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Attachment trauma and the developing right brain: Origins of pathological dissociation

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The concept of dissociation has a long history of bridging psychiatry, psychology, and neurology. Because it is inextricably linked to the concept of trauma, theoretical and clinical models of dissociation have spanned the psychological and biological realms. Although the relationship between trauma early in the life span and dissociation was noted at the end of the nineteenth century, it is only recently that a developmental perspective is being used as a source of deeper understanding of the etiological mechanisms that underlie dissociation and dissociative disorders. Thus the problem of dissociation, like a broad spectrum of other clinical phenomena, is now being viewed through an interdisciplinary lens.

There is a growing appreciation that developmental models can make unique contributions to one of the fundamental problems of the human condition, psychopathogeneis. This applies to the problem of dissociation, which offers "potentially very rich models for understanding the ontogeny of environmentally produced psychiatric conditions" (Putnam, 1995, p. 582). In the following I will suggest that regulation theory (Schore, 1994, 2003a, b), which integrates developmental affective neuroscience, attachment theory, and psychiatry, can provide such models. Towards that end I will present recent observations on infant behavior from developmental psychology, current data on brain development from neuroscience, updated basic research in biological psychiatry on stress mechanisms, and new information on the essential functions of the autonomic nervous system from developmental psychobiology in order to offer an interdisciplinary model of the etiology of pathological dissociation, as well as the psychoneurobiological mechanisms that underlie dissociation. As a paradigm case of dissociative disorder I will use posttraumatic stress disorder, an Axis I environmentally produced psychiatric disorder. In particular I will discuss the earliest expression of dissociation in human infancy, its impact on the development of the maturing right brain, and its role in the first manifestation of PTSD in the life span, pediatric posttraumatic stress disorder.

Introduction

Although an important distinction is made between nonpathological and pathological experiences of dissociation (Waller, Putnam, & Carlson, 1996), the focus of this chapter will be on the latter. Dissociation is defined in DSM-IV as "a disruption in the usually integrated functions of consciousness, memory, identity, or perception of the environment" (American Psychiatric Association, 1994). In a parallel conception, the latest edition of the International Classification of Diseases (ICD-10) describes dissociation as "a partial or complete loss of the normal integration between memories of the past, awareness of identity and immediate sensations, and control of body movements" (World Health Organization, 1992). Notice that while both stress a deficit in integration, only the second classification system refers to an alteration of bodily processes. And in another widespread definition, Spiegel and Cardena characterize dissociation as "a structured separation of mental processes (e.g., thoughts, emotions, conation, memory, and identity) that are ordinarily integrated" (1991, p. 367). In contrast to the others, these authors include emotion in their definition.

The concept of dissociation, one of the most enduring in modern psychiatry, traces directly back to the work of Pierre Janet in the late nineteenth century. Janet (1887, 1889) defined (pathological) dissociation as a phobia of memories, expressed as excessive or inappropriate physical responses to thought or memories of old traumas. This dissociation of cognitive, sensory, and motor processes is adaptive in the context of overwhelming traumatic experience, and yet such unbearable emotional reactions result in an altered state of consciousness. Janet also described an "abaissement du niveau mental", a lowering of the mental level, a regression down a hierarchy to a state that is constricted and disunified. Following Charcot's work (1887) Janet also posited that a history of early trauma plays a fundamental role in the psychopathogenic origins of hysteria. Freud (1893/1955), who cited Janet in his early pre-psychoanalytic work, defined dissociation as a splitting of consciousness, frequently associated with bizarre physical symptoms. Although in his early writings Freud accepted the idea that developmental trauma is related to the characterological use of pathological dissociation, he later moved away from

this idea, and posited that repression and not dissociation was the primary mechanism of psychopathogenesis.

Summarizing the essentials of Janet's model, van der Kolk, Weisaeth, and van der Hart state:

> Janet proposed that when people experience "*vehement emotions*," their minds may become incapable of matching their *frightening experiences* with existing cognitive schemes. As a result the memories of the experience cannot be integrated into personal awareness; instead, they are split off [dissociated] from consciousness and voluntary control…extreme *emotional arousal* results in failure to integrate traumatic memories…The memory traces of the trauma linger as unconscious "fixed ideas" that cannot be "liquidated"…they continue to intrude as terrifying perceptions, obsessional preoccupations, and *somatic reexperiences.* (1996, p.52, my italics)

Janet also proposed that traumatized individuals "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" (1911, p. 532). Translating the concept of personality in contemporary terms, van der Kolk, van der Hart, and Marmar conclude that "'Dissociation' refers to a compartmentalization of experience: Elements of a trauma are not integrated into a unitary whole or an integrated sense of *self*." (1996, p. 306, my italics).

Furthermore, at the very dawn of modern psychiatry every major pioneer of the concept of dissociation, including not only Janet and Freud but also Charcot and Hughlings Jackson, was also interested in the underlying neurology of the phenomenon, that is understanding dissociation in terms of structure-function relationships. Indeed, as noted by Devinsky (2000), late nineteenth-century clinicians linked specifically the right hemisphere with emotion (Luys, 1881) and dissociative phenomena (Myers, 1885; Richer, 1881). He cites Jackson's (1876) work on the duality of the brain, and the role of the right hemisphere in "emotional" speech, as opposed to "voluntary expression and conscious awareness of propositional speech" of the left hemisphere. Dissociative psychopathology continues to be of great interest to the epilepsy literature. Patients with intractable epilepsy show high rates of "dissociative convulsions" (De Wet et al., 2003), and "dissociative pseudoseizures" are common sequelae of traumatic experiences (Harden, 1997), especially in patients with histories of sexual and physical abuse (Alper et al., 1993) and diagnoses of personality disorders and depression (Bowman & Markand, 1996).

Writing in the current neurological and neuropsychiatry literatures, Brown and Trimble (2000) refer back to the work of Janet and Freud and conclude that we now need to move beyond a purley descriptive approach: "The first goal must be to provide a precise definition of dissociation based on a conceptually coherent and empirically justified account of the processes underlying these phenomena" (p. 288). Other investigators are asserting "a precise definition of the term 'dissociation' must be established, based on a coherent and empirically checkable concept. Furthermore, it is important to discover the primary pathophysiologic mechanism that leads to the dissociative symptoms, using neurobiological research mechanisms" (Prueter, Schultz-Venrath, & Rimpau, 2002, p. 191).

Over the last few decades a small number of authors have offered neurobiological models of dissociation. Although these studies vary in terms of different psychiatric and neurological populations, all are on adults. Whitlock (1967) and Ludwig (1972) suggested that the primary pathophysiological mechanism involved in the creation and maintenance of dissociative symptoms is an attentional dysfunction resulting from an increase in the corticofugal inhibition of afferent stimulation. As a result of this inhibition, partially processed information fails to be integrated into awareness, thereby generating dissociative symptoms. More recently J. Krystal et al. (1998), Scarr (2001), and Nijenhuis, Van der Hart, and Steele (2002) have offered contributions on the psychobiology of dissociation. And as I will discuss in upcoming sections, current neuroimaging research is adding greatly to our knowledge of the structure-function relationships of dissociation, though these too currently focus only on mature brain systems.

In psychological studies of adults Loewenstein notes that "Dissociation is conceptualized as a basic part of the psychobiology of the human trauma response: a protective activation of altered states of consciousness in reaction to overwhelming psychological trauma" (1996, p. 312). And in neuropsychiatric research on adult trauma patients, Bremner and his colleagues demonstrate that there are two subtypes of acute trauma response, hyperarousal and dissociation (1999), that dissociation represents an effective short-term strategy that is detrimental to long-term functioning (Bremner & Brett, 1997), and that exposure to extreme stress signals the invocation of neural mechanisms that result in long-term alterations in brain functioning (J. Krystal et al., 1998). Meares also concludes that in all stages "dissociation, at its first occurrence, is a

consequence of a ‘psychological shock’ or high arousal” (1999, p. 1853).

In the following I will offer evidence to show that every one of these observations of dissociation in adults applies to dissociation as it occurs in infants. I will argue that developmental studies can offer not only specific models of how early trauma alters the ontogenetic trajectory and imprints a predisposition for later pathological dissociation, but also a deeper understanding of its underlying neurobiological mechanisms. And I will describe how the current point of contact of development and trauma and dissociation is attachment theory, “the dominant approach to understanding early socioemotional and personality development during the past quarter-century of research” (Thompson, 2000, p. 145). Disorganized-disoriented insecure attachment, a primary risk factor for the development of psychiatric disorders (Main, 1996), has been specifically implicated in the etiology of the dissociative disorders (Chefetz, in press; Liotti, 1992; Schore, 1997). In longitudinal studies, attachment researchers demonstrate an association between traumatic childhood events and proneness to dissociation (Ogawa et al., 1997).

Current models of the neurobiology of attachment are focusing on the impact of both regulated and dysregulated attachment experiences on the formation of the implicit self system, located in the early maturing right brain (Schore, 1994, 2001a). Researchers are now asserting that an emphasis on fearful arousal and the relational modulation of that arousal lies at the heart of attachment theory, and that relational trauma triggers states of hyperarousal and dissociation in the developing brain. In later sections of this chapter I will cite studies to show that abuse and neglect, the first forms of survival threat to the developing infant elicit dissociative defenses, which in turn negatively impact the critical period growth of cortical, limbic, brainstem, and autonomic centers in the right brain.

Recent developmental studies strongly support Janet’s ideas about early trauma and dissociation, and clearly indicate that experiences with a traumatizing caregiver negatively impact the child’s attachment security, stress coping strategies, and sense of self (Crittenden & Ainsworth, 1989; Erickson, Egeland, & Pianta, 1989). There is now a large and convincing body of evidence to show that psychic trauma in childhood results in an arrest of affective development, whereas trauma in adulthood leads to regression in affective development (H. Krystal, 1988), and that the most significant consequence of early relational trauma is the lack of capacity for emotional self-regulation (Toth & Cicchetti, 1998), expressed in the loss of the ability to regulate the intensity and duration of affects (van der Kolk & Fisler, 1994). In total, this chapter presents an argument that these established principles of early emotional development must be incorporated into an overarching model of dissociation.

With this introduction in mind, in this chapter I will discuss how an interdisciplinary perspective can be used to generate a more complex model of dissociation and its role in psychopathogenesis. I will then present a brief overview of the neurobiology of a secure attachment, followed by the neurobiology of infant relational trauma and dissociation and the developmental neuropsychology of dissociation found in ““type D”” disorganized attachment. In the latter sections of the chapter I will discuss the role of right brain processes in dissociation at later stages of the life span, and will end with some speculations about the basic biological mechanisms that mediate dissociation and the implications of this work for DSM-V.

The neurobiology of a secure attachment

In the last decade significant advances have occurred in our knowledge of normal and abnormal social and emotional development. The essential task of the first year of human life is now seen as the creation of a secure attachment bond of emotional communication between the infant and the primary caregiver. In order to enter into this communication, the mother must be psychobiologically attuned to the dynamic crescendos and decrescendos of the infant’s bodily-based internal states of arousal. Within a context of visual-facial, gestural, and auditory-prosodic communications each partner learns the rhythmic structure of the other and modifies his or her behavior to fit that structure, thereby co-creating a specifically fitted interaction. During mutual gaze episodes of bodily-based affective communications the spatiotemporal patterning of the primary caregiver’s exogenous sensory stimulation is synchronized with the spontaneous expressions of the infant’s endogenous organismic rhythms. This contingent responsivity allows her to appraise the nonverbal expressions of her infant's internal arousal and affective states, regulate them, and then communicate them back to the infant. To effectively accomplish this interactive regulation, the mother must modulate nonoptimal high *or* low levels of stimulation which would induce supra-heightened or extremely low levels of arousal in the infant.

