8 Attachment Trauma and the Developing Right Brain: Origins of Pathological Dissociation

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OUTLINE

The concept of dissociation has a long history of bridging psychiatry, psychology, and neurology. Because dissociation is inextricably linked to trauma, theoretical and clinical models of dissociation have spanned the psychological and biological realms. Although the relationship between childhood trauma and dissociation was noted at the end of the 19th century, only recently has a developmental perspective been used to understand dissociation's etiological mechanisms. Dissociative phenomena are now being viewed through an interdisciplinary lens.

There is a growing appreciation of the unique contributions that developmental models can make to psychopathogenesis. As Putnam (1995) noted, a developmental view of dissociation offers "potentially very rich models for understanding the ontogeny of environmentally produced psychiatric conditions" (p. 582). In particular, I will suggest that regulation theory (Schore, 1994, 2003a, 2003b) can provide such models. Towards that end I will draw upon (1) recent findings about infant behavior from developmental psychology, (2) current data on brain development from neuroscience, (3) updated basic research in biological psychiatry on stress mechanisms, and (4) new information from developmental psychobiology on the essential functions of the autonomic nervous system in order to construct a model of the etiology and underlying psychoneurobiological mechanisms of pathological dissociation. I will use posttraumatic stress disorder as a paradigm for dissociative disorder. I will discuss the earliest expression of dissociation in human infancy—pediatric posttraumatic stress disorder—and its enduring impact on the experience-dependent maturation of the right brain, including the characterological use of dissociation at later points of interpersonal stress.

8.1 INTRODUCTION

This chapter will focus on pathological dissociation (Waller, Putnam, & Carlson, 1996). Dissociation

is defined by DSM-IV as "a disruption in the usually integrated functions of consciousness, memory, identity, or perception of the environment" (American Psychiatric Association, 1994) and by the 10th edition of the International Classification of Diseases (ICD-10) 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). Although both stress a deficit in integration, only ICD-10 includes an alteration of bodily processes. Finally, Spiegel and Cardeña (1991) characterized dissociation as "a structured separation of mental processes (e.g., thoughts, emotions, conation, memory, and identity) that are ordinarily integrated" (p. 367). Note that Spiegel and Cardeña include emotion in their definition of dissociation, whereas DSM-IV and ICD-10 did not.

The concept of dissociation can be directly traced to the work of Pierre Janet. Janet (1887, 1889) considered (pathological) dissociation to be a phobia of memories that was expressed as excessive or inappropriate physical responses to thought or memories of old traumas (see Van der Hart & Dorahy, this volume). 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 described an *abaissement du niveau mental*, a lowering of the mental level, a regression to a state that is constricted and disunified. Furthermore, Janet speculated that dissociation was the result of a deficiency of psychological energy. Due to early developmental factors, the quantity of psychological energy is lowered below a critical point, and thus individuals with pathological dissociation are deficient in binding together all their mental functions into an organized unity under the control of the self.

Following Charcot (1887), Janet also posited that early trauma is a fundamental psychopathogenic factor in the etiology 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 Freud initially considered developmental trauma to be essential to hysteria, he soon rejected this idea and posited that repression—not dissociation—was the primary psychopathogenic mechanism.

Summarizing the essentials of Janet's model, Van der Kolk, Weisaeth, and Van der Hart stated:

> 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)

In Janet's view, 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 Janet's concept of personality into contemporary terms, Van der Kolk, Van der Hart, and Marmar concluded 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).

At the dawn of modern psychiatry, many major pioneers (e.g., Charcot, Janet, Freud, Hughlings Jackson) were interested in the neurology of dissociation (i.e., its structure-function relationships). Devinsky (2000) has noted that late 19th-century clinicians linked the right hemisphere with emotion (Luys, 1881) and dissociative phenomena (Myers, 1885; Richer, 1881); he cited Jackson's (1876) work on the duality of the brain, and the role of the right hemisphere in "emotional" speech, as opposed to the left hemisphere's "voluntary expression and conscious awareness of propositional speech." Dissociative psychopathology continues to be a focus of 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).

Recently, Brown and Trimble (2000) have argued that we must move beyond a purely 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 have made similar assertions:

> 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 few authors have proposed neurobiological models of dissociation in adults. Whitlock (1967) and Ludwig (1972) suggested that the primary pathophysiological mechanism of dissociative symptoms is an attentional dysfunction that results from an increase in the corticofugal inhibition of afferent stimulation. This inhibition impairs the processing of essential information, which subsequently fails to be integrated into awareness, and thereby generates dissociative symptoms. More recently J. Krystal et al. (1998), Scaer (2001), and Nijenhuis, Van der Hart, and Steele (2002) have made contributions to the psychobiology of dissociation (see also Nijenhuis & Den Boer, 2008, this volume). Current neuroimaging research is also contributing new information about the structure-function relationships of dissociation in mature brain systems.

Several important observations about dissociation have been advanced. In psychological studies of adults, Loewenstein noted 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). In neuropsychiatric studies of adult trauma patients, Bremner and colleagues demonstrated that (1) there are two subtypes of acute trauma response, hyperarousal and dissociation (1999), (2) dissociation represents an effective short-term strategy that is detrimental to long-term functioning (Bremner & Brett, 1997), and (3) extreme stress invokes neural mechanisms that produce long-term alterations of brain functioning (Krystal et al., 1998). Finally, Meares concluded that "dissociation, at its first occurrence, is a consequence of a 'psychological shock' or high arousal" (1999, p. 1853).

I will offer evidence that each of the above observations about dissociation in adults applies to infants as well. I will argue that developmental studies offer (1) specific models of the process whereby early trauma alters the human ontogenetic trajectory and creates a predisposition for later pathological dissociation. These models, in turn, afford a deeper understanding of the neurobiological mechanisms of dissociation. I believe that attachment theory, "the dominant approach to understanding early socioemotional and personality development during the past quarter-century of research" (Thompson, 1990, p. 145), best describes the interactions among development, trauma, and dissociation. 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, 2004; Liotti, 2004; Schore, 1997). Longitudinal attachment studies have demonstrated an association between traumatic childhood events and proneness to dissociation (Ogawa et al., 1997).

Current neurobiological models of attachment focus on the formation of the implicit self system, located in the early maturing right brain (Schore, 1994, 2001a). Researchers now assert that fearful arousal and the relational modulation of that arousal lie at the heart of attachment theory, and that relational trauma triggers states of hyperarousal and dissociation in the developing brain. I will show that abuse and neglect elicit dissociative defenses in the developing infant. As such, they represent a deleterious influence during the critical growth period of cortical, limbic, brainstem, and autonomic centers in the early maturing right brain.

Janet's ideas about early trauma and dissociation are strongly supported by recent developmental studies. A traumatizing caregiver negatively impacts the child's attachment security, strategies for coping with stress, and sense of self (Crittenden & Ainsworth, 1989; Erickson, Egeland, & Pianta, 1989). There is substantial and convincing evidence that childhood trauma arrests affective development; conversely, trauma in adulthood produces a regression in affective development (H. Krystal, 1988). The most significant consequence of early relational trauma is the child's failure to develop the capacity for emotional self-regulation (Toth & Cicchetti, 1998); the child (and subsequent adult) cannot adequately regulate affective intensity and duration (Van der Kolk & Fisler, 1994). This chapter contends that these established principles of early emotional development must be incorporated into an overarching model of dissociation.

