whether the affect arousal is extremely negative or positive. At later ages "greater right hemisphericity" is associated with a history of more frequent negative affect and lower self esteem (Persinger & Makarec, 1991), that is, chronic difficulties in affect regulation.

With respect to the association of type D attachments and a predisposition to relational violence (Lyons-Ruth & Jacobvitz, 1999), impaired right hemispheric functioning has also been reported in disinhibited aggressive patients with orbitofrontal brain damage (Starkstein & Robinson, 1997), autonomic physiological studies of high-hostility subjects (Demaree & Harrison, 1997), and neuroimaging research of murderers, where Raine et al. conclude, "reductions in right orbitofrontal functioning may be a particularly important predisposition to violence" (1998a, p. 6).

The right brain circuitry that is involved in the regulation of "primary" emotions (Ross, Homan, & Buck, 1994) and in "intense emotional-homeostatic processes" (Porges et al., 1994) is organized in the first two years of life. Exposure to extensive and long-enduring traumatic states interferes with this organization and predisposes the disorganized/disoriented infant (later, the unresolved/disorganized adult) to a vulnerability, at later points of stress, to develop chronic difficulties in affect regulation. Due to its unique anatomical connections into the reticular formation, the right hemisphere is dominant for the bilateral regulation of arousal (Heilman & Van Den Abell, 1979) and its dysfunction is therefore central to the arousal dysregulation that characterizes the severe coping deficits of fear dysregulation of posttraumatic stress disorders. The right cortex is responsible for maintaining important controls over autonomic activities (Heilman et al., 1977) and for generating the most comprehensive and integrated map of the body state available to the brain (Damasio, 1994). An impairment of the right brain is thus central to the disordered mind-body functions that are found in children and adults who continue to experience the relational trauma of their infancy.

Indeed, van der Kolk (1996) specifically implicates right brain dysfunction in posttraumatic stress disorders, and this lateralization effect is observed in studies that

expose the patient to a personal high arousal stressor (Rauch et al., 1996; Shin et al., 1997) and those that induce a stressful startle response (Morgan et al., 1997). Patients with panic disorders show greater activation of a right frontal avoidance-withdrawal system in negatively valenced situations (Wiedemann et al., 1999) and altered GABAergic receptor patterns in the right insula and orbitofrontal cortices (Malizia et al., 1998).

Research may also tell us more about the triggers of right hemispheric dysregulation. It is now thought that traumatic early life events predispose certain individuals to later psychiatric disturbance when they are "rechallenged" with a "matching event" or recurrence of the stressor. Previously I spoke of the right brain imprinting into procedural memory of the abusive caregiveris threatening face. Current evidence shows that the right amygdala (Morris et al., 1999) is involved in the storage of fearful faces (vs. the left in linguistic threat; Isenberg et al., 1999). The right amygdala is also implicated in the expression of emotionally influenced memory of aversive experiences (Coleman-Mensches & McGaugh, 1995). Similarly, the right orbitofrontal cortex shows an enhanced response to anger expressions that correlate with expression intensity (Blair et al., 1999).

The "visuospatial" right hemisphere contains a "nonverbal affect lexicon" of facial expressions (Bowers, Bauer, & Heilman, 1993), and these are imprinted in mother-infant affective transactions. This hemisphere appraises facial expression at levels beneath awareness (Schore, 1994; 1998b, 1999a; in press a, b), and if a match is registered with a stored image an affective response occurs. Autonomic changes in the body are evoked when angry facial expressions are subliminally presented to the right, and not the left hemisphere (Johnsen & Hugdahl, 1991). Right hemispheric impairments in processing facial (Deldin et al., 2000) and vocal affective-prosodic (Snow, 2000) interpersonal stimuli have profound consequences for interpersonal behavior, isolating the individual from the social environment. A growing literature demonstrates that neglected children have difficulty in recognizing emotion in faces, and that physically abused children dis[play a response bias for angry facial expressions (Pollak, Cicchetti, Hornung, & Reed, 2000).

I suggest that visual and auditory stressors that are nonconsciously processed (Mogg, Bradley, Williams, & Mathews, 1993) in an inefficient right hemisphere, especially the perception or memory of images and sounds of threatening and humiliating faces are potent triggers of dysregulation and dissociation in early traumatized patients. In support of this, Main (1995) reports the dissociative responses of disorganized/disoriented children to the faces of a family photograph:

One child, happily interacting with the examiner just previously, bent silently over the photograph for 12 seconds, then looked up, silent and depressed. Another looked into the photograph for some time then murmured softly, "Where are you, Mama? For children who had been disorganized/disoriented with mother in infancy, then, the visual presentation of the parent, self, or family presented within the photograph seemed to have an overwhelming and absorbing quality that drew attention away from the immediate situation (p. 435).