If attachment is the regulation of interactive synchrony, stress is defined as an asynchrony in an interactional sequence, and following this, a period of re-established synchrony allows for stress recovery and coping. The regulatory processes of affect synchrony that co-creates states of positive arousal and interactive repair that

modulates states of negative arousal are the fundamental building blocks of attachment and its associated emotions, and resilience in the face of stress is an ultimate indicator of attachment security. Attachment, the outcome of the child's genetically encoded biological predisposition and the particular caregiver environment, thus represents the regulation of biological synchronicity between and within organisms, and imprinting, the learning process that mediates attachment, is defined as synchrony between sequential infant-maternal stimuli and behavior

Current research supports earlier proposals (Schore, 1994) that the long enduring effects of the regulation embedded in the attachment relationship are due to their impact on brain development. According to Ziabreva et al. (2003),

> [T]he mother functions as a regulator of the socio-emotional environment during early stages of postnatal development…subtle emotional regulatory interactions, which obviously can transiently or permanently alter brain activity levels…may play a critical role during the establishment and maintenance of limbic system circuits (Ziabreva et al., 2003, p. 5334).

In a number of writings I have suggested that the attachment mechanism is embedded in right hemisphere-to-right hemisphere regulated affective transactions between the primary caregiver and her infant (Schore, 1994, 2000, 2003a, b). In light of the facts that the human limbic system myelinates in the first year-and-a-half (Kinney et al., 1988), and that the early maturing right hemisphere (Chiron et al. 1997; Geschwind & Galaburda, 1987), which is deeply connected into the limbic system (Tucker, 1992) is in a growth spurt at this time, attachment experiences specifically impact limbic and cortical areas of the developing right cerebral hemisphere (Henry, 1993; Schore, 1994; Siegel, 1999; Wang, 1997).

This model is supported by a growing body of recent research. At two months of age, the onset of a critical period during which synaptic connections in the developing occipital cortex are modified by visual experience (Yamada et al., 1997, 2000), infants show right hemispheric activation when exposed to a woman's face (Tzourio-Mazoyer, 2002). The development of the capacity to efficiently process information from faces requires visual input to the right (and not left) hemisphere during infancy (Le Grand et al., 2003). Mutual gaze engages face processing areas of the right hemisphere (Pelphrey, Viola, & McCarthy, 2004; Watanabe, Miki, & Kakigi, 2002), and the tendency of mothers to cradle infants on their left side "facilitates the flow of affective information from the infant via the left ear and eye to the center for emotional decoding, that is, the right hemisphere of the mother" (Manning et al., 1997, p. 327). With respect to the communication of gestural and auditory information, expressive gestures that express inner feeling states activate right hemispheric structures (Gallagher & Frith, 2004), and the human maternal response to an infant's cry, a fundamental behavior of the attachment dynamic, is accompanied by activation of the mother's right brain (Lorberbaum et al., 2002).

The neurobiology of relational trauma

Optimally regulated attachment communications directly influence the maturation of both the postnatally maturing central nervous system (CNS) limbic system that processes and regulates social-emotional stimuli and the autonomic nervous system (ANS) that generates the somatic aspects of emotion. It is important to stress that the developmental attainment of an efficient internal system that can adaptively regulate various forms of arousal and psychobiological states, and thereby affect, cognition, and behavior, only evolves in a growth-facilitating emotional environment. The good-enough mother of the securely attached infant permits access to the child after a separation and shows a tendency to respond appropriately and promptly to his/her emotional expressions. She also allows for the interactive generation of high levels of positive affect in co-shared play states. These regulated events support an expansion of the child's coping capacities, and account for the principle that security of the attachment bond is the primary defense against trauma-induced psychopathology.

In contrast to this scenario is a relational growth-inhibiting early environment, in which the abusive caregiver not only shows less play with her infant, but also induces traumatic states of enduring negative affect in the child. 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 therefore shows minimal or unpredictable participation in the various types of arousal regulating processes. Instead of modulating she induces extreme levels of stimulation and arousal, very high in abuse and/or very low in neglect. And because she provides no interactive repair the infant's intense negative states last for long periods of time.

More specifically, the infant's psychobiological reaction to trauma is comprised of two separate response patterns, hyperarousal and dissociation (Perry et al., 1995; Schore, 1997). The first stage is described by Beebe in her

observation of “mutually escalating overarousal” in 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 screaming, the mother keeps going (2000, p. 436).

In this initial stage of threat, an alarm or startle reaction is expressed, reflecting activation of the infant’s right hemisphere, the locus of the startle mechanism (Bradley, Cuthbert, & Lang, 1996). This triggers a sudden increase of the sympathetic component of the ANS, resulting in significantly elevated heart rate, blood pressure, and respiration. Distress is expressed in crying and then screaming. Crying represents an autonomic response to stress, whereby the nucleus ambiguus of the right vagus excites both the right side of the larynx and the sinoatrial node of the heart (Porges et al., 1994).

The infant’s state of “frantic distress,” or what Perry terms fear-terror is mediated by sympathetic hyperarousal, expressed in increased levels of the brain’s major stress hormone, corticotropin releasing factor, which in turn regulates sympathetic catecholamine activity (Brown et al., 1982), and so brain adrenaline, noradrenaline, and dopamine levels are significantly elevated, creating a hypermetabolic state within the developing brain. In addition, increased amounts of vasopressin are expressed, a hypothalamic neuropeptide associated with sympathetic activation that is specifically relaesed when an environment is perceived to be unsafe and challenging (Kvetnansky et al., 1989; 1990).

But a second later forming reaction to infant trauma is seen in dissociation, in which the child disengages from stimuli in the external world and attends to an “internal” world. Traumatized infants are observed to be “staring off into space with a glazed look”. Winnicott (1958) holds that a particular failure of the maternal holding environment causes a discontinuity in the baby’s need for “going-on-being, and ”Kestenberg (1985) refers to as dead spots in the infant's subjective experience, an operational definition of the restriction of consciousness of dissociation. This same response is described by Tronick and Weinberg :

> [W]hen infants’ attempts 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 infant’s reaction is reminiscent of the withdrawal of Harlow’s isolated monkey or of the infants in institutions observed by Bowlby and Spitz (1997, p. 66).

The child’s dissociation in the midst of terror involves numbing, avoidance, compliance and restricted affect (the same pattern as adult PTSD).

This parasympathetic dominant state of conservation-withdrawal occurs in helpless and hopeless stressful situations in which the individual becomes inhibited and strives to avoid attention in order to become "unseen" (Schore, 1994, 2001b). This metabolic shutdown state is a primary regulatory process, used throughout the life span, 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).

The neurobiology of the later forming dissociative hypoarousal is different than the initial hyperarousal response. In this passive state pain numbing and blunting endogenous opiates (Fanselow, 1986) are elevated. Furthermore, the dorsal vagal complex in the brainstem medulla is rapidly activated, decreasing blood pressure, metabolic activity, and heart rate, despite increases in circulating adrenaline. This elevated parasympathetic arousal, a survival strategy (Porges, 1997), allows the infant to maintain homeostasis in the face of the internal state of sympathetic hyperarousal. It is often overlooked that parasympathetic energy-conserving hypoarousal as well as sympathetic energy-expending hyperarousal represent states of Janetian “extreme emotional arousal.”

Vagal tone is defined as “the amount of inhibitory influence on the heart by the parasympathetic nervous system” (Field et al., 1995). But it now known that there are two parasympathetic vagal systems, a late developing “mammalian” or “smart” ventral vagal 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 “reptilian” 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). As opposed

to the ventral vagal complex that can rapidly regulate cardiac output to foster engagement and disengagement with the social environment, the dorsal vagal complex "contributes to severe emotional states and may be related to emotional states of 'immobilization' such as extreme terror" (Porges, 1997, p. 75).

Perry's description of the traumatized infant's sudden state switch from sympathetic hyperarousal into parasympathetic dissociation is reflected in Porges' 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)

H. Krystal also describes the state switch from sympathetic hyperaroused-terror into parasympathetic hypoaroused 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)

As opposed to the nucleus ambiguus which exhibits rapid and transitory patterns associated with perceptive pain and unpleasantness, the dorsal vagal nucleus shows an involuntary and prolonged characteristic pattern of vagal outflow. This prolonged state of dorsal vagal parasympathetic activation accounts for the extensive duration of "void" states associated with pathological dissociative detachment (Allen, Console, & Lewis, 1998).

Developmental neuropsychology of dissociation

The next question is, how would the trauma-induced neurobiological and psychobiological alterations of the developing right brain be expressed in the socioemotional behavior of an early traumatized toddler? In a classic study, Main and Solomon (1986) studied the attachment patterns of infant's 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, one found in 80% of maltreated infants (Carlson et al., 1989) and associated with pre- and/or postnatal maternal alcohol or cocaine-use (Espinosa et al., 2001; O'Connor, Sigman, & Brill, 1987). Hesse and Main (1999) point out that the disorganization and disorientation of type "D" attachment phenotypically resembles dissociative states. Main and Solomon (1986) conclude that these infants are experiencing low stress tolerance and 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 active coping strategy to deal with this emotional challenge.

These authors also documented, in some detail, the uniquely bizarre behaviors these 12-month-old infants show in Strange Situation observations. They note that these episodes of interruptions of organized behavior are often brief, frequently lasting only 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 shifts of state during the stress-inducing Strange Situation. Main and Solomon describe the child's entrance into a dissociated state:

> One infant hunched her upper body and shoulders at hearing her mother's call, then broke into extravagant 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 (1986, p. 119).

These behaviors generalize beyond just interactions with the mother. The authors note that the intensity of the

baby's dysregulated affective state is often heightened when the infant is exposed to the added stress of an unfamiliar person. At a stranger's 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." These infants exhibit "behavioral stilling" - that is, "dazed" behavior and depressed affect, behavioral manifestations of dissociation. One infant "became for a moment excessively still, staring into space as though completely out of contact with self, environment, and parent." 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." Yet another "fell face-down on the floor in a depressed posture prior to separation, stilling all body movements." Following up this work, Guedeney and Fermanian (2001) report an infant assessment scale of sustained withdrawal, associated with disorganized attachment, manifest in a *frozen*, absent facial expression, total avoidance of eye contact, immobile level of activity, absence of vocalization, absence of relationship to others, and the impression that the child is beyond reach.

In addition to attachment studies, the state of dissociation has also been explored by developmental researchers using the still-face procedure, an experimental paradigm of traumatic abuse, specifically neglect (see Figure 1). In this experimental procedure the infant is exposed to a severe relational stressor, as the mother, although maintaining eye contact with the infant, suddenly inhibits all vocalization and suspends any spontaneous emotionally expressive facial expression or gesture. This loss of dyadic regulation triggers an initial increase of interactive behavior and arousal in the infant. According to Tronick (2004), the infant's initial state of confusion and fearfulness at the break in connection is manifest in the cognition, "this is threatening." But this is soon followed by bodily collapse, loss of postural control, withdrawal, gaze aversion, sad facial expression, and self-comforting behavior.

Figure 1. An infant losing postural control and self-comforting in response to the mother being still-faced. From Tronick (2004).

Most interestingly, this behavior is accompanied by a "dissipation of the infant's state of consciousness" and a diminishment of self-organizing abilities, which in turn reflects "the disorganization of many of the lower level psychobiological states, such as metabolic systems." Recall that dissociation, a hypometabolic state, has been defined in the DSM as "a disruption in the usually integrated functions of consciousness" and described as "a protective activation of altered states of consciousness in reaction to overwhelming psychological trauma" (Loewenstein, 1996). In recent work Tronick (2004) suggests that infants who have a history of chronic breaks of connections exhibit an "extremely pathological state" of emotional apathy, equated with Spitz hospitalism effects and Romanian orphans who fail to grow and develop. These infants ultimately adopt a communication style described as "stay away, don't connect." This defensive strategy reflects a state of very early-forming yet already chronic pathological dissociation associated with loss of ventral vagal and dominance of dorsal vagal parasympathetic states.

Note that the still-face induction of hyperarousal and dissociation occurs within the maternal-infant face-to-face context. The mother's face is the most potent visual stimulus in the child's world, and it is well known that direct gaze can mediate not only loving but powerful aggressive messages. In coding the mother's frightening behavior Hesse and Main (1999, p. 511) describe "in non-play contexts, stiff-legged 'stalking' of infant on all fours in a hunting posture; exposure of canine tooth accompanied by hissing; deep growls directed at infant." Thus, during the trauma, the infant is presented with an aggressive expression on the mother's face. The image of

this aggressive face, as well as the chaotic alterations in the infant's bodily state that are associated with it, are indelibly imprinted into limbic circuits as a "flashbulb memory," and thereby stored in imagistic procedural memory in the visuospatial right hemisphere, the locus of implicit (Hugdahl, 1995) and autobiographical (Fink et al., 1996) memory.