8.2 THE NEUROBIOLOGY OF SECURE ATTACHMENT

The essential task of the first year of human life is the creation of a secure attachment bond between the infant and his/her primary caregiver. Secure attachment depends upon the mother's psychobiological attunement with the infant's internal states of arousal. Through visual-facial, gestural, and auditory-prosodic communication, caregiver and infant learn the rhythmic structure of the other and modify their behavior to fit that structure, thereby cocreating a specifically fitted interaction. During the

bodily based affective communications of mutual gaze, the attuned mother synchronizes the spatiotemporal patterning of her exogenous sensory stimulation with the infant's spontaneous expressions of endogenous organismic rhythms. Via this contingent responsivity, the mother appraises the nonverbal expressions of her infant's internal arousal and affective states, regulates them, and communicates them to the infant. To accomplish this, the mother must successfully modulate nonoptimal high *or* nonoptimal 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, then attachment *stress* is an asynchrony in that interactional synchrony. In optimal interpersonal contexts, following such stress, a period of reestablished synchrony allows the child to recover his/her regulatory equilibrium. Resilience in the face of stress is an ultimate indicator of attachment security. The regulatory processes of affect synchrony in a secure attachment relationship (1) cocreate positive arousal and (2) repair states of negative arousal. Thus, attachment represents biological regulation between and within organisms.

Research supports the proposal (Schore, 1994) that the long-enduring regulatory effects of attachment are due to their impact on brain development. According to Ziabreva et al. (2003):

> [T]he mother functions as a regulator of the socioemotional 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. (p. 5334)

I have suggested that the attachment mechanism is embedded in infant-caregiver right-hemisphere-to-right-hemisphere affective transactions (Schore, 1994, 2000, 2003a, 2003b). Because (1) the human limbic system myelinates in the first year and a half (Kinney et al., 1988) and (2) the early-maturing right hemisphere (Allman et al., 2005; Bogolepova & Malofeeva, 2001; Chiron et al., 1997; Geschwind & Galaburda, 1987)—which is deeply connected into the limbic system (Tucker, 1992)—is undergoing a growth spurt at this time, attachment experiences specifically impact limbic and cortical areas of the developing right cerebral hemisphere (Henry, 1993; Schore, 1994, 2005b; Siegel, 1999; Wang, 1997).

This model accounts for a body of recent developmental neurobiological 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), and mutual gaze activates face-processing areas of the right hemisphere (Pelphrey, Viola, & McCarthy, 2004; Watanabe, Miki, & Kakigi, 2002). Spontaneous gestures that express feeling states communicated within a dyad also activate right hemispheric structures (Gallagher & Frith, 2004). With respect to prosody, 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). Finally, the human maternal response to an infant's cry is accompanied by activation of the mother's right brain (Lorberbaum et al., 2002).

8.3 THE NEUROBIOLOGY OF RELATIONAL TRAUMA

Optimal attachment communications directly affect the maturation of (1) the central nervous system (CNS) limbic system that processes and regulates social-emotional stimuli and (2) the autonomic nervous system (ANS) that generates the somatic aspects of emotion. It is important to stress that a growth-facilitating emotional environment is required for a child to develop an internal system that can adaptively regulate arousal and other psychobiological states (and thereby affect, cognition, and behavior), The good-enough mother offers her securely attached infant access to her after a separation; she tends to respond appropriately and promptly to his/her emotional expressions. She also allows high levels of positive affect to be generated during co-shared play states. Such events as these support an expansion of the child's coping capacities and illustrate why secure attachment is the primary defense against trauma-induced psychopathology.

In contrast to caregivers who foster secure attachment, abusive caregivers not only play less, but also induce enduring negative affect in the child. Such caregivers provide little protection against other environmental impingements, including that of an abusive father. This caregiver is emotionally inaccessible, given to inappropriate and/or rejecting responses to her infant's expressions of emotions and stress, and provides minimal or unpredictable regulation of the infant's states of overarousal. Instead, she induces extreme levels of stimulation and

arousal (i.e., the very high stimulation of abuse and/or the very low stimulation of neglect). And finally, because she provides no interactive repair, she leaves the infant to endure intense negative states for long periods of time.

The infant has two psychobiological response patterns to trauma: hyperarousal and dissociation (Perry et al., 1995; Schore, 1997). Beebe describes the "mutually escalating overarousal" of a disorganized attachment pair:

> Each one escalates the ante, as the infant builds to a frantic distress, may scream, and, in this example, finally throws up. In an escalating overarousal pattern, even after extreme distress signals from the infant, such as ninety-degree head aversion, arching away … or screaming, the mother keeps going. (2000, p. 436)

In this initial stage of threat, the child's alarm or startle reaction indicates activation of the infant's right hemisphere (Bradley, Cuthbert, & Lang, 1996). This, in turn, evokes a sudden increase of ANS sympathetic activity, 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 that is expressed in increased secretion of corticotropin releasing factor (CRF)—the brain's major stress hormone. CRF regulates sympathetic catecholamine activity (Brown et al., 1982). Thus, brain adrenaline, noradrenaline, and dopamine levels are significantly elevated, creating a hypermetabolic state within the developing brain. In addition, there is increased secretion of vasopressin, a hypothalamic neuropeptide that is released when the environment is perceived to be unsafe and challenging (Kvetnansky et al., 1989, 1990).

Hyperarousal is the infant's first reaction to stress. Dissociation is a later reaction to trauma, wherein the child disengages from the stimuli of the external world. Traumatized infants are observed to be "staring off into space with a glazed look":

> [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. (Tronick & Weinberg, 1997, p. 66)

Winnicott (1958) holds that a particular failure of the maternal holding environment causes a discontinuity in the baby's need for "going-on-being." Kestenberg (1985) refers to dead spots in the infant's subjective experience, an operational definition of dissociation's restriction of consciousness.

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 state of metabolic shutdown is a primary regulatory process that is used throughout the life span. In conservation-withdrawal, 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). This parasympathetic mechanism mediates the "profound detachment" (Barach, 1991) of dissociation. If early trauma is experienced as "psychic catastrophe" (Bion, 1962), then dissociation is a "detachment from an unbearable situation" (Mollon, 1996), "the escape when there is no escape" (Putnam, 1997), "a last resort defensive strategy" (Dixon, 1998).

The neurobiology of dissociative hypoarousal is different from than of hyperarousal. In this passive state of pain-numbing and pain-blunting, endogenous opiates (Fanselow, 1986) are elevated. The dorsal vagal complex in the brainstem medulla is activated, which decreases blood pressure, metabolic activity, and heart rate—despite increases in circulating adrenaline. This elevated parasympathetic arousal is a survival strategy (Porges, 1997) that allows the infant to maintain homeostasis in the face of the internal state of sympathetic hyperarousal. It is seldom acknowledged that (1) parasympathetic energy-conserving hypoarousal and (2) sympathetic energy-expending hyperarousal are both Janetian states of "extreme emotional arousal."