Furthermore, the infantis transactions with an emotionally misattuned and unresponsive caregiver who induces traumatic states and provides poor interactive repair are stored in the infantis developing corticolimbic circuitries as imagistic, visceral, and nonverbal implicit-procedural memories. Interpersonal contexts of interactive repair contain facially expressed safety signals that can be associated with "switching off" the traumatized state, and so the lack of such internal representations deprives the traumatized individual of an internal regulatory mechanism that can terminate the traumatic reaction.

Kiersky and Beebe (1994) state that nonverbal presymbolic forms of relating constitute adult versions of the early interaction structures that protected the infant from trauma and continue to be used by patients to avoid retraumatization. Fonagy (1991) asserts that the mental representations of early traumatic interactions with an abusive parent lead the child to defensively disregard perceptions of the thoughts and feelings of the parent. These unconscious working models of disorganized-disoriented attachment encode an enduring prototypical cognitive-affective schema of a dysregulated-self-in-interaction-with-a-misattuning-other (Schore, 1994, 1997b, c, in press b).

Such "pathological" representations are accessed when the individual is stressed, and they are stored in the right hemisphere which is dominant for unconscious processes (Schore, 1994; 1997b, 1999; in press a) and for episodic and autobiographical memory (Fink et al., 1996). Early abusive memories are recorded in the right hemisphere outside of conscious awareness, and this realm represents the traumatic memories in imagistic form along with the survival behavior employed as a result of the abuse. The cortical hemispheres contain two different types of representational processes and separate, dissociable memory systems (Zaidel, Esiri, & Beardsworth, 1998), and this allows for the fact that early emotional learning of the right, especially of stressful, threatening experiences, can be unknown to the left (Joseph, 1982). In clinical psychoanalytic writings Bromberg describes:

Dissociated experience thus tends to remain unsymbolized by thought and language, exists as a separate reality outside of self-expression, and is cut off from authentic human relatedness and deadened to full participation in the life of the rest of the personality (1991, p. 405).

A limited representational capacity is thus another deficit derived from early relational trauma. According to Reid,

Where trauma has occurred in infancy, before there is adequate differentiation of self from other, and before the development of the capacity to symbolize, the child cannot withdraw into daydreaming and fantasy, which has been noted in adults and children suffering from post-traumatuc stress disorders (1999, pp. 99-100).

In a similar description, Meares (1993) demonstrates that in cases of early abuse, the older childis capacity for positively charged symbolic play is not adequately established. Slade reports that insecurely attached children have fewer episodes, shorter periods, and less complex symbolic play (1994), and emphasizes the links between play, the consolidation of affect, meaning, and representation (1987). These essential capacities to generate and maintain postively charged autoregulatory representations heavily depend upon efficient right hemispheric activity.

Another right hemispheric cognitive activity may be detrimentally affected by relational trauma during its initial period of maturation. In light of the known involvement of the right hemisphere in attention (Tucker & Derryberry, 1994; Posner & Peterson, 1990; Coule et al., 1996; Sturm et al., 1999) and joint attention (Kingstone, Friesen, & Gazzaniga, 2000), it is tempting to speculate that joint attention experiences in the first year tune the attentional mechanisms of these right laterlized circuits. Unmedicated children with attention-deficit hyperactivity disorder (ADHD) show a disruption of right hemispheric attentional systems, due to a difficulty sustaining attention over short time intervals and a failure of inhibition (Carter et al., 1995; Castellanos et al., 1996; Epstein et al., 1997; Pliszka, Liotti, & Woldorff, 2000). Elevated dopamine levels in right midbrain areas that control attention are correlated with symptom severity (Ernst et al., 1999).

Very recent evidence suggests that developmental dyslexia is a left hemispheric dysfunction, while developmental hyperactivity is a right hemispheric dysfunction (Braun

et al., 2000). A number of researchers have been describing this latter clinical entity. In early work Weintraub and Mesulam documented right hemispheric learning disability as:

a syndrome of early right hemisphere dysfunction...that is associated with introversion, poor social perception, chronic emotional difficulties, inability to display affect, and impairments in visuospatial representation (1983, p. 468).