But in traumatic episodes 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 mother's dysregulated states, and that this synchronization is registered in the firing patterns of the stress-sensitive corticolimbic regions of the infant's brain, especially in the right brain that are in a critical period of growth. It is now established that maternal care influences both the infant's reactivity (Menard et al., 2004) and defensive responses to threat "which serve as the basis for the transmission of individual differences in stress responses from mother to offspring" (Weaver et al., 2004, p. 847). In light of the fact that many of these mothers have suffered from unresolved trauma themselves, this spatiotemporal imprinting of the chaotic alterations of the mother's dysregulated state facilitates the down-loading of programs of psychopathogenesis, a context for the intergenerational transmission of both trauma and of the dissociative defense against overwhelming and dysregulating affective states that disorganize the nascent self system.

Right brain processes and dissociation throughout the life span

In an early history of traumatic attachment the developing infant/toddler is too frequently exposed to a massively misattuning primary caregiver who triggers and does not repair long-lasting intensely dysregulated states. The growth-inhibiting environment of relational trauma generates dense and prolonged levels of negative affect associated with extremely stressful states of hyper- and hypoarousal. And so for self-protective purposes he/she severely restricts the overt expressions of an attachment need for dyadic regulation. The child thus significantly reduces the output of its emotion-processing, limbic-centered attachment system. When stressed, defensive functions are rapidly initiated that quickly shift the brain from interactive regulatory modes into long-enduring, less complex autoregulatory modes. These patterns are primitive strategies for survival that remain on-line for long intervals of time, periods in which the developing brain is in a hypometabolic state, detrimental to the substantial amounts of energy required for critical period biosynthetic processes. This hypometabolic brain state is responsible for dissociative "encoding failures" (Allen et al., 1998) of autobiographical memory of the developing self.

Attachment trauma between the infant and primary caregiver thereby sets the stage for the characterological use of primitive autoregulation, for the continuity of pathological dissociation over all later stages of human development. In accord with this model, Draijer and Langeland (1999) reported that specifically severe early maternal dysfunction is associated with level of dissociation in psychiatric patients. This finding has been replicated in a very recent study of the effects of childhood traumatization by Roelofs et al. (2002), who show that physical abuse and parental dysfunction by the mother – not the father – is associated with somatoform dissociative experience. In congruent findings attachment studies reveal that individuals with the "type D" classification utilize dissociative behaviors in later stages of life (van Ijzendoorn et al., 1999). The characterological use of dissociation by certain personalities is described 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).

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. During these intervals it is shut-down to the external relational environment, and thus totally closed and impermeable to attachment communications and interactive regulation, critical sources of possible further emotional development. Dissociative detachment (Allen et al., 1998) thus becomes an attractor state. Intimate social relationships are habitually appraised at a nonconscious level to be dangerous, because these contexts are always potential triggers of "vehement emotions." The avoidance of emotional connections, especially those containing novel and more complex affective information, prevents emotional learning, which in turn precludes any advances

of right brain emotional intelligence (Schore, 2001a) or what Janet (1889) calls an “enlargement” of personality development.

A fundamental question that must be addressed in any developmental model of dissociation is what is the precise mechanism by which the early psychological events of “maltreatment-related” (Beer & De Bellis, 2002) or “pediatric” (Carrion et al., 2001) posttraumatic stress disorder effect the later behavior of the self system as it develops at further stages of the life cycle? I suggest that a purely psychological conception can not answer this question, and that a psychoneurobiological perspective that integrates both biological structure and psychological function is required to model how and why pathological dissociation becomes characterological in certain developing personalities. Findings in the field of developmental traumatology now clearly indicate that “the overwhelming stress of maltreatment in childhood is associated with adverse influences on brain development” (1999, p. 1281).

In the first years of life, a time of right brain growth (Trevarthen, 1996) and right brain dominance (Chiron et al., 1997), this principle specifically applies to an adverse influence on the development of particularly the right brain. In the infant brain, states become traits (Perry et al., 1995), and so the imprinting of early relational trauma as well as dissociation, the major defense against attachment trauma are embedded into the core structure of the evolving right brain. Indeed, there is now evidence to show that early relational trauma is particularly expressed in right hemisphere deficits. Very recent studies reveal that maltreated children diagnosed with PTSD manifest right-lateralized metabolic limbic abnormalities (De Bellis et al., 2000), and that adults severely abused in childhood (Raine et al., 2001) and diagnosed with PTSD (Galletly et al., 2001) show reduced right hemisphere activation during a working memory task. This research supports van der Kolk’s (1996) and my own (1997) earlier assertions that the symptoms of PTSD fundamentally reflect an impairment of the right brain, and Spivak et al.’s (1998) proposal that the right hemisphere plays a paramount role in perceptual and cognitive processing and in the regulation of biological responses in PTSD patients.

Neurobiological research thus suggests a continuity over the course of the life span of the expression of not only the stress coping deficits of PTSD but also the use of the primitive defense of pathological dissociation in patients with a history of relational trauma. The principle that severe attachment psychopathologies frequently access this “primitive” mode of autoregulation to cope with Janetian “vehement emotions” can be translated into the clinical tenet that in PTSD in infancy, childhood, and adulthood (and other early-forming severe pathologies of the self), the individual is cut off (dis-associated) from experiencing intense affective states. Lane and his colleagues state that “traumatic stress in childhood could lead to self-modulation of painful affect by directing attention away from internal emotional states” (1997, p. 840).

This deficit results from attachment trauma that induces an enduring impairment of what Emde (1983) terms the “affective core”, the primordial central integrating structure of the nascent self, and Joseph (1992) describes as the "childlike central core" that maintains the self image and all associated emotions, cognitions, and memories that are formed during childhood. Joseph localizes this core system in the right brain and limbic system. Recall Devinsky’s (2000) assertion that optimal right hemispheric functions allow for the operations of “a coherent, continuous, and unified sense of self,” and citation of nineteenth century authors who postulated a connection between right hemispheric dysfunction and dissociation.

At the outset of this chapter I stated my intention to use PTSD as a model system to study pathological dissociation, and argued that dissociation in adults also applies to dissociation as it occurs in infants. Both developmental (Perry et al., 1995; Schore, 1997) and adult (Bremner, 1999) studies support the propositions that there are two subtypes of acute trauma response in PTSD, hyperarousal and dissociative. I suggest that in all stages of life dissociation is a consequence of a ‘psychological shock’ or high arousal (Meares, 1999) and that “at extremely high levels of arousal, coherent integration of sensory information breaks down and dissociative symptoms emerge” (J. Krystal et al., 1995). Indeed current neuropsychiatric research indicates that both hyperarousal and dissociative responses are essentially right brain-driven processes.

With regard to hyperarousal, Metzger et al. (2004) report “PTSD arousal symptoms are associated with increased right-sided parietal activation” (p. 324). Similarly, Bonne et al. (2003) document “regional blood flow in right precentral, superior temporal, and fusiform gyri in posttraumatic stress disorder was higher than in healthy controls” (p. 1077), a finding that “may represent continuous preparatory motor activation, reflecting an increased basal level of anxiety and arousal,” and suggest, “this may reflect a component common to all survivors of trauma” (p. 1081). And Lanius et al. (2004) observe that PTSD patients (as opposed to traumatized patients without PTSD) who experience traumatic memories with heart rate increases (an autonomic indicator of hyperarousal), show a

pattern of right brain connectivity of activation of the right posterior cingulate, right caudate, right occipital and right parietal lobe. They conclude that this right-lateralized pattern "may account for the nonverbal nature of traumatic memory in PTSD subjects" and cite other studies showing that "subjects who had experienced early trauma displayed…right dominance during memory recall."

Other recent studies reveal that dissociation in PTSD is also centered in right brain processes. In fMRI research Lanius and her colleagues (2002) studied PTSD patients while they were in a dissociative state, as reflected in a lack of increase in heart rate when exposed to their traumatic script. These authors note that

> activation effects in the superior and middle temporal gyrus, anterior cingulate, medial parietal lobe, and medial frontal gyres in the dissociated PTSD subjects were lateralized to the right side. The possibility that childhood trauma sets the stage for lateralized responses is given credence by report from Schiffer et al. (1995) who showed right hemisphere activation …during recall of unpleasant memories in adults with a history of childhood abuse (p. 309).

They conclude "prefrontal and limbic structures underlie dissociative responses in PTSD" and state that activation of the right superior and middle temporal gyri in dissociated PTSD patients is consistent with a corticolimbic model of dissociation. Citing this work, Gundel et al. (2004) note that in dissociating (and alexithymic) patients "have difficulties in integrating aspects of certain neuropsychological functions, namely memories and feelings, into current awareness", and propose that the right anterior cingulate "may represent the structural, neuroanatomical correlate of an active inhibitory system causing a down regulation of emotional processing during the…expressive aspects of emotion" (p. 138).

Very similar findings are reported by Spitzer et al. (2004) in a transcranial magnetic stimulation study. These researchers state that their data confirms the idea that dissociation may involve

> …a lack of integration in the right hemisphere. This corresponds with the idea that the right hemisphere has a distinct role in establishing, maintaining, and processing personally relevant aspects of an individual's world. Thus a right hemispheric dysfunction might result in an altered sense of personally relevant familiarity, which resembles phenomenologically the dissociative symptoms of depersonalization and derealization...trauma-related conditions, which themselves are closely-associated with dissociative psychopathology, lack right hemispheric integration (p. 167)

Citing the DSM-IV definition they conclude, "In dissociation-prone individuals, a trauma that is perceived and processed by the right hemisphere will lead to a 'disruption in the usually integrated functions of consciousness' " (p. 168).

Dysregulation of right-lateralized limbic-autonomic circuits and dissociation

These studies, which reflect the evolution of an early dysregulated developing system over the course of the life span, provide further evidence that prefrontal cortical and limbic areas of particularly the right hemisphere are centrally involved in the dissociative response. This hemisphere, more so than the left, is densely reciprocally interconnected with limbic regions and with subcortical areas that generate the physiological aspect of emotions, including fear-terror (Adamec, 1999; Adolphs, Tranel, & Damasio, 2001; Borod, 2000; Gainotti, 2000; Tucker, 1992). Hecaen and Albert (1978) have described the much overlooked importance of hierarchical vertical corticosubcortical functional systems:

Cortical neural mechanisms of one hemisphere would be responsible for a particular performance, and subcortical structures connected to these cortical zones would participate in the realization of the performance, creating a complex, corticosubcortical functional system specific to each hemisphere. (p. 414)

This "vertical" model of cortical-subcortical circuits directly applies to models of the right hemisphere, "the emotional brain":

Neural processing of emotions engages diverse structures from the highest to the lowest levels of the neuraxis. On the one hand, high-order association areas are necessary to understand the significance of an emotional situation, and on the other hand, low level structures must be activated to express the emotion through changes in the rhythm of peripheral organs. (Barbas et al., 2003)

These vertical circuits also account for the fact that the right hemisphere contains the major circuitry of emotion regulation (Brake et al., 2000; Porges, Doussard-Roosevelt, & Maiti, 1994; Schore, 1994).

I suggest that dissociation, a primitive coping strategy of affect regulation, is best understood not in terms of a horizontal disconnection of the communication of emotional information across right and left hemispheres, but as a loss of vertical connectivity between cortical and subcortical limbic areas within the right hemisphere. The former, such as propounded by J. Krystal et al. (1998) emphasizes "shifts in interhemispheric processing" and "cortical disconnectivity" between higher frontal and limbic structures. Ontogenetically, dissociation appears well before the frontal areas of the cerebral cortex are myelinated and before callosal connections are functional (Bergman, Linley, & Fawcus, 2004; Schore, 2001a). Models of this early appearing defense against organismic threat, like models of PTSD hyperarousal and hypoarousal (Schore, 2002) must move down the neuraxis, into the brainstem that generates states of arousal.

In a recent conception congruent with this vertical model, Scaer postulates that dissociation:

> is elicited by internal and external cue-specific stimuli, but because the threat itself has not been resolved, internal cues persist without inhibition from external messages of safety, and kindling is triggered in the *cortical, limbic, and brainstem centers* (2001, p. 84, my italics).