Although vagal tone is defined as "the amount of inhibitory influence on the heart by the parasympathetic nervous system" (Field et al., 1995), it is now known that there are two parasympathetic vagal systems. The late-developing "mammalian" or "smart" ventral vagal system in the nucleus ambiguus enables contingent social interactions via the ability to communicate with facial expressions, vocalizations, and gestures. The early developing "reptilian" or "vegetative" system in the dorsal motor nucleus of the vagus shuts down metabolic activity during immobilization, death feigning, and hiding

behaviors (Porges, 1997). As opposed to the mammalian ventral vagal complex that can rapidly regulate cardiac output to foster engagement and disengagement with the social environment, the reptilian 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).

There is now agreement that sympathetic nervous system activity manifests in tight engagement with the external environment and high level of energy mobilization and utilization, while the parasympathetic component drives disengagement from the external environment and utilizes low levels of internal energy (Recordati, 2003). Perry's description of the traumatized infant's sudden switch from high-energy sympathetic hyperarousal to low-energy parasympathetic dissociation is reflected in Porges's 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)

Similarly, H. Krystal has described the switch from sympathetic hyperaroused terror to 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)

Whereas the nucleus ambiguus exhibits rapid and transitory patterns (associated with perceptive pain and unpleasantness), the dorsal vagal nucleus exhibits an involuntary and prolonged pattern of vagal outflow. This prolonged dorsal vagal parasympathetic activation explains the lengthy "void" states that are associated with pathological dissociative detachment (Allen, Console, & Lewis, 1998).

8.4 DEVELOPMENTAL NEUROPSYCHOLOGY OF DISSOCIATION

How are the trauma-induced alterations of the developing right brain expressed in the socioemotional behavior of a traumatized toddler? Main and Solomon's (1986) classic study of attachment in traumatized infants revealed a new attachment category, Type D, an insecure-disorganized/disoriented pattern that occurs in 80% of maltreated infants (Carlson et al., 1989). Type D attachment is also associated with pre- and/or postnatal maternal alcohol or cocaine use (Espinosa et al., 2001; O'Connor, Sigman, & Brill, 1987). Hesse and Main (1999) noted that Type D disorganization and disorientation is phenotypically similar to dissociative states. Main and Solomon (1986) concluded that Type D infants have low stress tolerance and that their disorganization and disorientation indicate that the infant is alarmed by the parent. Because infants inevitably seek the parent when alarmed, Main and Solomon concluded that frightening parents placed infants in an irresolvable bind wherein they could neither approach their parents, shift their attention, nor flee. These infants are utterly unable to generate a coherent way to cope with their frightening parents.

Main and Solomon detailed the uniquely bizarre behaviors of 12-month-old Type D infants in the Strange Situation procedure. These infants displayed brief (frequently only 10 to 30 seconds) but significant interruptions of organized behavior. At such times, Type D infants may exhibit a contradictory behavior pattern such as "backing" toward 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 overrode 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 exhibit apprehension, confusion, and very rapid shifts of state during the 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 are not restricted to the infant's interactions with the mother; 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; another

FIGURE 8.1 An infant losing postural control and self-comforting in response to the mother being still-faced. From Tronick 2004.

"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. These are 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." Guedeney and Fermanian (2001) have developed an alarm distress scale that assesses the sustained withdrawal that is associated with disorganized attachment; it assesses frozen, absent facial expression; total avoidance of eye contact; immobility; absence of vocalization; absence of relating to others; and the impression that the child is beyond reach.

Dissociation in infants has also been studied with the *still-face procedure*, an experimental paradigm of traumatic neglect (see Figure 8.1). In the still-face procedure, the infant is exposed to a severe relational stressor; the mother maintains eye contact with the infant, but she suddenly inhibits all vocalization and suspends all emotionally expressive facial expressions and gestures. This triggers an initial increase of interactive behavior and arousal in the infant. According to Tronick (2004), the infant's confusion and fearfulness at the break in connection is accompanied by the idea that "this is threatening." This is rapidly followed by bodily collapse, loss of postural control, withdrawal, gaze aversion, sad facial expression, and self-comforting behavior.

Most interestingly, this behavior is accompanied by a "dissipation of the infant's state of consciousness" and a diminishment of self-organizing abilities that reflect "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). Tronick (2004) suggests that infants who have a history of chronic breaks of connections exhibit an "extremely pathological state" of emotional apathy; she

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equates this state with Spitz's concept of hospitalism and Romanian orphans who fail to grow and develop. Such infants ultimately adopt a communication style of "stay away, don't connect." This defensive stance is a very early forming, yet already chronic, pathological dissociation that is associated with loss of ventral vagal activation and dominance of dorsal vagal parasympathetic states.

The still-face induction of hyperarousal and dissociation occurs face-to-face with the mother. The mother's face is the most potent visual stimulus in the child's world; it is well known that direct gaze can mediate not only loving, but aggressive messages. Hesse and Main (1999, p. 511) described a mother's frightening behavior: "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. Both the image of this aggressive face and the associated alterations in the infant's bodily state are indelibly imprinted into limbic circuits; they are stored in the imagistic procedural memory of the visuospatial right hemisphere, the locus of implicit (Hugdahl, 1995) and autobiographical (Fink et al., 1996; Greenberg et al., 2005; Markowitsch et al., 2000) memory.

Main and Solomon (1986) noted that Type D infants often encounter a second kind of disturbing maternal behavior: a maternal expression of fear-terror. This occurs when the mother withdraws from the infant as though the *infant* were frightening; such mothers of Type D infants exhibit dissociated, trancelike, and fearful behavior. Current studies have shown a link between frightening maternal behavior, dissociation, and disorganized infant attachment (Schuengel, Bakersmans-Kranenburg, & Van IJzendoorn, 1999). In recent work, Hesse and Main observe that when the mother enters a dissociative state, a fear alarm state is triggered in the infant. The caregiver's entrance into the dissociative state is expressed as "parent suddenly completely 'freezes' with eyes unmoving, half-lidded, despite nearby movement; parent addresses infant in an 'altered' tone with simultaneous voicing and devoicing" (2006, p. 320). In describing the mother as she submits to the freeze state, they note:

> Here the parent appears to have become completely unresponsive to, or even aware of, the external surround, including the physical and verbal behavior of their infant. … [W]e observed one mother who remained seated in an immobilized and uncomfortable position with her hand in the air, blankly staring into space for 50 sec. (p. 321)

During these episodes, I suggest that 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, which is in a critical period of growth. It has been established that maternal care influences both the infant's reactivity (Menard et al., 2004) and the infant's defensive responses to threat; these "serve as the basis for the transmission of individual differences in stress responses from mother to offspring" (Weaver et al., 2004, p. 847). Because many mothers suffer from unresolved trauma, their chaotic and dysregulated alterations of state become imprinted into the developing brain and self-system of the child. This is the psychopathogenetic mechanism for the intergenerational transmission of (1) trauma and (2) dissociative defenses against overwhelming and dysregulating affective states.