In more recent work, this group differentiates children with left hemisphere dyslexia from right hemisphere social emotional processing disorder, the latter displaying difficulties in interpreting and producing nonverbal aspects of communication including prosody, facial expression, and gesture, as well as poor emotional adjustment and psychiatric disorder (Manoach et al., 1997). Similar descriptions are seen in developmental right hemisphere syndrome, expressed in emotional and interpersonal problems and avoidance of eye contact (Gross-Tsur et al., 1995), right hemispheric learning disability, showing episodic dyscontrol and psychiatric disorders (Grace & Malloy, 1992), and nonverbal learning disability, who in adolescence are high risk for depression and suicide (Rourke, Young, & Leenars, 1989). Recent neuroimaging research indicates that metabolic rate of the right amygdala correlates with negative affect in depressed patients (Abercrombie et al., 1998). I suggest that these more severely disturbed right brain learning disabilities are "type D" attachments.

One other defining clinical feature occurs in individuals who are high risk for later developing pathological traumatic reactions. Relational trauma in the second year would induce a severe pruning of the right hemispheric orbitofrontal callosal axons that are growing towards their counterparts in the left hemisphere. This would produce an interhemispheric organization in which facial expressions, bodily states, and affective information implicitly processed in the right brain would be inefficiently transmitted to the left hemisphere for semantic processing. Maltreated toddlers show a dramatic inability to talk about their emotions and internal states (Cicchetti, Ganiban, & Barnett,1991).

This represents the early expression of alexithymia, "no words for feelings," a common symptom of trauma patients (Taylor et al., 1997, 1999). Neuropsychological studies of alexithymia now demonstrate a right hemispheric dysfunction and a specific right to left deficit of callosal transfer (Dewaraja & Sasaki, 1990). A physiological disconnection of the two hemispheres results in an inability of the affective and symbolic energies of the right hemisphere to be externalized through the verbal expression of the left hemisphere. A hyporesponsivity in the prefrontal and orbital circuits has been suggested to underlie alexithymia (Hommer et al., 1997).

Indeed both alexithymia and PTSD share similar altered neuroendocrine patterns (Henry et al., 1992), and the extensive overlap between the two has been emphasized (Taylor et al., 1997). Alexithymic personalities manifest a deficit in the capacity for symbolization of emotions, a tendency for impulsive behavior, avoidance of social relationships, abnormal physiology resulting in disease, and an impaired capacity for self-care and self-regulation. Miller (1986, p. 138) points out that the noninsightful constricted mental state of the alexithymic resembles "the retraction of the field of consciousness" discussed by Janet (1924), as well as the dissociative reactions described in hysterical patients by Breuer and Freud (1893). Right hemisphere invovlement in hysterical paresthesia (Tiihonen et al., 1995) and somatization (Min & Lee, 1997) is now reported.

Alexithymic individuals become disorganized under stress, and the regulatory disturbance is manifest in dramatic outbursts of emotion that end as quickly as they begin as though a valve is turning on and shutting off (Nemiah & Sifneos, 1970), affective blocking in the face of unbearably intense pain during overwhelming experiences (Krystal, 1988), and deficits in spontaneous nonverbal expressions of negative affect (McDonald & Prkachin,1990). Alexithymia is thus fundamentally an impairment in emotional information processing (Lane et al., 1997), specifically a deficit in the cognitive

processing and regulation of emotions (Taylor, 2000), and is manifest in posttraumatic stress disorder, borderline personality disorders, substance abuse disorders, and somatoform disorders (Taylor et al., 1997). Developmental traumatic stress and neurobiological deficits in the anterior cingulate and orbitofrontal cortices (Lane et al., 1997) and in the right hemisphere (Taylor et al., 1997) have been implicated in alexithymic symptomatology. In discussing the etiology of alexithyma Rotenberg concludes:

The functional deficiency of the right hemisphere...may be caused by the lack of emotional relationships between the child and the parents. Such emotional relationships...stimulate the development of the right hemisphere functions and correspond to these functions as a key to the lock. If these emotional relationships are insufficient, the right hemisphere will become inefficient, its contribution in psychological defense mechanisms and emotional stabilization will be lost, and there will be a general predisposition to subsequent mental and psychosomatic disorders (1995, p. 59).

The right hemisphere ends its growth phase in the second year, when the left hemisphere begins one, but it cycles back into growth phases at later periods of the life cycle (Thatcher, 1994). This allows for potential continuing reorganization of the emotion-processing right brain. The orbitofrontal regions, which are involved in "emotion-related learning" (Rolls, Hornak, Wade, & McGrath, 1994) are unique in that they retain the neuroanatomic and biochemical features of early development, and for this reason they are the most plastic areas of the cortex (Barbas, 1995). If, however, in its earliest organizational history this system is exposed to frequent and intense caregiver-induced dysregulation, its primordial organization will be poorly capable of coping with the stresses inherent in human relationships. Maladaptive infant mental health describes a system that early on becomes static and closed, and due to its inability to respond to novel stimuli and challenging situations it does not expose itself to new forms of socioemotional experiences that are required for the continuing experience-dependent growth of the right brain.