Notice the reference to not only brainstem centers but also to *external and internal* cues, clearly implying top-down and bottom-up processing. It is now accepted that pathological dissociative detachment is a defensive state driven by fear, in which the stressed individual copes by pervasively and diffusely disengaging attention "from both the *outer and inner* worlds" (Allen et al., 1998, p. 164, my italics). In a similar conceptualization Putnam (1997) describes dissociation between "*an observing and experiencing ego*." However, the dissociation literature has been vague about specifying the exact nature of the "inner world" and the "experiencing ego."

In a number of works I have suggested that what is "experienced" are bodily states, and that the 'inner world", the source of "internal cues", is more so than cognitions, the realm of bodily processes, central components of emotional states (Schore, 1994). According to Allen and his colleagues, "dissociatively detached individuals are not only detached from the environment, but also from the self - their body, their own actions, and their sense of identity " (p. 165, my italics). Recall the ICD-10 definition of dissociation as "a partial or complete loss of the normal integration between memories of the past, awareness of identity and immediate sensations, and control of body movements."

More specifically, recent information about the autonomic nervous system, or what Jackson (1931) called the "physiological bottom of the mind" is central to a deeper understanding of the mind-body alterations of trauma and the mechanism of dissociation (Schore, 2001b, 2002). Indeed, the higher regulatory systems of the right hemisphere form extensive reciprocal connections with not only the limbic but also the sympathetic and parasympathetic branches of the autonomic nervous system (Erciyas et al., 1999; Spence, Shapiro, & Zaidel, 1996; Tucker, 1992; Yoon et al., 1997), and thereby controls the somatic components of a variety of emotional responses, especially patterns of autonomic physiological responses to social stimuli. Adaptive right brain emotion processing is thus dependent upon the integration of the activities of the central (CNS) and the autonomic (ANS) nervous systems (Hagemann, Waldstein, & Thayer, 2003).

According to Porges at al. (1994) the lower right side of the brainstem that controls the ANS is innervated by the amygdala and unnamed higher limbic structures, and this "vagal circuit of emotion regulation" provides the primary central regulation of homeostasis and physiological reactivity. As the name suggests, Porges' model emphasizes the lower structures in a vertical system. Although he details the brainstem components, he refers to the higher structures as "cortex" that processes information from the social environment. And yet there is a clear implication of a bidirectional system, in which both top-down and bottom-up processes are responsible for adaptive regulatory functioning.

The concept of CNS-ANS limbic-autonomic circuits has also been described by Benarroch (1997) in his model of a central autonomic network (CAN), an internal regulation system through which the brain controls visceromotor, neuroendocrine, and behavioral responses. Like Porges' model, the CAN is a bidirectional hierarchical system, but here more of the detailed focus is on the higher limbic rather than the lower brainstem structures. The CAN is composed of limbic areas in ventromedial (orbital) prefrontal cortex, anterior cingulate, insula, and amygdala, diencephalic areas in the hypothalamus, and brainstem structures in the periaqueductal grey matter, and the nucleus of the solitary tract and nucleus ambiguus in the medulla. Hagemann, Waldstein, and Thayer (2003) characterize the CAN as

> a network of neural structures that generate, receive, and integrate internal and external information in the service of goal-directed behavior and organism adaptability…These structures are reciprocally interconnected such that information flows in both directions – top-down

> and bottom-up. The primary output of the CAN is mediated through the preganglionic sympathetic and parasympathetic neurons. These neurons innervate the heart via the stellate ganglia and the vagus nerve. (pp. 83-84)

When this network is either completey uncoupled or rigidly coupled the individual is less able to dynamically assemble the components of the network to meet an environmental challenge and is less adaptive, and therefore displays deficits in emotional expression and affect regulation (Demaree et al., 2004). This finding leads back to the problem of psychopathogeneis – what events could be responsible for such deficits?

Authors are now describing the developmental process of "cerebral maturation in the vertical dimension" (Luu & Tucker, 1996). Both the ANS and the CNS continue to develop postnatally, and the assembly of these limbic-autonomic circuits (Rinaman, Levitt, & Card, 2000) is experience-dependent (Schore, 1994, 2001a). These experiences are provided in attachment transactions of the first and second year, in which the primary caregiver provides complex interpersonal stimuli and interactive affect regulation. Optimal early growth-facilitating environments that promote secure attachments allow for the organization of limbic-autonomic circuits and a right hemispheric limbic-modulated ventral vagal parasympathetic circuit of emotion regulation that mediates both emotion and communication processes (Porges et al., 1994).

This complex system exhibits an adaptive capacity to modulate, under stress, a flexible coping pattern in which homeostatic increases in the activity in one ANS division are associated with decreases in the other. An autonomic mode of coupled reciprocal sympathetic-parasympathetic control is behaviorally expressed in an organism that responds alertly and adaptively to a personally meaningful (especially social) stressor, yet as soon as the context is appraised as safe, immediately returns to the relaxed state of autonomic balance. Thus, the ANS is not only sensitive to environmental demands and perceived stresses and threats, but will, in a predictable order, also rapidly reorganize to different neural-mediated states (Porges, 2001).

In contrast to this developmental scenario is a disorganized-disoriented attachment context, wherein the traumatizing primary caregiver amplifies infant states of dysregulating hyperarousal and/or dissociative hypoarousal. This relational intersubjective context serves as a growth-inhibiting environment for the experience-dependent maturation of CNS-ANS links, which are more extensive on the right side of the brain. In this manner, as the title of this chapter denotes, dysregulation of the developing right brain is associated in the short-term with traumatic attachment and in the long-term with the psychopathogeneis of dissociation. An extensive parcellation of vertical circuits in the developing right brain would lead to an inefficient regulation of the ANS by higher centers in the CNS, functionally expressed as a dissociation of central regulation of sympathetic and hypothalamic-pituitary-adrenal systems (Young, Ross, & Landsberg, 1984).

This model of dissociation as a stress-induced disconnect between right brain CNS and ANS systems directly applies to the etiology and psychobiological mechanism of "somatoform dissociation," which is an outcome of early onset traumatization, often involving physical abuse and threat to life by another person. According to Nijenhuis (2000) somatoform dissociation is expressed as a lack of integration of sensorimotor experiences, reactions, and functions of the individual and his/her self-representation. Recall that optimal right hemispheric functions allow for the operations of "a coherent, continuous, and unified sense of self" (Devinsky, 2000).

Psychopathological regulatory systems thus contain poorly evolved CNS-ANS limbic-autonomic switching mechanisms that are inefficient or incapable of uncoupling and recoupling the sympathetic and parasympathetic components of the ANS in response to changing environmental circumstances. This "nonreciprocal mode of autonomic control" (Berntson et al., 1991) shows an inability to adapt to stress, and the continued inhibition of internal systems that is inappropriate to a particular environmental situation essentially defines the coping limitations of pathological dissociation. In other words, dissociation reflects the inability of the right brain cortical-subcortical system to recognize and coprocess external stimuli (exteroceptive information coming from the relational environment) and on a moment-to-moment basis integrate them with internal stimuli (interoceptive information from the body, the corporeal self). Neuroscience writers now refer to "a dissociation between the emotional evaluation of an event and the physiological reaction to that event, with the process being dependent on intact right hemisphere function" (Crucian et al., 2000, p. 643).

An immature right brain circuit of emotion regulation would show deficits in "intense emotional-homeostatic processes" (Porges et al., 1994), that is, it would too easily default from fast-acting ventral vagal to slow-acting dorsal vagal systems in moments of "vehement emotions," and thereby be unable to adaptively and flexibly shift internal

states and overt behavior in response to stressful external demands. Indeed, the ventral vagal complex is known to be defective in PTSD patients (Sahar, Shalev, & Porges, 2001), and this has been suggested to account for the basal hyperarousal and higher heart rates of these patients (Sack, Hopper, & Lamprecht, 2004). I suggest that under high stress an unstable ventral vagal system could be rapidly displaced by a dorsal vagal system, and therefore account for the low heart rate of dissociative hypoarousal.

The dis-association of higher corticolimbic areas of the CAN internal regulation system and Porges' right brain circuit of emotion regulation would preclude top-down control of lower brainstem and autonomic functions and the adaptive integration of CNS exteroceptive and ANS interoceptive information processing. This disinhibition would also release lower control structures in the right amygdala. In classic writings John Hughlings Jackson (1958) described this mechanism of the disorganization of the nervous system(s) as "dissolution:

> The higher nervous arrangements inhibit (or control) the lower, and thus, when the higher are suddenly rendered functionless, the lower rise in activity.

What do we now know about these higher control systems? In fact current neuroimaging research has indicated that the highest level of control (regulatory) structures in the human brain are located in frontolimbic systems of the right hemisphere.

Essential role of right frontolimbic structures in the regulation of dissociation

Note that the neuroanatomy of the right brain allows for a reciprocal connection between the highest level of the limbic system, the orbitofrontal and medial frontal cortices, and the lower in the brainstem medullary vagal systems that regulate parasympathetic hypoarousal and dissociation. A similar model is described by Phillips et al. (2003), who describe a "ventral" regulation system, including orbitofrontal cortex, insula, anterior cingulate, and amygdala. As opposed to a nonlimbic "dorsal" effortful regulation system in the dorsolateral cortex, hippocampus, and other structures involved in explicit processing of the "verbal components of emotional stimuli," this ventral system is important for the implicit identification of the emotional significance of environmental stimuli, and central to the "automatic regulation and mediation of autonomic responses to emotional stimuli and contexts accompanying the production of affective states" (p. 510).

In previous works I have described a model of dual limbic-autonomic circuits, a hierarchical sequence of interconnected limbic areas in orbitofrontal cortex, insular cortex, anterior cingulate, and amygdala. (Schore, 1994, 1996). Each component of this "rostral limbic system" interconnects with the other and with brainstem bioaminergic arousal and neuromodulatory systems, including vagal nuclei in the medulla and hypothalamic neuroendocrine nuclei that regulate the sympathetic and parasympathetic nervous systems (Schore, 1994, 2003a, b). Of particular importance are the highest levels of this vertical cortical-subcortical system, especially the orbitofrontal cortex which monitors and controls responses initiated by other brain regions and is involved in the selection and active inhibition of neural circuits associated with emotional responses (Rule, Shimamura, & Knight, 2002). This prefrontal system performs a "hot'" executive function, regulating affect and motivation via control of basic limbic system functions (Zelazo & Muller, 2002).

According to Barbas and her colleagues (2003),

> Axons from orbitofrontal and medial prefrontal cortices converge in the hypothalamus with neurons projecting to brainstem and spinal autonomic centers, linking the highest with the lowest levels of the neuraxis…Descending pathways from orbitofrontal and medial prefrontal cortices [anterior cingulate], which are linked with the amygdala, provide the means for speedy influence of the prefrontal cortex on the autonomic system, in processes underlying appreciation and expression of emotions…Repetitive activation of the remarkably specific and bidirectional pathways linking the amygdala with the orbitofrontal cortex may be necessary for conscious appreciation of the emotional significance of events.

But this top-down influence can either be excitatory or inhibitory, the latter expressed in the documented activation of the orbitofrontal cortex in defensive responses (Roberts et al., 2001). Recall Lanius et al's. (2002) conclusion that prefrontal and limbic structures underlie dissociative responses in PTSD, and Gundel et al's. (2004) proposal that the right anterior cingulate can act as an inhibitory system that triggers a down regulation of emotional processing, resulting in dissociation, i.e., an inability to integrate feelings into conscious awareness.

Indeed, this limbic-autonomic circuit is right-lateralized. The right orbitofrontal cortex, the hierarchical apex of the limbic system, comes to act as an executive control function for the entire right brain. The orbitofrontal areas of the right hemisphere are more critical to emotional

functions that those in the left (Tranel, Bechara, & Denburg, 2002), and within the orbitofrontal cortex the lateral orbital prefrontal areas are specialized for regulating positive emotional states, while medial orbitofrontal areas are specialized for processing negative emotional states (Northoff et al., 2000; Schore, 2001a). The functioning of these two limbic-autonomic circuits, one capped by the lateral orbitofrontal cortex and the other by the medial orbitofrontal cortex (which in earlier writings I termed the excitatory ventral tegmental limbic forebrain-midbrain circuit and the inhibitory lateral tegmental limbic forebrain-midbrain circuits respectively; Schore, 1994) are organized in attachment experiences of the first and second year.