8.5 RIGHT BRAIN PROCESSES AND DISSOCIATION THROUGHOUT THE LIFE SPAN

Early traumatic attachment takes place when infants and toddlers repeatedly encounter massive misattunement from caregivers who trigger (and do not repair) long-lasting intensely dysregulated states in the child. The growth-inhibiting environment of relational trauma generates dense and prolonged levels of negative affect associated with extremely stressful states of hyper- and hypoarousal. In self-defense the child severely restricts overt expression of attachment need and significantly reduces the output of the emotion-processing, limbic-centered, attachment system. When the child is 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 online for long intervals of time, periods in which the developing brain is in a hypometabolic state that is detrimental to the substantial amounts of energy required for critical period biosynthetic processes. This hypometabolic brain state (Janetian deficiency of psychological energy) causes dissociative "encoding failures" (Allen et al., 1998) in the autobiographical memory of the developing self.

Attachment trauma thus sets the stage for characterological use of primitive autoregulation—that is, pathological dissociation during subsequent stages of development. In accord with this model, (1) severe early maternal dysfunction is associated with high dissociation

in psychiatric patients (Draijer & Langeland, 1999); (2) physical abuse and parental dysfunction by the mother—not the father—is associated with somatoform dissociative symptoms (Roelofs et al., 2002); and (3) individuals with Type D attachment utilize dissociative behaviors in later stages of life (Van IJzendoorn et al., 1999). Allen and Coyne describe the characterological use of dissociation:

> 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)

This psychic-deadening defense is maladaptive not only because the individual resorts to dissociation at low levels of stress, but also finds it difficult to exit this state of conservation-withdrawal. During these episodes, the person is impermeable to attachment communications and interactive regulation. This deprives the person of input that is vital to emotional development. Dissociative detachment (Allen et al., 1998) thus becomes an attractor state whereby social intimacy is habitually deemed to be dangerous (because such intimacy is always a potential trigger of "vehement emotions"). The avoidance of emotional connections, especially those that contain novel and complex affective information, prevents emotional learning; this, in turn, precludes advances in right brain emotional intelligence (Schore, 2001a) or what Janet (1889) called "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 affect the later behavior of the self system as it develops at further stages of the life cycle? I maintain that a purely psychological conception cannot answer this question; a psychoneurobiological perspective that integrates both biological structure and psychological function is required. Research clearly indicates that "the overwhelming stress of maltreatment in childhood is associated with adverse influences on brain development" (1999, p. 1281).

During the first years of life when the right brain is growing (Trevarthen, 1996) and dominant (Chiron et al., 1997), adverse influences on brain development particularly impact the right brain (Allman et al., 2005). During this time, states of the infant brain become traits (Perry et al., 1995); thus, early relational trauma and dissociation will be imprinted and embedded into the core structure of the developing right brain. Indeed, evidence shows that early relational trauma is particularly expressed in right hemisphere deficits. 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 earlier assertions that (1) the symptoms of PTSD fundamentally reflect an impairment of the right brain (Schore, 1997; Van de Kolk, 1996) and (2) the right hemisphere is paramount in the perceptual and cognitive processing and the regulation of biological responses in PTSD patients (Spivak et al., 1998).

Thus, neurobiological research suggests that there is continuity over the life span in the expression of the coping deficits of PTSD and the use of pathological dissociation in persons who have a childhood history of relational trauma. The principle that severe attachment pathology frequently copes with Janetian "vehement emotions" via primitive modes of autoregulation can be translated into the clinical tenet that in PTSD (and other early forming severe pathologies of the self), the individual is cut off (disassociated) from experiencing intense affective states: "traumatic stress in childhood could lead to self-modulation of painful affect by directing attention away from internal emotional states" (Lane et al., 1997, p. 840). The right hemisphere is dominant not only for attachment regulation of affects, but also for attention (Raz, 2004) and pain processing (Symonds et al., 2006). Thus, the right brain strategy of dissociation represents the ultimate defense for blocking emotional pain.

This affective deficit ensues when attachment trauma produces an enduring impairment of the "affective core" (Emde, 1983), the primordial central integrating structure of the nascent self. Joseph (1992) describes this 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 (1) Devinsky's (2000) assertion that optimal right hemispheric functions allow for "a coherent, continuous, and unified sense of self," and (2) Devinsky's citation of 19th century authors who postulated a connection between right hemispheric dysfunction and dissociation.

Both developmental (Perry et al., 1995; Schore, 1997) and adult (Bremner, 1999) studies support the proposition 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). According to Gadea et al. (2005) mild to moderate negative affective experiences activate the right hemisphere, but an intense experience "might interfere with right hemisphere processing, with eventual damage if some critical point is reached" (p. 136). This damage is specifically hyperarousal-induced apoptotic cell death in the hypermetabolic right brain. Thus, via a switch into a hypoarousal, a hypometabolic state allows for cell survival at times of intense stress (Schore, 2003a).

8.5.1 Hyperarousal

Current research indicates that both hyperarousal and dissociative responses are essentially driven by right brain processes. Metzger et al. (2004) report "PTSD arousal symptoms are associated with increased right-sided parietal activation" (p. 324). Bonne et al. (2003) note that "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." They suggest that "this may reflect a component common to all survivors of trauma" (p. 1081). Similarly, Rabe et al. find that PTSD patients show a pattern of right hemisphere activation that is associated with anxious arousal during processing of trauma-specific information. In perhaps the most extensive investigation, Lanius et al. (2004) observe that PTSD patients (as opposed to traumatized patients without PTSD) who experience traumatic memories with heart rate increases (i.e., hyperarousal) show a pattern of right brain connectivity: activation of the right posterior cingulate, right caudate, right occipital, and right parietal lobe. They deduced that this right-lateralized pattern "may account for the nonverbal nature of traumatic memory in PTSD subjects" and cited other studies showing that "subjects who had experienced early trauma displayed … right dominance during memory recall."

8.5.2 Hypoarousal

Dissociation in PTSD is also centered in right brain processes. fMRI research of 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) revealed:

> 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. (Lanius et al., 2002, p. 309)

These authors concluded that "prefrontal and limbic structures underlie dissociative responses in PTSD" and stated that activation of the right superior and middle temporal gyri in dissociated PTSD patients is consistent with a corticolimbic model of dissociation. In a more recent study, Lanius et al. (2005) reported predominantly right-hemispheric frontal and insula activation in PTSD patients while they are dissociating, and concluded that patients dissociate in order to escape from the overwhelming emotions associated with the traumatic memory, and that dissociation can be interpreted as representing a nonverbal response to the traumatic memory.

Gundel et al. (2004) noted that dissociating (and alexithymic) patients "have difficulties in integrating aspects of certain neuropsychological functions, namely memories and feelings, into current awareness" and proposed 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 were reported by Spitzer et al. (2004) in a transcranial magnetic stimulation study; they argue that their data show 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, 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).