Implications for Models of Early Intervention

It is important to remember that "type D" behaviors are found in neurologically impaired infants, and infants with early neurological insults to the orbitofrontal cortex show long-term deficits, despite optimal environments. This means that relational trauma can not be automatically inferred from deflections, even severe deflections of a normal developmental course. That being the case, the opportunity for a maturing individual, even one with a constitutional deficit, to optimize its developmental trajectory is greatly enhanced by forming a dyadic system with a primary caregiver who is sensitive to its unique strategies of processing and expressing social emotional information.

Orbitofrontal deficiencies and affect regulatory disturbances are not solely found in patients with severe relational trauma. This system is also impaired in neurological patients (Brazzelli, Colombo, Della Sala, & Spinnler, 1994), schizophrenia (Norman et al., 1997), autism (Baron-Cohen, 1995), manic state of bipolar disorder (Blumberg et al., 1999), unipolar (Biver et al., 1994) and major (Biver et al., 1997) depression, obsessive-compulsive disorder (Mcguire et al., 1994; Rauch et al., 1994), and, indeed, Alzheimeris disease (van Hoesen, Parvizi, & Chu, 2000). It is also dysfunctional in alcoholism (Hommer et al. 1997; Volkow et al., 1999; Volkow et al., 1997) and drug addiction (London et al., 2000; Volkow & Fowler, 2000), and it is tempting to speculate that the origins of a predisposition to addiction lie in prenatal exposure to maternal drug use during pregnancy (Jacobson et al., 1996; Espy et al 1999) and postnatal relational stressors embedded in "type D" parenting (OiConnor, Sigman, & Brill, 1987).

Abnormalities in the limbic system, in the frontal lobe, temporal lobe, basal ganglia, reticular formation, and the hypothalamic-pituitary-adrenal axis connectivity play a

critical role in the pathophysiology of each of these disorders, but which limbic circuit and what point in a circuit is metabolically unstable and inefficient would determine the particular expression of affect dysregulation of a specific psychiatric syndrome. The most severe disturbances would involve cell death of dopamine or noradrenaline or hypothalamic neurons at the base of the hierarchical circuits, since disruption of their trophic functions would lead to widespread alterations of subcortical and cortical structures. Less severe would be loss of the receptors for these bioagents, many of which are found on astrocyes that regulate the metabolic activity and connectional plasticity of brain synapses (Laming et al., 2000). The postnatal proliferation and growth of astrocytic processes that surround synapses is influenced by events in the social environment (Jones & Greenough, 1996).

It should also be remembered that the process of prefrontal-subcortical parcellation continues over the life stages (Keshavan, Anderson, & Pettegrew, 1994), and this process, for example, as the brain reorganizes in adolescence may pare down an already thinned cortical-subcortical system, and therefore result in the massive dysregulatory symptoms of the psychopathologies that "first appear" at this time. Yet the early regulatory deficts of social-emotional information processing of these disorders are manifest in attachment disturbances in infancy, and may be treatable at this early time.

Although I have mainly focused upon disorganized insecure attachments, what of the the other insecure categories? Insecure organized attachments also express partial orbitofrontal hypo- or hypermetabolic disturbances under stress (see Schore, 1994, 1996 for insecure resistant/ambivalent and avoidant brain organizations). However, these involve stress impairments of only one of the two limbic circuits, the sympathetic ventral tegmental limbic forebrain-midbrain circuit or the parasympathetic lateral tegmental limbic forebrain-midbrain circuit. The insecure avoidant infant/dismissing adult and insecure resistant-ambivalent/preoccupied adult organizations show inhibitory or excitatory biased orbitofrontal systems. Under stress the former can access a passive coping strategy of autoregulation, the latter an active coping capacity of interactive regulation, while the disoriented, neither. The availability of single circuit strategy is limiting, yet it allows for an organized if limited coping mechanism. The affect regulating dysfunctions of insecure organized personalities are not as severe as the disorganized-disoriented insecures, but as Main (1996) points out, these attachments are also high risk for psychiatric disorders.