Optimal maturation of this prefrontolimbic system allows for the highest level of integration of exteroceptive and interoceptive information to take place in the brain. The right orbitofrontal cortex, in conjunction with the right anterior insula, supports a representation of visceral responses accessible to awareness, and provides a substrate for subjective feeling states and emotional depth and awareness (Craig, 2004; Critchley et al., 2004). In contrast to this, recall pathological dissociation is defined in ICD-10 as a loss of "awareness of identity and immediate sensations, and control of body movements." Just as secure attachment constrains trauma and dissociation, optimal functioning of the orbitofrontal system opposes somatoform dissociation.

Furthermore, the right prefrontal cortex, the "senior executive of limbic arousal" (Joseph, 1996) is most directly linked to stress-regulatory systems (Brake et al., 2000), and therefore essential for the regulation of the hyperaroused and hypoaroused states that accompany traumatic stress. Right-sided human prefrontal brain activation occurs during acquistion of conditioned fear (Fischer et al., 2002), and this cortical-subcortical regulatory mechanism allows for orbitofrontal modulation of the right amygdala that is specialized for fear conditioning (Baker & Kim, 2004) and processing frightening faces (Whalen et al., 1998; Adolphs, Tranel, & Damasio, 2001). The right amygdala directly projects to the brainstem startle center (Bradley, Cuthbert, & Lang, 1996; Davis, 1989) and to the dorsal motor vagal nucleus (Schwaber et al., 1982), and the amygdala's connections with the dorsolateral periaqueductal gray in the brainstem mediates the defensive freeze response (Oliveira et al., 2004; Vianna et al., 2001). In this manner the right orbitofrontal cortex "organizes the appropriate cortical and autonomic response based on the implications of …sensory information for survival. The orbitofrontal cortex therefore functions as a master regulator for organization of the brain's response to threat" (Scarr, 2001, p. 78).

These data strongly suggest that an individual with an impaired or developmentally immature orbitofrontal system resulting from early relational trauma will be vulnerable to pathological dissociation under stress. LeDoux concludes that without orbital prefrontal feedback regarding the level of threat, the organism remains in an amygdala-driven defensive response state longer than necessary (Morgan & LeDoux, 1995), and that in humans, conditioned fear acquisition and extinction are associated with right hemisphere dominant amygdala function (La Bar et al., 1998). These amygdala-driven startle and fear-freeze responses would be intense, because they are totally unregulated by the orbitofrontal (and medial frontal) areas that are unavailable for the correction and adjustment of emotional responses. Indeed, neurological studies of adults confirm that dysfunction of the right frontal lobe is involved in PTSD symptomatology (Freeman & Kimbrell, 2001) and dissociative flashbacks (Berthier et al., 2001).

In classic neurological primate research, Ruch and Shenkin (1943) lesioned the orbitofrontal cortex (Brodman 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." And neurological patients with orbitofrontal damage show altered responses to a startle and a "dissociation among autonomic measures." In anticipation of and in reaction to an aversive stimulus, instead of an increase in heart rate, an adaptive defensive reaction of fight-flight, they exhibit a decrease in heart rate (Roberts et al., 2004). Recall the heart rate deceleration observed in traumatized dissociating infants and dissociating adult psychiatric patients.

In support of earlier proposals (Schore, 1994) it is now accepted that orbitofrontal maturation is experience-dependent (Neddens et al., 2001; Poeggel, Nowicki, & Braun, 2003), that human prefrontal function first emerge early in development, around the end of the first year (Happeney, Zelazo, & Stuss, 2004), and that conditions which modify early maternal variability in infancy produce "significant differences in right but not left adult prefrontal volumes, with experience-dependent asymmetric variation most clearly expressed in ventral medial cortex" (Lyons et al., 2002, p. 51). During these critical periods extensive hypometabolic states preclude optimal organization and thereby the functional capacity of the highest frontolimbic levels of the right brain. Pathological dissociation reflects an impairment of the affect regulatory functions of the higher centers in the orbitofrontal cortex. Through its connections with the ANS the orbitofrontal system is

implicated in "the representation of emotional information and the regulation of emotional processes" (Roberts et al., 2004, p. 307) and in "the conscious appreciation of the emotional significance of events" (Barbas et al., 2003). In contrast, in the dorsal vagal parasympathetic-dominant state of dissociation, the individual is cut off (dis-associated) from both the external and the internal environment, and therefore emotions are not consciously experienced.

Although dissociation is triggered by subcortical mechanisms it is regulated by higher corticolimbic centers. Pathological dissociation is the product of an inefficient frontolimbic system that can not regulate the onset and offset of the dissociative response. Rather, for long periods disinhibited lower subcortical centers, especially the right amygdala, drive the dissociative response, a mechanism that reflects a Janetian regression down a hierarchy to a state that is constricted and disunified. The integration of information from the external world and the internal world (especially "messages of safety") is a product of adequate orbitofrontal activity, and "such integration might provide a way whereby incoming information may be associated with motivational and emotional states to subserve processes such as selective attention and memory formation and retrieval." (Pandya & Yeterian, 1985, p. 51). Loss of orbitofrontal functions that maintain "the integration of past, present, and future experiences, enabling adequate performance in behavioral tasks, social situation, or situations involving survival (Lipton et al., 1999, p. 356) is reflected in pathological dissociation, defined in the DSM-IV as "a disruption in the usually integrated functions of consciousness, memory, identity, or perception of the environment."

Further speculations on the biological mechanism of dissociation

In the introduction to this chapter I cited Prueter's (2002) call for an understanding of the "primary pathophysiologic mechanism that leads to the dissociative symptoms, using neurobiological research mechanisms." Towards that end, in previous sections of this chapter I applied regulation theory to offer a model of the earliest psychobiological expression of dissociation in human infancy. I argued that this basic survival mechanism is a strategy for coping with intense states of energy-expending hyperarousal by shifting into an energy-conserving hypometabolic state, and that this state of hypoarousal, reflected in heart rate deceleration in response to stress, remains unchanged over the life span. This model is based in part on the developmental observations of Main (Main & Solomon, 1986; Main & Hesse, 1999), who asserts that the disorganization and disorientation of type "D" attachment phenotypically resembles dissociative states, and Tronick (Tronick & Weinberg, 1997; Tronick, 2004) who uses the still-face procedure as an experimental paradigm of traumatic abuse, a threatening interpersonal context that triggers "massive disengagement."

I have suggested that these paradigms used with infants under one year of age describe the same state of dissociation described by clinicians as "profound detachment" (Barach, 1991), "detachment from an unbearable situation" (Mollon, 1996), and "dissociative detachment" (Allen et al., 1998). Furthermore, the mechanism is identical – at all points in the life span the functional aspects of Janetian "extreme emotional arousal" and dissociation reflect a structural alteration in arousal systems in the brainstem associated with a loss of ventral vagal and dominance of dorsal vagal parasympathetic states. In this section I will offer further speculations about the basic biological mechanisms that underlie dissociation.

Studies of dissociation in the developmental literature report that under stress type "D" infants show "a dazed facial appearance...accompanied by a stilling of all body movement, and sometimes a *freezing* of limbs which had been in motion" (Main & Solomon, 1986). These experiences of traumatic freezing are encoded in enduring implicit-procedural memory, representing what Janet termed unconscious "fixed ideas" that cannot be "liquidated." Indeed, the relationship between freeze behavior and dissociation has been noted by authors in a number of disciplines. In psychophysiological research Porges (1997) describes a trauma-induced "immobilized state" associated with the dorsal vagal complex. In one of the most important psychiatric texts on trauma written in the last century, Henry Krystal (1988) describes a traumatic "catatonoid" affective response to "the perception of fatal helplessness in the face of destructive danger," and equates this "pattern of surrender" with the freeze response of animal states of "*cataleptic immobility*." In the trauma literature I have described this behavior in 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 (Schore, 2001a).

In parallel neurological writings Scaer (2001) postulates that dissociation "is initiated by a failed attempt at defensive/escape efforts at the moment of a life threat, and is perpetuated if spontaneous recovery of the resulting *freeze response* is blocked or truncated" (p. 84, my italics). In discussing this state of catatonic immobility and "suspended animation" he concludes,

> If deterrence of the threat through defense or fight fails, the animal enters a state of helplessness, associated by a marked increase in dorsal vagal complex tone, initiating the freeze/immobility response…The extremes of vagal parasympathetic tone as manifested in the state of dorsal vagal activation, therefore, contribute greatly to the generation of severe emotions, especially those of terror and helplessness. Although freeze/immobility states…may be useful for short-term survival, prolongation or repeated activation of that state clearly has serious implications for health and long-term survival (Scarr, 2001, p. 81)

A number of studies indicate that the freeze response is right-lateralized. 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. Basic research indicates that right parietal lesions are associated with a conditioned freezing deficit (Hogg, Sanger, & Moser, 1998), and human studies (Northoff et al., 2000) show a right lower prefronto-parietal cortical dysfunction in catatonia, a basic somatic defense mechanism associated with "immobilization of anxieties," a description very similar to dissociation.

But other studies in the developmental literature, those of Tronick, describe not freeze behavior but a collapsed state of "profound disengagement" (see Figure 2). Tronick (2004) observes not only a suspension of any spontaneous emotionally expressive facial expression or gesture, but a "dissipation of the infant's state of consciousness," that ultimately is associated with "the disorganization of many of the lower level psychobiological states, such as metabolic systems." How does this relate to the above characterized freezing? Keep in mind that the full manifestation of the fear-freeze response is a late occurring behavior in human infants, occurring in the second half of the first year. But dissociation is seen in the hypoxic human fetus (Reed et al., 1999) and soon after birth (Bergman et al., 2004).

Figure 2. Close-up of the still-face induced collapse. From Tronick (2004).

Again, clues come from studies in basic biology and neuroscience. Citing this literature Scarr observes that freeze behavior is a state of alert immobility in the presence of a predator. He points out that freeze may proceed to flight or if attacked and captured by a predator to a "*deeper state of freeze*, one associated with apparent unresponsiveness and with marked changes in basal autonomic state" (2001, p. 76, my italics). This state of helplessness, which lasts for up to 30 minutes is accompanied by marked bradycardia (heart rate deceleration) and a pronounced state of "deep" parasympathetic vagal tone. Recall Porges (1995) describes "the involuntary and often prolonged characteristic pattern of vagal outflow from the dorsal vagal nucleus" (1995, p. 228). I equate this with a deep dissociative state, which if prolonged, is the psychobiological engine of pathological dissociation.

Other studies in basic biology offer further information about the psychobiological mechanism of this deeper state of freeze. Gabrielsen and Smith (1985) have explored the physiological responses that underlie basic defenses, defined as "threat-induced behavior," in all animals. In reaction to an environmental threat (a predator) an organism can respond in various ways: it can display offensive behavior, aggression (fight), or locomotor flight (associated with a fear state). Both are associated with increased activity and heart rate tachycardia, reflective of states of sympathetic hyperarousal. In addition to the active defense of fight-flight these authors also describe two different types of parasympathetic passive defenses, freezing and paralysis. These immobile and thus passive forms of defense differ, in that freezing occurs to visual or auditory stimuli of predator approach, while paralysis

occurs in response to strong tactile stimulation by the predator.

Intriguingly, the organism is alert in freeze, yet "unconscious" in paralysis, and parasympathetic heart rate deceleration, which they term "emotional bradycardia" occurs in both. Biologists have described this state as "fear bradycardia" or "alarm bradycardia" (Jacobsen, 1979). I suggest that this differentiation of freeze vs. paralysis describes the difference between Scarr's freeze and deep freeze, and Main's type "D" freezing when the infant is "alarmed by the parent" vs. Tronick's still-face collapse, loss of postural control, and "dissipation of consciousness." In light of the fact that high levels of dorsal vagal activation are associated with potentially life-threatening bradycardia, these data strongly suggest that lack of maternal interactive repair of infant dissociative states of deep freeze would be a potent generator of psychopathogenesis. Recall, "dissociation represents an effective short-term strategy that is detrimental to long-term functioning" (Bremner & Brett, 1997).