8.6 DYSREGULATION OF RIGHT-LATERALIZED LIMBIC-AUTONOMIC CIRCUITS AND DISSOCIATION

These studies reflect the ontogenetic development of an early-dysregulated system, and provide further evidence that prefrontal cortical and limbic areas, particularly of the right hemisphere, are central to dissociative response. More so than the left, the right hemisphere is densely interconnected with limbic regions and 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 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; Sullivan & Dufresne, 2006).

I suggest that dissociation, a primitive coping strategy of affect regulation, is best understood as a loss of vertical connectivity between cortical and subcortical limbic areas within the right hemisphere. In contrast, J. Krystal et al. (1998) emphasize "shifts in interhemispheric processing" and "cortical disconnectivity" between higher frontal and limbic structures. Ontogenetically, however, 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). Thus, models of early dissociative defense against organismic threat must move down the neuraxis into the brain stem that generates states of arousal.

In a congruent 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 that Scaer's reference to brain stem centers and *external and internal* cues clearly implies both top-down and bottom-up processing. 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.*" Such terms (i.e., "inner world," "experiencing ego"), however, have not been clearly defined by the dissociation literature.

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). This is reminiscent of the ICD-10 definition of dissociation: "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."

Specifically, recent findings about the autonomic nervous system, or what Jackson (1931) called the "physiological bottom of the mind," are vital to understanding 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 the limbic, sympathetic, and parasympathetic branches of the ANS (Aftanas et al., 2005; Critchley et al., 2000; Erciyas et al., 1999; Spence, Shapiro, & Zaidel, 1996; Tucker, 1992; Yoon et al., 1997). These control the somatic components of many emotional responses, especially autonomic physiological responses to social stimuli. Adaptive right-brain emotion processing depends upon an integration of the activities of the CNS and the ANS (Hagemann, Waldstein, & Thayer, 2003).

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

Benarroch (1997) describes such CNS-ANS limbic-autonomic circuits 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's model, Benarroch's CAN is a bidirectional hierarchical system. Benarroch, however, focuses more on higher limbic structures than lower brain stem structures. The CAN is composed of (1) limbic areas in the ventromedial (orbital) prefrontal cortex, anterior cingulate, insula, and amygdala, (2) diencephalic areas in the hypothalamus, (3) brain stem structures in the periaqueductal grey matter, and (4) 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 completely uncoupled or rigidly coupled, the individual is less able to dynamically and adaptively assemble the components of the network to meet an environmental challenge, thereby displaying deficits in emotional expression and affect regulation (Demaree et al., 2004). This finding leads back to the problem of psychopathogenesis—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; importantly, the assembly of these limbic-autonomic circuits (Rinaman, Levitt, & Card, 2000) is experience dependent (Schore, 1994, 2001a). These experiences are provided by attachment transactions of the first and second year, during which the primary caregiver provides complex interpersonal stimuli and interactive regulation of the infant's core systems of central and autonomic arousal. Optimal environments promote secure attachments that facilitate 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).

Under stress, this complex system manifests itself as 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 evident when an organism responds alertly and adaptively to a personally meaningful (especially social) stressor, yet promptly returns to the relaxed state of autonomic balance as soon as the context is appraised as safe. Thus, the ANS is not only sensitive to environmental demands and perceived stresses and threats, but will also, in a predictable order, rapidly reorganize to different neural-mediated states (Porges, 2001).

In contrast to this healthy developmental scenario, traumatizing primary caregivers amplify the infant's states of hyperarousal and/or dissociative hypoarousal. This relational intersubjective context inhibits the experience-dependent maturation of CNS-ANS links (which are more extensive on the right side of the brain). In this manner, dysregulation of the developing right brain is associated in the short term with traumatic attachment and in the long term with the psychopathogenesis of dissociation. An extensive apoptotic 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. In somatoform dissociation there is a lack of integration of sensorimotor experiences, reactions, and functions of the individual and his/her self-representation (Nijenhuis, 2000). Recall Devinsky's (2000) assertion: optimal right hemispheric functions allow for the operations of "a coherent, continuous, and unified sense of self."

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) is unable to adapt to stress; in fact, the coping limitations of pathological dissociation are essentially defined by these systems' overly rigid and continuing inhibition of certain internal systems. In other words, dissociation reflects the inability of the right brain cortical-subcortical system to (1) recognize and co-process exteroceptive information from the relational environment and (2) on a moment-to-moment basis integrate this information from moment to moment with interoceptive information from the body. 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 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); this may account for the basal hyperarousal and high 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; this would account for the low heart rate of dissociative hypoarousal.

The disassociation of higher corticolimbic areas of the CAN internal regulation system and Porges's right brain circuit of emotion regulation precludes (1) top-down control of lower brain stem and autonomic functions and (2) adaptive integration of CNS exteroceptive and ANS interoceptive information processing. This disinhibition releases lower control structures in the right amygdala via a mechanism that Hughlings Jackson (1958) called *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 know about higher control systems? Current neuroimaging research indicates that the highest level of regulatory control structures in the human brain are located in frontolimbic systems of the right hemisphere.

8.6.1 The 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 brain stem medullary vagal systems that regulate parasympathetic hypoarousal and dissociation. A similar model is proposed by Phillips et al. (2003), who described 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 is central to the "automatic regulation and mediation of autonomic responses to emotional stimuli and contexts that accompany the production of affective states" (p. 510).

I have described a model of dual limbic-autonomic circuits, a hierarchical sequence of interconnected limbic areas in the orbitofrontal cortex, insular cortex, anterior cingulate, and amygdala (Schore, 1994, 1996). Each component of this "rostral limbic system" interconnects with the other and with brain stem 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, 2003b). 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.

This top-down influence can either be excitatory or inhibitory; the latter expressed in the documented activation of the orbitofrontal cortex during defensive responses (Roberts et al., 2001). Recall Lanius's et al. (2002) conclusion that prefrontal and limbic structures underlie dissociative responses in PTSD, and Gundel's et al. (2004) proposal that the right anterior cingulate can act as an inhibitory system that down-regulates 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, exercises executive control over the entire right brain. Right orbitofrontal areas are more critical to emotional functions than left orbitofrontal areas (Tranel, Bechara, & Denburg, 2002). 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 by the 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. 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, recall that 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, so does optimal functioning of the orbitofrontal system oppose 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, is essential for the regulation of the hyperaroused and hypoaroused states that accompany traumatic stress. During the acquisition of conditioned fear (Fischer et al., 2002), the right prefrontal brain is activated. This cortical-subcortical regulatory mechanism allows for orbitofrontal modulation of the right amygdala that is specialized for fear conditioning (Baker & Kim, 2004; Moses et al., 2007) and processing frightening faces (Whalen et al., 1998; Adolphs, Tranel, & Damasio, 2001). The right amygdala directly projects to the brain stem 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 brain stem mediate 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" (Scaer, 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. 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). In humans, conditioned fear acquisition and extinction are associated with right-hemisphere-dominant amygdala function (La Bar et al., 1998). Such amygdala-driven startle and fear-freeze responses are intense because they are totally unregulated by the orbitofrontal (and medial frontal) cortex. Indeed, 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"). Neurological patients with orbitofrontal damage show a "dissociation among autonomic measures" and an altered response to a startle. Such patients show a *decrease* in heart rate in anticipation of, or in response to, an aversive stimulus (Roberts et al., 2004). This is reminiscent of the deceleration of

heart rate that has been observed in traumatized dissociating infants and dissociating adult psychiatric patients.