I want to stress the point that I do not believe that only trauma in the first two years of life is psychopathogenic or self-disorganizing. I also am not undervaluing the long-term negative impact that an abusing father can have on the the developing child. Indeed most forms of sexual abuse are perpetrated by the father. What I am saying is that what a particular individual appraises to be stressful, how he or she characteristically consciously and especially unconsciously responds to stressors, and how efficiently he or she psychobiologically copes with these stressors, are uniquely and indelibly influenced by events in early and late infancy, especially events that involve abuse by the primary caregiver, who in the vast majority of cases is the mother. Whatis more, these early interactive experiences determine whether, in later times of crisis, the individual can allow himself to go to others for interpersonal support, that is, to avail himself of interactive regulation within an intimate or psychotherapeutic relationship when his own autoregulatory mechanisms have temporarily failed.

The promotion of affect regulation is now seen as a common mechanism in all forms of psychotherapy (Bradley, 2000). Furthermore, current developmental models clearly suggest that psychotherapeutic treatment for severe attachment disorders should begin as early in the life span as possible. Osofsky and her colleagues demonstrate that effective therapeutic interventions can be made in traumatized 2 year olds. They conclude, "Helping young children acquire self-regulation through reciprocal management of affects with an emotionally available therapist" can allow for a "return to a healthy developmental pathway" (1995, p. 605). The interactive regulation embedded in the

therapeutic relationship functions as a "growth facilitating environment," specifically for the experience-dependent maturation of right orbitofrontal systems (Schore, 1994, 1997a, in press a, b, c). This context can alter attachment patterns from "insecurity" to "earned security" (Phelps, Belsky, & Crnic, 1998).

A recently published fMRI study (Hariri et al., 2000) provides evidence that higher regions of specifically the right prefrontal cortex attenuate emotional responses at the most basic levels in the brain, that such modulating processes are "fundamental to most modern psychotherapeutic methods" (p. 43), that this lateralized neocortical network is active in "modulating emotional experience through interpreting and labeling emotional expressions" (p. 47), and that "this form of modulation may be impaired in various emotional disorders and may provide the basis for therapies of these same disorders" (p. 48). Furthermore, the co-construction of a coherent narrative of the trauma may emerge in a relational context which promotes a callosal transfer of affective information from the right to left orbitofrontal regions. This structural advance allows for left hemispheric retrieval and explicit semantic processing of right hemispheric emotional states encoded in implicit-procedural memory (Schore, in press, b).

This model also has practical implications for programs of early prevention. A logical outcome of psychological, psychoanalytic, and psychiatric theories that emphasize the centrality of early development to later functioning is that early prevention is an essential goal. In accord with clinical findings (e.g., Eckenrode et al., 2000), the latest psychoneurobiological developmental models which focus on the effects of early environmental interactions on evolving brain-behavior relationships also emphatically stress the fundamental importance of early intervention. A core postulate of classical developmental biology and now of developmental neurobiology is the concept of critical periods. This construct emphasizes that certain detrimental early influences lead to particular irreversible or only partially reversible enduring effects, highlighting the fact that limitations of biological organization set into place once systems differentiate.

Itis important to remember, however, that the flip side of the critical period concept emphasizes the extraordinary sensitivity of developing dynamic systems to their environment, and asserts that these systems are most plastic in periods when they are in the process of differentiating. The right hemisphere, which is centrally involved in both the capacity to perceive the emotional states of other human beings and the control of vital functions supporting survival and enabling the individual to cope actively and passively with stress, is in a growth spurt in the first year-and-a-half of life and is dominant for the first three. Its maturation is "experience-dependent", and this "experience" is embedded in the attachment relationship between caregiver and infant. Developmentally-focused clinicians are familiar with the various patterns of emotional transactions of securely and insecurely attached dyads. But they also have extensive clinical knowledge of how the relationship between the patient and therapist co-creates a safe environment that facilitates what Emde (1990) calls a mobilization of the patientis "biologically prepared positive developmental thrust." These same interpersonal skills and intersubjective sensitivities are valuable assets in preventive programs.

As a number of issues of this journal document, attachment researchers in association with infant mental health workers are now devising interventions that effectively alter the affect-communicating capacities of mother-infant systems, and thereby the attachment experiences of high risk dyads. By providing an optimal context for the co-creation of a system of interactive regulation that is timed to critical periods of socioemotional development, such interventions can facilitate the maturation of neurobiologically adaptive regulatory systems. Early interventions thus have lifelong effects on the adaptive capacities of a developing self. These efforts, if expanded onto a larger scale, could make deep inroads into not only altering the intergenerational transmission of psychiatric disorders but improving the quality of a life throughout the lifespan. A deepening social and political commitment to early treatment and prevention programs would thus be a major contribution to the problems our societies are now facing.

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