Furthermore, Gabrielsen and Smith (1985) refer to another term for the deep freeze state – "feigned death" - a defense mechanism utilized by a number of vertebrates, amphibians, reptiles, birds, and mammals (including humans). In an animal model, the American opossum, a mild threat (the face of a human in this study) elicited freezing and a 12% decrease in heart rate. But a more severe threat (vigorous tactile shaking) induced death feigning and an immediate and more intense heart rate deceleration (46% or 100 beats per minute!). In a conception congruent with the neurobiological model of dissociation outlined in this chapter, Gabrilelsen and Smith (1985) postulate that the suddenness of the depression in heart and respiration rates strongly indicates direct neural control by higher CNS structures on the parasympathetic cardiovascular 'centres' in the medulla, and that this alteration reflects a severe decrease in oxygen consumption and body temperature.

I propose that the freeze response is a dorsal vagal parasympathetic energy-conserving state coupled with but dominant over a weaker state of energy-expending sympathetic arousal, while in the collapsed state of death feigning the two ANS components are uncoupled. Thus in the deep freeze there is no sympathetic activity (low levels of vasopressin, catecholamines, cortisol), only pure dorsal vagal activation, known to produce massive bradycardia (Cheng et al., 1999). Furthermore, the decrease in oxygen consumption in dissociative death feigning is congruent with role of the dorsal vagal system in hypoxic responses (Porges, 2001; Potter & McCloskey, 1986) and in the reptilian diving reflex, an energy conservation strategy of heart rate deceleration that acts as a "metabolic defense" (Boutiler, 2001; Guppy & Withers, 1999). This shift into hypoxia also mediates "suspended animation" in developing systems (Padilla & Roth, 2001; Teodoro & O'Farrell, 2003). These data support my model of dissociation as a hypometabolic state (Schore, 2001b).

Note the similarity of this "emotional bradycardia" to the earlier psychoneurobiological portraits of the infant's parasympathetic-driven heart rate deceleration and dissociative response to attachment trauma, Kestenberg's (1985) dead spots in the infant's subjective experience, and Powles (1992) state of conservation-withdrawal in which the stressed individual passively disengages by "the risky posture of feigning death." In the clinical literature, dissociation is characterized as "a last resort defensive strategy" (Dixon, 1998) and "a submission and resignation to the inevitability of overwhelming, even psychically deadening danger" (Davies & Frawley, 1994, p. 65).

Conclusions and implications for DSM–V

I began this chapter with a description of the concept of dissociation in the current DSM-IV and I want to end with some thoughts about the implications of this work for the next DSM. We are currently experiencing a period of rapid change within and perhaps more importantly between the theoretical and applied sciences. The DSM-V conception of dissociation should be substantially impacted by the advances in basic science and clinical knowledge that have occurred over "the decade of the brain." The following is a brief synopsis of the relevance of the interdisciplinary information outlined here for not only future research directions but for models of more effective clinical diagnosis and practice.

This chapter has stressed the importance of not only a developmental understanding of dissociation, but also a perspective that integrates biological structure and psychological function. With what we now understand about development and brain-behavior (structure-function) relationships, can we more precisely characterize the classic statement of Classen, Koopman, and Spiegel?,

> 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 (1993, p. 29, my italics).

In other words, what is the relationship of cognition and emotion to dissociation, and can we identify these

cognitive and emotional structures in known brain systems?

Cognitive structures and dissociation

Dissociation is defined in DSM-IV as a disruption in the usually integrated functions of consciousness, perception, and memory. Each of these three functions is cognitive, reflecting the dominance of cognition in current psychology, psychiatry, and neuroscience. With respect to memory, it is now well established that this cognitive function is not a single process, and this should be reflected in the definition of dissociation in next DSM. In fact studies on trauma and dissociation have been an important contributor to the distinction between declarative-explicit-semantic memory and the conscious recall of traumatic experiences, and procedural-implicit-nonverbal memory and the unconscious organization of emotional memories and storage of conditioned sensorimotor traumatic responses. According to Scaer (2001), "Although declarative memory may account for much of the arousal-based cognitive symptoms of PTSD, procedural memory provides the seemingly unbreakable conditioned link that perpetuates the neural cycle of trauma and dissociation" (p. 76).

Recent data from developmental and affective neuroscience also bear upon the importance of implicit-procedural memory in dissociation. It has been noted that "the infant relies primarily on its procedural memory systems" during "the first 2-3 years of life" (Kandel, 1999), a period of right hemispheric dominance (Chiron et al., 1997). With respect to the long-term storage of relational trauma Gaensbauer concludes, "The clinical data, reinforced by research findings, indicate that preverbal children, even in the first year of life, can establish and retain some form of internal representation of a traumatic event over significant periods of time" (2002, p. 259). This early representation is encoded in early maturing nonverbal implicit-procedural memory, before later maturing verbal explicit-declarative memory processes are operational. Such representations of attachment trauma are encoded as a "frozen whole" (Gendlin, 1970), and they include "nonverbal presymbolic forms of relating" that "protect the infant from trauma and continue to be used by patients to avoid retraumatization" (Kiersky & Beebe, 1994, p. 389), that is, the right brain defensive regulatory strategy of dissociation.

A growing body of studies now indicates that "the right hemisphere has been linked to implicit information processing, as opposed to the more explicit and more conscious processing tied to the left hemisphere" (Happaney et al., 2004, p. 7). Recall, pathological dissociative detachment "escapes conscious control and is often experienced passively, as automatic or reflexive" (Allen et al., 1998, p. 163). Although trauma seriously impairs left-lateralized declarative memory and hippocampal function, dissociative mechanisms are efficiently encoded in right-lateralized amygdala-driven implicit memory, which is primarily regulatory, automatized, and unconscious. Much has been written about the memory mechanisms of PTSD, and until recently the focus has been upon deficits in hippocampal function and impairments of conscious explicit memory. Stress-induced elevations of cortisol impair declarative memory (Kirschbaum et al., 1996), and the hippocampal dysfunction observed in PTSD is more lateralized to the left hemisphere (Mohanakrishnan Menon et al., 2003).

But current PTSD models are shifting from hippocampus to amygdala, from the explicit memory of places to the implicit memory of faces. Very recent research demonstrates that chronic stress induces contrasting patterns of dendritic remodeling in hippocampal and amygdaloid neurons, leading to a loss of hippocampal inhibitory control, as well as a gain of excitatory control by the amygdala and thereby an imbalance in HPA axis function (Vyas, Mitra, Shankaranarayana Rao, & Chattarji, 2002). This work is complimented by current clinical models of PTSD which suggest that amygdala inhibition of hippocampal function at high levels of arousal mediates the diminution of conscious explicit memory in peritraumatic events (Layton & Krikorian, 2002), but also that the amygdala is centrally involved in the consolidation of the traumatic experience and in the storage of perceptual implicit memory for trauma-related information (McNally & Amir, 1996).

It is important to note that more than explicit memory is impaired in dissociation – higher levels of implicit memory associated with intact orbital prefrontal function are also dysfunctional. J. Krystal and his colleagues are describing the disconnection that occurs under extremes of arousal between the explicit dorsal regulation system involved in the "verbal components of emotional stimuli" and the implicit ventral regulation system involved in the automatic regulation of emotional stimuli (Phillips et al., 2003). This results in manifestations of cognitive dissociation, but somatic dissociation and indeed the fundamental mechanism of pathological dissociation represents an impairment within the ventral regulation system and therefore a deficit in the implicit identification and regulation of autonomic responses and production of affective states.

I now want to point out two common misunderstandings that have confounded the dissociation literature. The first

is that consciousness is frequently defined narrowly as reflective consciousness, and correlated with left hemispheric verbal functions. But another form of consciousness exists - primary consciousness, which relates visceral and emotional information pertaining to the biological self to stored information processing pertaining to outside reality. Edelman (1989) lateralizes primary consciousness to the right brain. Somatic dissociation thus fundamentally represents a disruption of primary consciousness. A second common error is that cognition equates to conscious verbal mentation, and because of this the left hemisphere is seen as the sole domain of cognition. But this is untrue. Cognition refers to the faculty of knowledge, but this knowing can be acquired through conscious and nonconscious processes. The appraisal of information about the external and internal environment occurs through nonconscious as well as conscious mechanisms.

In fact the right brain appraisal of safety or danger in the social environment is essentially performed implicitly at very fast time frames below conscious awareness (se Schore, 2003b, 2004, in press). Thus cognition also refers to the right-lateralized social cognition of face processing, which in a relational intersubjective context allows for the appraisal of exteroceptive social cues. In addition, interoceptive sensitivity (Barrett et al., 2004), the tracking of somatovisceral information coming up from the body, is also a cognitive process. Both of these cognitive functions are operations of the right hemisphere, the locus of implicit learning (Hugdahl, 1995).

The cognitive implicit appraisals of essential stimuli in the external and internal worlds are both impaired in pathological dissociation. These data, which reflect a current shift from explicit to implicit memory and left to right hemispheres need to be incorporated into updated models of dissociation.

Right brain emotional structures and dissociation

In the present DSM clinical manifestations of dissociative disorders include amnesia for autobiographical information and derealization, definitions that like consciousness, memory, and perception reflect a heavy emphasis on cognition. However, another aspect of the current paradigm shift in psychology, psychiatry, and neuroscience is to affect and affect regulation. This recent work on the primacy of affect, a common convergence of a number of disciplines including developmental psychology, biological psychiatry, affective neuroscience, and psychoanalysis indicates that emotion needs to be reincorporated into the DSM-V definition of dissociation. The contemporary revitalization of the work of Janet (Nemiah, 1989; Putnam, 1989) clearly implies a return to a model in which “vehement emotions” and “extreme emotional arousal” are central, not secondary to cognition. A large body of converging clinical studies and experimental research suggests that more so than cognition, severe affect dysregulation of intense fear-terror, psychobiological responses to threatening contexts associated with challenges to organismic survival, are at the core of the dis-integration that occurs in the dissociative response to overwhelming traumatic experience.

The original Janetian conception of dissociation implies that the trigger for dis-integration is an appraisal of an overwhelming traumatic experience and an unbearable emotional reaction. In other words, what is dis-associated is the structural system that is centrally involved in rapidly detecting and coping with an overwhelming survival threat and in processing unbearable potentially dysregulatng emotional information. This characterization applies to the right brain, which is dominant for the reception (Adolphs et al., 1996; Anderson & Phelps, 2000; Borod et al., 1998; George et al., 1996; Lucas et al., 2003; Nakamura et al., 1999) and expression (Borod, Haywood, & Koff, 1997; Mandal & Ambady, 2004) of emotion, as well as responding to preattentive negative emotional stimuli (Kimura et al., 2004), coping with negative affects (Davidson et al., 1990; Silberman & Weingartner, 1986), and controlling vital functions that support survival and enable the organism to cope with stressors (Wittling & Schweiger, 1993).

There is now a large body of convincing evidence to show that the human threat detection system is located in the subcortical areas of the right brain, especially in the right amygdala, which is specialized for detecting “unseen fear” (Morris et al., 1999), for fear conditioning (Fischer et al., 2002), for stress and emotionally related processes (Scicli et al., 2004), and for the expression of memory of aversively motivated experiences (Coleman-Mesches & McGaugh, 1995). In a study of predator stress-activation of specifically the right amygdala and periaqueductal gray, Adamaec, Blundell, and Burton (2003) report, “these data implicate neuroplasticity in right hemispheric limbic circuitry in mediating long-lasting changes in negative affect following brief but severe stress” (p. 1264). The right amygdala is in turn regulated by the right insula, right anterior cingulate, and ultimately right orbitofrontal cortex. This latter prefrontal hierarchical apex of the limbic system is activated in “situations involving survival” (Lipton et al., 1999) and functions as “a master regulator for organization of the brain’s response to threat” (Scarr, 2001). Indeed, “the right ventral medial prefrontal cortex plays a primary role in optimizing cautious and adaptive

behavior in potentially threatening situations" (Sullivan & Gratton, 2002, p. 69).