In support of earlier proposals (Schore, 1994), it is now well established that orbitofrontal maturation is experience dependent (Neddens et al., 2001; Poeggel, Nowicki, & Braun, 2003), that human prefrontal functions emerge around the end of the first year (Happeney, Zelazo, & Stuss, 2004), and that conditions that 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 optimal 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 "the conscious appreciation of the emotional significance of events" (Barbas et al., 2003). In the dorsal vagal parasympathetic-dominant state of dissociation, however, the individual is cut off (dis-associated) from both the external and the internal environment and, therefore, emotions are not consciously experienced.

Although triggered by subcortical mechanisms, dissociation is regulated by higher corticolimbic centers. Pathological dissociation is the product of an inefficient frontolimbic system that cannot regulate the onset and offset of the dissociative response. Instead, for long periods of time, disinhibited lower subcortical centers (especially the right amygdala) drive the dissociative response; this reflects a Janetian regression to a constricted and disunified state. Adequate orbitofrontal activity is needed to integrate information from the external world and the internal world (especially "messages of safety"); "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: "a disruption in the usually integrated functions of consciousness, memory, identity, or perception of the environment" (APA, 1994). Indeed, patients using pathological dissociation who experience severe alterations of consciousness and loss of identity—dissociative identity disorder—show significant reduction of blood flow and therefore hypoactivation of the orbitofrontal cortices (Şar et al., in press).

8.7 FURTHER SPECULATIONS ON THE BIOLOGICAL MECHANISM OF DISSOCIATION

As previously noted, Prueter (2002) has called for an understanding of the "primary pathophysiologic mechanism that leads to the dissociative symptoms, using neurobiological research mechanisms." Towards that end, I have used regulation theory to offer a model of the earliest psychobiological expression of dissociation in human infancy. I argued that dissociation is a basic survival mechanism for coping with intense states of energy-expending hyperarousal by shifting into an energy-conserving hypometabolic state. This regulation strategy of hypoarousal, which is reflected in heart rate deceleration in response to stress, remains unchanged over the life span. This model is based in part on (1) Main's observations (Main & Solomon, 1986; Main & Hesse, 1999, 2006; i.e., that the disorganization and disorientation of type D attachment phenotypically resembles dissociative states), and (2) Tronick's (Tronick & Weinberg, 1997; Tronick, 2004) still-face procedure—a threatening interpersonal context that triggers "massive disengagement."

I have suggested that these paradigms describe the same state of dissociation that clinicians have described as "profound detachment" (Barach, 1991), "detachment from an unbearable situation" (Mollon, 1996), and "dissociative detachment" (Allen et al., 1998). 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 brain stem 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.

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). 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 from various

disciplines. In psychophysiological research, Porges (1997) described 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) described a traumatic "catatonoid" affective reaction to "the perception of fatal helplessness in the face of destructive danger," and equates this "pattern of surrender" with the "cataleptic immobility" of animals. In the trauma literature, I have described (1) the "frozen watchfulness" of the abused child who waits warily for parental demands, responds quickly and compliantly, and then returns to her previous vigilant state, and (2) the "frozen state" of speechless terror seen in adult PTSD patients (Schore, 2001a).

In 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):

> 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. (Scaer, 2001, p. 81)

Several studies indicate that the freeze response is right lateralized. Freezing in primate infants, which is elicited by eye contact, correlates with extreme right frontal EEG activity and high basal cortisol levels (Kalin et al., 1998). Right parietal lesions in rats are associated with a conditioned freezing deficit (Hogg, Sanger, & Moser, 1998). In human catatonia, a basic somatic defense mechanism associated with "immobilization of anxieties," there is a right lower prefronto-parietal cortical dysfunction (Northoff et al., 2000).

But other studies in the developmental literature, those of Tronick, describe not freeze behavior but a collapsed state of "profound disengagement" (see Figure 8.2). Tronick (2004) observed both a suspension of spontaneous emotional expression and gesture, and a "dissipation of the infant's state of consciousness" that is associated with "the disorganization of many of the lower level psychobiological states, such as metabolic systems." How does this relate to freezing? Keep in mind that the full manifestation of the fear-freeze response is a late-occurring behavior; in human infants, it occurs 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).

Again, clues come from studies in basic biology and neuroscience. Citing this literature, Scaer states that freeze behavior is a state of alert immobility in the presence of a predator. He points out that a freeze may be succeeded by flight or, if attacked and captured by a predator, by 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 lasts for up to 30 minutes, and is accompanied by marked bradycardia (heart rate deceleration) and a pronounced state of "deep" parasympathetic vagal tone. Recall that Porges (1995) described an "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.

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 (i.e., "threat-induced behavior") in all animals. In reaction to an environmental threat (a predator), an organism can respond in various ways: the organism may fight or flee in fear. Both responses are associated with tachycardia and increased activity, reflective of sympathetic hyperarousal. Gabrielson and Smith describe two *active*

FIGURE 8.2 Close-up of the still-face induced collapse. From Tronick 2004.

defenses (i.e., ight or flight) and two passive defenses (i.e., freezing and paralysis). The passive, immobile defenses differ; freezing occurs in response to visual or auditory stimuli of a predator's approach, whereas paralysis occurs in response to strong tactile stimulation by the predator.

Intriguingly, the organism is alert during a freeze, but "unconscious" during paralysis; parasympathetic heart-rate deceleration, which they term *emotional bradycardia*, occurs in both. Biologists call this *fear bradycardia* or *alarm bradycardia* (Jacobsen, 1979). I suggest that the differentiation of freeze versus paralysis is the same difference as (1) Scaer's freeze versus deeper state of freeze, and (2) Main's type D freezing when the infant is "alarmed by the parent" versus Tronick's still-face collapse, loss of postural control, and "dissipation of consciousness." Because high levels of dorsal vagal activation are associated with dangerous bradycardia, these data strongly suggest that the mother's failure to repair infant dissociative states of deep freeze would be a potent generator of psychopathogenesis. Recall Bremner and Brett's (1997) caution: "dissociation represents an effective short-term strategy that is detrimental to long-term functioning."

Gabrielsen and Smith (1985) discussed another term for the deep freeze state—*feigned death*—a defense mechanism that is utilized by a number of vertebrates, amphibians, reptiles, birds, and mammals (including humans). A mild threat (the face of a human in this study) to the American opossum elicited freezing and a 12% decrease in heart rate. A more severe threat (vigorous tactile shaking), however, induced death feigning and a stunning 46% decrease of heart rate deceleration. In a conception that is congruent with the neurobiological model of dissociation outlined in this chapter, Gabrielsen and Smith (1985) have postulated that (1) the sudden depression in heart rate and respiration strongly indicates that higher CNS structures are directly controlling the parasympathetic cardiovascular "centres" in the medulla and (2) this alteration reflects a severe decrease in oxygen consumption and body temperature.