I earlier described how secure attachment experiences allow for the maturation of the brain's major regulatory system in the right orbitofrontal cortex. Thus the psychological principle that security of the attachment bond is the primary defense against trauma-induced psychopathology is directly related to the developmental neurobiological tenet that optimal secure attachment experiences facilitate the experience-dependent maturation of a right-lateralized affect regulatory system that can efficiently modulate the extreme emotional arousal and vehement emotions of trauma. The capacity to consciously experience regulated negative (and positive) emotional states is adaptive, because affects act as internal evaluations of what is happening in an encounter with the environment (Lazarus, 1991) and allow for actual or expected changes in events that are important to the individual (Frijda, 1988).

In contrast, the relational context of a disorganized-disoriented insecure attachment acts as a growth-inhibiting environment that generates immature and inefficient orbitofrontal systems, thereby precluding higher complex forms of affect regulation. Under stress these immature prefrontal corticolimbic systems rapidly disorganize, disinhibiting lower subcortical systems that activate either states of hyperarousal or the primitive defense of dissociation that counterbalances these states. When dissociated from "top-down" orbitofrontal influences, an "exaggerated amygdala" response to masked facially expressed fearful reminders of traumatic events occurs in PTSD patients (Rauch et al., 2000). The characterological use of this "last resort defensive strategy" precludes the capacity to consciously experience affective states, and thereby forfeits their use in adaptive interpersonal and intraorganismic functioning and further emotional development.

The symptomatology of dissociation can thus be conceptualized as a structural impairment of a right brain regulatory system and the resultant functional deficit of affect regulation. The clinical principle that dissociation is detrimental to long-term functioning (Bremner & Brett, 1997) is directly related to the developmental observations that early-forming yet enduring disorganized insecure attachment associated with dissociative states is a primary risk factor for the development of mental disorders (Hesse & Main, 1999; Main, 1996), and to the neuropsychiatric observations that affect dysregulation and right hemisphere dysfunction play a prominent role in all psychiatric disorders (Cutting, 1992; Taylor et al., 1997).

Returning to Classen's dictum that individuals who lack the cognitive and emotional structures to assimilate trauma are predisposed to use dissociation, it is important to point out that efficient orbitofrontal function is essential for not only affect regulation but also for "the conscious appreciation of the emotional significance of events" (Barbas et al., 2003). While in normal subjects the right orbitofrontal cortex shows "an enhanced response to consciously perceived, as opposed to neglected fearful faces" (Winston, Vuillemer, & Dolan, 2003, p. 1827), a dysfunction of the right frontal lobe is seen in PTSD patients exhibiting dissociative flashbacks (Berthier et al., 2001).

The operations of the orbitofrontal system is also critical to the processing of cognitive-emotional interactions (Barbas, 1995). This "thinking part of the emotional brain" (Goleman, 1995) functions as an "internal reflecting and organizing agency" (Kaplan-Solms & Solms, 1996) involved in "emotion-related learning" (Rolls, Hornak, Wade, & McGrath, 1994). It acts to "integrate and assign emotional-motivational significance to cognitive impressions; the association of emotion with ideas and thoughts" (Joseph, 1996) and "presents an important site of contact between emotional or affective information and mechanisms of action selection" (Rogers et al., 1999). These data suggest that the cognitive and emotional structures that are lacking in dissociating trauma victims are located in the right orbitofrontal structure and its cortical and subcortical connections.

Furthermore, both the current DSM and ICD definitions of dissociation refer to a traumatic dis-association of a normally integrated system, but neither identify this system. In 1994 I described the unique neuroanatomical interconnectivity of the right hemisphere:

> This hemisphere, with dense reciprocal interconnections with limbic and subcortical structures (Tucker, 1981), is specialized to regulate arousal (Levy, Heller, Banich, & Burton, 1983) and to *integrate perceptual processes* (Semmes, 1968)… It contains larger cortical areas than the left of intermodal associative zones that *integrate* processing of the three main sensory modalities (Goldberg & Costa, 1981)…This right hemisphere, more so than the left, is structurally specialized for greater cross-modal *integration* (Chapanis, 1977; Tucker, 1992), perhaps due to the facts that it contains more myelinated fibers that optimize transfer across regions than the left (Gur et al., 1980), and that it is specialized to represent multiple information channels in parallel (Bradshaw & Nettleton, 1981). (Schore, 1994, p.

308, my italics)

More recent studies demonstrate that when the intracortical connections within this hemisphere are functioning in an optimal manner, it adaptively integrates cross-sensory information and thereby subserves the integration of different representational information systems (Calvert et al., 2001; Raij et al., 2000). However, under the extreme stress of both hyperarousal and hypoarousal the right cortical hemisphere loses it capacity to integrate posterior sensory processing, thus causing the disruption in the integration of perceptual information depicted in the current DSM-IV. But in addition, under these intensely stressful periods, the right brain loses it capacity to act as an integrated vertical cortical-subcortical system.

In this condition limbic-autonomic information is processed only at the lowest right amygdala level, blocked from access to higher right anterior cingulate and orbitofrontal areas, and so such "partially processed" information (Whitlock, 1967; Ludwig, 1972) can not be integrated into awareness as a conscious, subjectively experienced emotion. Such "partially processed" somatic information is expresed as what Janet termed "excessive or inappropriate physical responses" and Freud described as "bizzare physical symptoms". The clinical description of dissociation thus describes the loss of this integrative capacity of the vertically organized emotional right brain.

The right brain emotional-corporeal self and somatic dissociation

The current DSM definition of dissociation prescribes a disruption in not only consciousness, memory, and perception, but also in identity. In the contemporary traumatology literature the term identity has been supplanted by the concept of self (Schore, 1994, 2003a,b), as in van der Kolk's (1996) statement that "Dissociation refers to a compartmentalization of experience: Elements of a trauma are not integrated into a unitary whole or an integrated sense of self." Similarly, in the psychoanalytic literature Kohut (1971) postulates a shattering of the self in trauma survivors, and Krystal (1988) states that the focus of treatment in trauma survivors is integration of the self. And in the developmental literature authors posit that experiences with a traumatizing caregiver negatively impact the child's attachment security, stress coping strategies, and sense of self (Crittenden & Ainsworth, 1989; Erickson, Egeland, & Pianta, 1989).

The concept of self has also been absorbed into developmental neuroscience. Indeed, the self-organization of the right brain and the origin of the self have been an essential theme of my own writings (Schore, 1994, 2003a,b). A central principle of this work dictates that "The self-organization of the developing brain occurs in the context of a relationship with another self, another brain" (Schore, 1996). Decety and Chaminade (2003) echo this in their assertion, "The sense of self emerges from the activity of the brain in interaction with other selves." These authors also conclude that "self-awareness, empathy, identification with others, and more generally intersubjective processes, are largely dependent upon...right hemisphere resources, which are the first to develop" (p. 591). Indeed the larger neuroscience literature is also now very interested in the self. In fact there is a growing consensus that "The self and personality, rather than consciousness, is the outstanding issue in neuroscience" (Davidson, 2002). Note that this statement is relevant to the current over-emphasis on consciousness in the current DSM definition of dissociation.

It is currently thought that there are dual representations of self, one in each hemisphere. Verbal self-description is mainly a linguistic process associated with a left hemisphere advantage, while self-description in terms of affective tone is associated with a right hemisphere advantage (Faust, Kravetz, & Nativ-Safrai, 2004). This dual model is echoed in LeDoux's statement, "That explicit and implicit aspects of the self exist is not a particularly novel idea. It is closely related to Freud's partition of the mind into conscious, preconscious (accessible but not currently accessed), and unconscious (inaccessible) levels" (2002, p. 28). This dichotomy reflects the aforementioned link between the right hemisphere and nonconscious implicit processing, and the left with conscious explicit processing (Happaney et al., 2004). In support of earlier theoretical proposals on the relationship between right hemispheric operations and the implicit self (Schore, 1994), a substantial amount of current research indicates that the right hemisphere is specialized for generating self-awareness and self-recognition, and for the processing of "self-related material" (Craiket al., 1999; Decety & Chaminade, 2003; Decety & Sommerville, 2003; Fossati et al., 2004; Keenan et al., 2000, 2001; Kircher et al., 2001; Miller et al., 2001; Perrin et al., 2005; Platek, Thomson, & Gallup, 2004; Ruby & Decety, 2001).

According to Miller and his colleagues, "a nondominant frontal lobe process, one that connects the individual to emotionally salient experiences and memories underlying self-schema, is the glue holding together a sense of self" (2001, p. 821). Traumatic overwhelming emotional experiences dissolve this right frontal "glue" function that integrates the self. Keeping in mind the DSM definition of dissociation, Stuss and Alexander state that the right

prefrontal cortex plays a central role in "the appreciation, integration, and modulation of affective and cognitive information" and serves as "a specific convergence site for all of the neural processes essential to affectively personalize higher order experience of self and to represent awareness of that experience" (1999, p. 223). Clearly, the major debilitating impact of trauma is on this right-lateralized implicit system, and not the language functions of the left hemisphere.

Devinsky (2000) argues that the emotion-processing right hemisphere plays an evolutionary role in "maintaining a coherent, continuous, and unified sense of self" and in "identifying a corporeal image of self." The concept of self clearly implies more than a mental conception, but rather a psychobiolgical, right-lateralized bodily-based process. In recent research Lou et al. (2004) report "a role for the right lateral parietal region in representation of the physical Self" (p. 6831), and Decety and Chaminade (2003) show that the right inferior parietal cortex is involved in somatic experience related to awareness, and therefore participates in the sense of self. The rostral part of the posterior parietal cortex sends efferents to the insular cortex (Cavada & Goldman-Rakic, 1989). As previously mentioned the right anterior insula, in conjunction with the right orbitofrontal cortex, generates a representation of visceral responses accessible to awareness, thereby providing a somatosensory substrate for subjective emotional states experienced by the corporeal self (Critchley et al., 2004). This limbic structure, buried in the right temporal lobe, is centally involved in visceral and autonomic functions that mediate the generation of an image of one's physiological state (Craig et al., 2000).

Once again, these neurobiological data mirror the ICD-10 description of dissociation as a partial or complete loss of control of body movements. Recall Crucian's (2000) description of a right hemisphere dependent dissociation between the emotional evaluation of an event and the physiological reaction to that event, and Spitzer's (2004) observation that the dissociative symptoms of depersonalization reflects a lack of right hemispheric integration. In a study of "out-of-body" experiences, defined as episodes in which "a person's consciousness seems to become detached from the body and take up a remote viewing position," Blanke et al. (2002) report that "the experience of dissociation of self from the body is a result of failure to integrate complex somatosensory and vestibular function" (p. 269). Importantly, right medial temporal lobe activation is seen during the patient's dissociative episode (see Figure 3).

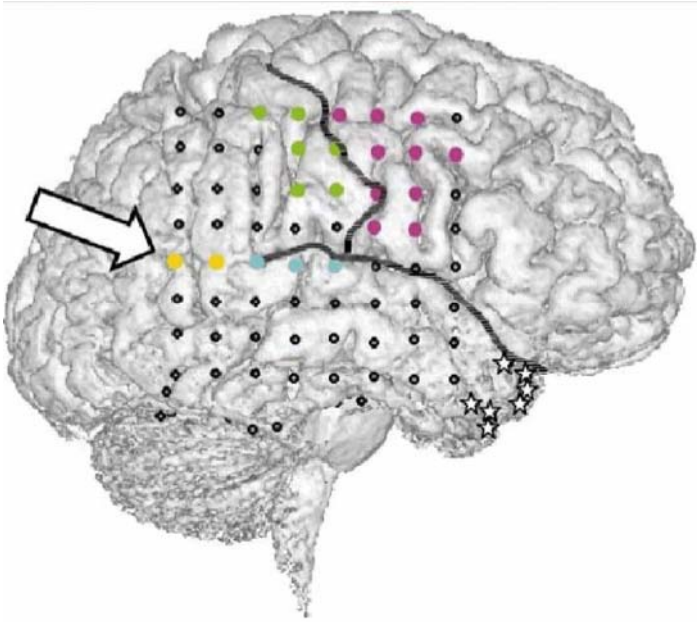

Figure 3. Electrodes stimulated at right hemispheric sites trigger depersonalization reactions in a 43-year-old woman with right temporal lobe (starred) epilepsy. Locations are magenta, motor; green, somatosensory cortex; turquoise, auditory cortex. Yellow, site at which out-of-body experience, body-part illusions and vestibular responses were induced (arrow). During these dissociative states the patient states "I see myself lying on the bed, from above, but I only see my legs and lower trunk." From Blanke et al., 2002.