I propose: (1) the freeze response is a dorsal vagal parasympathetic energy-conserving state that is coupled with, but dominant over, a weaker state of energy-expending sympathetic arousal; and (2) during 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) and pure dorsal vagal activation that produces massive bradycardia (Cheng et al., 1999) and a hypometabolic state. This decrease in oxygen consumption during dissociative death feigning is congruent with the role of the dorsal vagal system in hypoxic responses (Porges, 2001; Potter & McCloskey, 1986) and with the reptilian diving reflex, an energy conservation strategy of heart rate deceleration that acts as a "metabolic defense" (Boutiler, 2001; Guppy & Withers, 1999).

Parasympathetic vagal tone also increases "during entrance in hibernation, a long lasting disengagement from the external environment characterized by decreases in heart rate, breathing frequency, and metabolic rate" (Recordati, 2003, p. 4). The hypometabolic changes in brain plasticity (Von der Ohe et al., 2006) and in mitochondrial energy generation (Eddy et al., 2006) during the hibernation state of torpor (apathy, low responsiveness) may thus be directly related to the neurobiological mechanism of dissociation. 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), a Janetian deficiency of psychological energy.

Note the similarity of this "emotional bradycardia" to (1) the earlier psychoneurobiological portraits of the infant's parasympathetic-driven heart rate deceleration and dissociative response to attachment trauma, (2) Kestenberg's (1985) dead spots in the infant's subjective experience, and (3) Powles's (1992) state of conservation-withdrawal in which the stressed individual passively disengages by "the risky posture of feigning death." The clinical literature refers to dissociation 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).

8.8 CONCLUSIONS AND IMPLICATIONS FOR DSM–V

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 during "the decade of the brain."

With what we now understand about development and brain behavior (structure-function) relationships, can we now 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)

In other words, how do cognition and emotion relate to dissociation? Can we now locate these cognitive and emotional structures in known brain systems?

8.8.1 Cognitive Structures and Dissociation

DSM-IV defines dissociation as a disruption in the usually integrated functions of consciousness, perception, and memory. It is now well established that memory is not a single process; DSM-V's definition of dissociation should reflect this fact. In fact studies on trauma and dissociation have made important contributions to the distinction between declarative-explicit-semantic memory (i.e., conscious recall of traumatic experiences) and procedural-implicit-nonverbal memory (i.e., unconscious organization of emotional memories and storage of conditioned sensorimotor traumatic responses). According to Scaer, "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" (2001, p. 76).

Recent data from developmental and affective neuroscience reflect the importance of implicit-procedural memory in dissociation. Kandel (1999) has noted that "the infant relies primarily on its procedural memory systems" during "the first 2-3 years of life," a period of right hemispheric dominance (Chiron et al., 1997). Relational trauma can be stored at an early age: "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" (Gaensbauer, 2002, p. 259). This early representation is encoded in nonverbal implicit-procedural memory that matures well before verbal explicit-declarative memory does. Such representations of attachment trauma are encoded as a "frozen whole" (Gendlin, 1970); 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 show 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 that is primarily regulatory, automatized, and unconscious. Research on the memory mechanisms of PTSD has recently focused on deficits in hippocampal function and impairments of conscious explicit memory. Stress-induced elevations of cortisol impair declarative memory (Kirschbaum et al., 1996). Hippocampal dysfunction in PTSD is more lateralized to the left hemisphere (Mohanakrishnan Menon et al., 2003).

PTSD models are now shifting from the hippocampus to the amygdala, from explicit memory of places to implicit memory of faces. Chronic stress induces contrasting patterns of dendritic remodeling in hippocampal and amygdaloid neurons, leading to (1) a loss of hippocampal inhibitory control, (2) a gain of excitatory control by the amygdala, and (3) a resulting imbalance in HPA functioning (Vyas, Mitra, Shankaranarayana Rao, & Chattarji, 2002). Recent clinical models of PTSD suggest that the amygdala inhibits hippocampal functioning during high levels of arousal, thereby mediating a diminution of explicit memory for peritraumatic events (Layton & Krikorian, 2002). McNally and Amir (1996) argue 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.

It is important to note that dissociation not only impairs explicit memory, but also impairs higher levels of implicit memory. 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 disconnection produces cognitive dissociation. On the other hand, somatic dissociation—indeed the fundamental mechanism of pathological dissociation itself—reflects an impairment in the ventral regulation system and, thus, a deficit in the implicit identification and regulation of autonomic responses and the production of affective states.

Two common misunderstandings have confounded the dissociation literature. The first common misunderstanding is to define consciousness narrowly as (1) reflective consciousness and (2) correlated with left hemispheric verbal functions. In fact, another form of consciousness exists; primary consciousness relates emotional and visceral information about the biological self to information about outside reality. Edelman (1989) claims that primary consciousness is lateralized to the right brain. Thus, *somatic dissociation represents a disruption of primary*

consciousness. The second common misunderstanding is to equate cognition with conscious verbal mentation and to view the left hemisphere as the sole domain of cognition. This is untrue. Cognition is the faculty of knowledge, but knowing can be both conscious and nonconscious. Information about external and internal environments is appraised via nonconscious as well as conscious mechanisms.

In fact, right brain appraisal of threat in the social environment is performed implicitly and very quickly, below conscious awareness (see Schore, 2003b, 2004, 2005a). Thus, cognition includes right-lateralized social cognition of faces; this allows for the appraisal of exteroceptive social cues in a relational intersubjective context. Interoceptive sensitivity (Barrett et al., 2004), the tracking of somatovisceral information coming up from the body, is also a cognitive process. Both exteroceptive processing of social cues and interoceptive sensitivity to the body are cognitive operations of the right hemisphere, the locus of implicit learning (Hugdahl, 1995).

Pathological dissociation impairs implicit cognitive appraisal of the external world and the internal world. New models of dissociation must reflect these findings that shift the emphasis from explicit to implicit memory and from left hemisphere to right hemisphere.

8.8.2 Right Brain Emotional Structures and Dissociation

In DSM-IV, the clinical manifestations of dissociation include derealization and amnesia for autobiographical information and derealization, phenomena that reflect a heavy emphasis on cognition. However, psychiatry, psychology, and neuroscience are now emphasizing the primacy of affect and affect regulation. This convergence suggests that DSM-V should (re)incorporate emotion into the definition of dissociation. The contemporary revitalization of the work of Janet (Nemiah, 1989; Putnam, 1989; Van der Hart, Nijenhuis, & Steele, 2006) clearly implies a return to a model of dissociation in which "vehement emotions" and "extreme emotional arousal" are central, rather than secondary, to cognition. A large body of converging clinical and experimental research suggests that *severe affect dysregulation lies at the core of the disintegration that occurs in the dissociative response to overwhelming traumatic experience.*

The original Janetian concept of dissociation implies that the trigger for disintegration is an unbearable emotional reaction and an appraisal that the experience is overwhelming. What is disassociated is a structural system that rapidly detects, processes, and copes with unbearable emotional information and overwhelming survival threat. 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).

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-related stress-activation of the right amygdala and periaqueductal gray, Adamaec, Blundell, and Burton (2003) reported findings that "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 regulated by the right insula, right anterior cingulate, and right orbitofrontal cortex; this 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" (Scaer, 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).