The assertion by neuroscience authors that "impaired self-awareness seems to be associated predominantly with right hemisphere dysfunction" (Andelman et al., 2004, p. 831) thus describes a deficit in the right brain corporeal self during dissociative disruptions of identity. Scaer (2001) concludes that the least appreciated manifestations of traumatic dissociation are in the area of perceptual alterations and somatic symptoms. In earlier work I have offered a model by which attachment trauma alters the development of right-lateralized limbic-autonomic circuits that process visceral-somatic information and set the stage for the characterological predisposition to somatoform dissociation (Schore, 2001b, 2002). This chapter's contribution of regulation theory further elaborates this model, and strongly indicates somatoform dissociation must be incorporated into DSM-V.

This model also gives important clues for identifying psychobiological markers of this manifestation of pathological dissociation. In previous sections of this chapter I have described the hypoarousal and heart rate deceleration of dissociating traumatized human infants and adults, and presented biological data to show that this passive defense mechanism is common to all vertebrates. In this "last resort defensive strategy" significant heart rate deceleration (bradycardia, an abnormal slowness of heart

action) occurs in response to survival threat. This rapid shift from a hypermetabolic state of hyperarousal into a hypometabolic state of hypoarousal reflects a significant homeostatic alteration of brain-cardiovascular interactions through higher CNS adjustments of the sympathetic and especially the medullary dorsal vagal parasympathetic energy-conserving branches of the ANS. The activation of "the escape when there is no escape," of somatic dissociation represents a re-organization of vertical circuits in the right hemisphere, which is dominant for cardiovascular (Erciyas et al., 1999; Yoon et al., 1997) and survival (Wittling & Schweiger, 1993) functions.

In traumatizing contexts where active coping mechanisms are blocked or irrelevant and therefore non-adaptive, lateralized limbic-autonomic structures of the central autonomic network (ventromedial prefrontal cortex, anterior cingulate, insula, and amygdala) trigger an instantaneous re-organization of the vagal circuit of emotion regulation on the right side of the brain (Porges et al., 1994), specifically a shift in dominance from ventral vagal to dorsal vagal parasympathetic systems. Older basic research studies revealed orbitofrontal, cingulate, and insula control of bradycardia (Buchanan, Powell, & Valentine, 1984; Hardy & Holmes, 1988; Kaada, 1960). Tracing down this limbic-autonomic vertical circuit, each of these cortical structures, like the central nucleus of the amygdala, regulates the lateral hypothalamus (Loewy,1991); the lateral hypothalamus modulates dorsal vagal complex neurons (Jiang, Fogel, & Zhang, 2003); cardiac vagal motoneurons lateralized on right side of the medulla, down the right vagus regulate the heart (Rentero et al., 2002); and ultimately parasympathetic efferent neurons that are primarily located in the right atrial ganglionated plexus (Stauss, 2003) trigger a hypometabolic response of "emotional bradycardia."

Porges (2001) describes this same pattern of dis-organization, which occurs in "posttraumatic stress disorders and the consequences of child abuse":

> [W]hen mobilization strategies (fight-flight behaviors) are ineffective in removing the individual from the stressor and modulating stress, then the nervous system may degrade to a phylogenetically earlier level of organization…(This) may reflect a neural strategy associated with immobilization (e.g. passive avoidance, death feigning, *dissociative states*) that would require a reduction of energy resources (p. 15, my italics).

In earlier sections of this chapter I have reported a number of clinical studies that indicate heart rate deceleration (parasympathetic emotional bradycardia) is a psychobiological marker of pathological dissociation. In addition to the work of Lanius et al. (2002), peritraumatic dissociation associated with low heart rate has also been reported by Griffin, Resick and Mechanic (1997), Williams, Haines and Sale (2003), and Koopman et al. (2004). In a clinical study Schmahl and his (2002) colleagues document heart rate decline while a PTSD patient with a history of childhood physical and sexual abuse was dissociating (Figure 4).

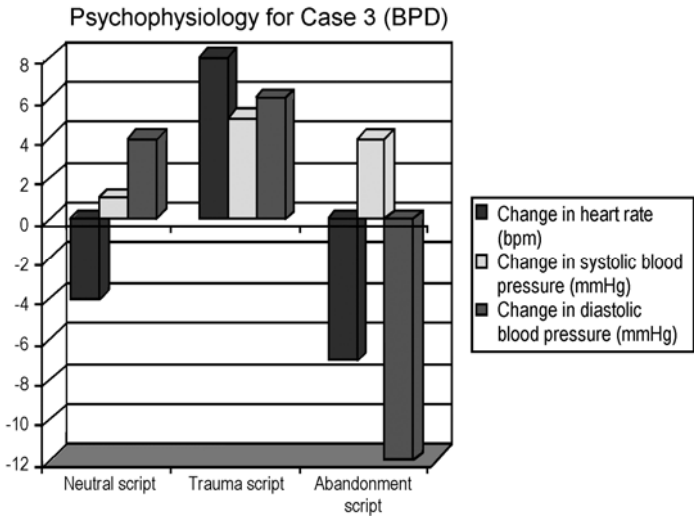

Figure 4. As this patient diagnosed with PTSD and borderline personality disorder heard her trauma script, she displayed an intense emotional reaction and her heart rate rose by 7 bpm. While listening to an abandonment script she dissociated. She had the impression that things were moving in slow motion, that things seemed unreal, and that she was watching the situation as an observer. She felt disconnected from her own body and the sense of her body felt changed. During this period her heart rate fell by 7 bpm. After the interview the dissociative state lasted for a few more minutes. From Schmahl et al. 2002.

[Note: I changed the wording a bit; you might want to change it some more, because, in reading the bars, compared to the neutral script, her heart beat rose by 11 pbm (from –4 to +7), and then fell about 14 bpm (+7 to –7) for the abandonment script, 3 bpm below the neutral script. JO'N]

Very recent studies showing that the human right insula is activated by perceptual awareness of threat (Critchley, Mathias, & Dolan, 2002) and involved in harm avoidance (Paulus et al., 2003), and that early neurological damage of the right insula is associated with abnormal bradycardia (Seeck et al., 2003) and increased right insula activity is found in adult subjects with bradycardia (Volkow et al., 2000), suggests that this structure, with its dense interconnections into the ANS, may play a key role in somatic dissociation.

The developmental and neurobiological data thus suggest that the next DSM can specify a neuropsychobiological marker for somatic dissociation – heart rate deceleration in

response to intersubjective contexts associated with nonconsciously perceived survival threat.

Early attachment trauma and the psychopathogenesis of dissociation

In the final part of this work, I would like to return to the problem of psychopathogenesis. Over 15 years ago van der Kolk and van der Hart (1989) and Speigel and Cardena (1991) returned to the work of Janet and proposed that dissociation is fundamentally a response to "overwhelming" emotional experience, particularly in childhood. In an overview of the field at the end of the last decade a number of major theoreticians in traumatology echoed this conclusion. In parallel contributions Putnam et al. asserted that "numerous clinical studies have established that elevated levels of dissociation are significantly associated with histories of antecedent trauma" (1996, p. 673), and van der Kolk and his colleagues stated that "numerous studies have demonstrated a strong relation between trauma and dissociative symptoms" (1996, p. 85). Indeed, a large body of research in the psychiatric and psychological (e.g., Bowman & Markand, 1996; Chu & Dill, 1990; Coons et al., 1989; Draijer & Langeland, 1999; Gershuny & Thayer, 1999; Irwin, 1994; Lipschitz et al., 1996; Merckelbach & Muris, 2001; Mulder et al., 1998; Nash et al., 1993; Sanders, McRoberts, & Tollefsin, 1989) and neurological (Alper et al., 1993; Kuyk et al., 1999) literatures now supports the link between childhood trauma and pathological dissociation. Until recently most of these studies have focused primarily on sexual abuse, but the role of emotional neglect, physical abuse, and now attachment trauma is being emphasized.

Although these studies are convincing, the precise mechanism of how early trauma transmutes into a later predisposition to pathological dissociation has been unresolved. The important matter of how developmental traumatic psychological experience generates deficits of later adaptive functioning is however, a central issue of the problem of psychopathogenesis. It is here that an interdisciplinary perspective can provide more detailed and complex models. Developmental psychopathology, an outgrowth of attachment theory, provides a theoretical perspective for "understanding the causes, determinants, course, sequelae, and treatment of psychopathological disorders by integrating knowledge from multiple disciplines within an ontogenetic framework" (Cicchetti, 1994, p. 286). Focusing upon longitudinal studies of the intergenerational transmission of trauma, developmental psychologists in this field have also demonstrated a link between developmental attachment trauma and dissociation (Ogawa et al., 1997). Neuropsychiatrists in this area, working in developmental traumatology have established that "the overwhelming stress of maltreatment in childhood is associated with adverse influences on brain development" (De Bellis, 1999, p. 1281).

My own work in developmental psychopathology integrates attachment theory, psychiatry, and developmental affective neuroscience in order to provide a multidisciplinary perspective to explore how attachment trauma alters the developmental trajectory of the right brain (Schore, 1994, 2003a, b). From a developmental psychology viewpoint, the profound negative psychological effect of early abuse and neglect is the generation of a disorganized-disoriented attachment, which endures over the later stages of childhood, adolescence and adulthood, and acts as a risk factor for later psychiatric disorders (Schore, 2001b). From a psychiatry viewpoint, "maltreatment-related" (Beer & De Bellis, 2002) or "pediatric" (Carrion et al., 2001) PTSD is the short-term negative effect, and a predisposition to later psychiatric disorders, the long-term effect. And from a developmental neuroscience viewpoint, the immediate detrimental impact is on critical period growth of the developing right brain, and the lasting impairment is an immature and functionally limited right brain capacity to regulate later life stressors that generate intense affective states. Note that all of these perspectives converge on the basic developmental principle that early trauma is critical to the genesis of an enduring predisposition to pathological dissociation.

In this and other contributions I have offered extensive evidence to show that relational traumatic attachment experiences are "affectively burnt in" (Stuss & Alexander, 1999) limbic-autonomic circuits of the cortical and subcortical components of the right brain during its critical period of growth. Basic research in neuroscience and neuropsychiatry now firmly supports the principles that "early adverse developmental experiences may leave behind a permanent physiological reactivity in limbic areas of the brain" (Post, Weiss, & Leverich, 1994), that emotional and social deprivation interferes with the normal development of the synaptic architecture and leads to "neurological scars" that underlie "subsequent behavioral and cognitive deficits" (Poeggel & Braun, 1996; Poeggel et al., 1999), 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). Although I have focused here on an Axis I psychiatric disorder, PTSD, in other works I have shown that this same developmental neurobiological description applies to the ontogeny of pathological dissociation in an Axis II environmentally produced psychiatric disorder, borderline personality disorder (Schore, 2003b, 2003c).

In the introduction to this further elaboration of regulation theory I cited Brown and Trimble's (2000) invocation for a more "precise definition of dissociation based on a conceptually coherent and empirically justified account of the processes underlying these phenomena." This chapter suggests that such a definiton must include a developmental model of dissociative phenomena. In total the interdisciplinary data cited here indicates that the developing brain imprints not only the overwhelming affective states that are at the core of attachment trauma but also the early appearing primitive defense used against these affects, the regulatory strategy of dissociation. The developmental principle that maltreatment in childhood is associated with adverse influences on brain development specifically refers to an impairment of higher corticolimbic modulation of the vagal circuit of emotion regulation on the right side of the brain that generates the psychobiological state of dissociation. This model accounts for the findings that somatoform dissociation is specifically associated with maternal dysfunction, and that early onset traumatization expressed as emotional neglect and abuse and interpersonal threat to the body predict somatoform dissociation. It also strongly supports Putnam's (1995) assertion that dissociation offers "very rich models for understanding the ontogeny of environmentally produced psychiatric conditions."

Although it has been one of the most controversial issues in the history of psychiatry, psychoanalysis, and psychology, there is now solid evidence converging from a number of disciplines for the direct relationship between early trauma and pathological dissociation, and the next DSM should reflect this advance in our knowledge.

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