Earlier in this chapter, I showed that secure attachment experiences allow for optimal maturation of the right orbitofrontal cortex. Accordingly, the psychological principle that secure attachment is the primary defense against trauma-induced psychopathology is directly related to the developmental neurobiological tenet that healthy 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 profoundly adaptive. Affects provide an internal evaluation of our encounters with the environment (Lazarus, 1991); they 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). Characterological use of this "last-resort defensive strategy" precludes the capacity to consciously experience affective states, thereby forfeiting their adaptive use in interpersonal and intraorganismic functioning and further emotional development.

The symptomatology of dissociation reflects a structural impairment of a right brain regulatory system and its accompanying deficiencies 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 (i.e., that individuals who lack the cognitive and emotional structures to assimilate trauma are predisposed to dissociation), it is important to note that efficient orbitofrontal function is essential for "the conscious appreciation of the emotional significance of events" (Barbas et al., 2003). 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); in PTSD patients that exhibit dissociative flashbacks, their right frontal lobe is dysfunctional (Berthier et al., 2001).

The orbitofrontal system is also critical for processing 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) that is 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 dissociating trauma victims' deficient cognitive and emotional structures are located in the right orbitofrontal structure and its cortical and subcortical connections.

The DSM and ICD definitions of dissociation both refer to a dis-association of a normally integrated system, but neither the DSM nor the ICD 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)

Recent studies demonstrate that when the intracortical connections within this hemisphere are functioning in an optimal manner, the hemisphere 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 its capacity to integrate posterior cortical sensory processing, thus causing the disruption in the integration of perceptual information depicted in the current DSM-IV. Moreover, under these intensely stressful periods, the right brain also loses its capacity to act as an integrated vertical cortical-subcortical system.

When this happens, limbic-autonomic information is processed only at the lowest right amygdala level and blocked from access to higher right anterior cingulate and orbitofrontal areas. Such "partially processed" information (Whitlock, 1967; Ludwig, 1972) cannot be integrated into awareness as a conscious, subjectively experienced emotion. Instead, such "partially processed" somatic information is expressed as what Janet termed "excessive or inappropriate physical responses" and Freud described as "bizarre physical symptoms." In short, *dissociation* refers to the loss of the integrative capacity of the vertically organized emotional right brain.

8.8.3 The Right Brain Emotional-Corporeal Self and Somatic Dissociation

DSM-IV uses the term *identity* in its definition of dissociation. The contemporary traumatology literature prefers to use the term *self* (Schore, 1994, 2003a, 2003b). For example, "Dissociation refers to a compartmentalization of experience: Elements of a trauma are not integrated into a unitary whole or an integrated sense of self" (Van der Kolk, 1996). Similarly, in the psychoanalytic literature Kohut (1971) postulates that trauma survivors have a shattered self; Krystal (1988) states that the focus of treatment for trauma survivors is integration of the self. Developmentalists contend that traumatizing caregivers 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, 2003b). A central principle of my 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: "The sense of self emerges from the activity of the brain in interaction with other selves." They 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 neuroscience literature is also very interested in the self. There is a growing consensus that "The self and personality, rather than consciousness, is the outstanding issue in neuroscience" (Davidson, 2002). Note the relevance of this statement to DSM-IV's overemphasis on consciousness in its 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" (Craik et al., 1999; Decety & Chaminade, 2003; Decety & Sommerville, 2003; Feinberg & Keenan, 2005; Fossati et al., 2004; Keenan et al., 2000, 2001; Kircher et al., 2001; Miller et al., 2001; Molnar-Szakacs et al., 2005; 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 emotional experiences dissolve the right frontal "glue" that integrates the self. Similarly, 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). The major debilitating impact of trauma is clearly on this right-lateralized implicit system—*not* the language functions of the left hemisphere.

Devinsky (2000) argues that the right hemisphere plays an ontogenetic role in "maintaining a coherent, continuous, and unified sense of self" and in "identifying a corporeal image of self." This concept of self is not just a mental one; it is a psychobiological, right-lateralized bodily-based process. Lou et al. (2004) report "a role for the right lateral parietal region in representation of the physical Self" (p. 6831); Decety and Chaminade (2003) show that the right inferior parietal cortex is involved in somatic experience that is 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 noted above, the right anterior insula and the right orbitofrontal cortex jointly generate a representation of visceral responses that is accessible to awareness; this provides a somatosensory substrate for subjective emotional states that are experienced by the corporeal self (Critchley et al., 2004). This right limbic structure is centrally involved in visceral and autonomic functions that mediate the generation of an image of one's physiological state (Craig et al., 2000).

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 reflect a lack of right hemispheric integration. In a study of "out-of-body" experiences (i.e., 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 8.3).

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Thus, the assertion that "impaired self-awareness seems to be associated predominantly with right hemisphere dysfunction" (Andelman et al., 2004, p. 831) refers to a deficit in the right brain corporeal self during dissociative disruptions of identity. Scaer (2001) contends that the least appreciated manifestations of traumatic dissociation are 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 elaborates this model and strongly indicates that somatoform dissociation should be incorporated into DSM-V.

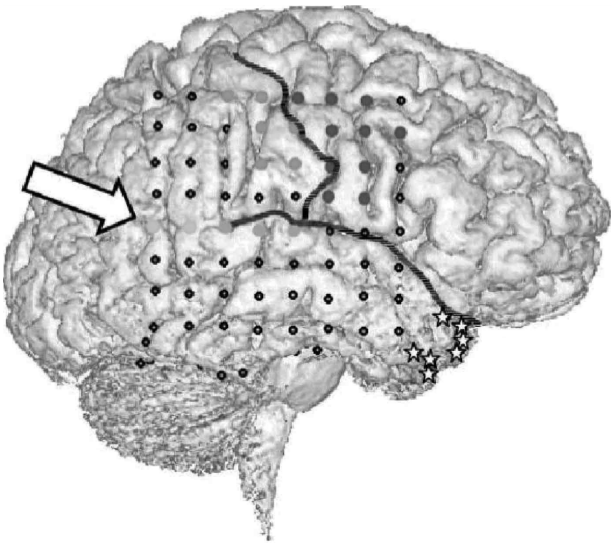

FIGURE 8.3 Electrodes stimulated at right hemispheric sites trigger depersonalization reactions in a 43-year-old woman with right temporal lobe (starred) epilepsy. Locations are magenta, motor; green, somatosensory cortex; turquoise, auditory cortex. Yellow, site at which out-of-body experience, body-part 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.

This model also gives important clues for identifying psychobiological markers of somatoform dissociation. I have described the hypoarousal and heart rate deceleration of dissociating human infants and adults. In addition I have also presented biological data to show that this passive defense mechanism is common to all vertebrates. In this "last resort defensive strategy," bradycardia 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" (i.e., somatic dissociation) represents a reorganization 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, lateralized limbic-autonomic structures of the central autonomic network (ventromedial prefrontal cortex, anterior cingulate, insula, and amygdala) trigger an instantaneous reorganization 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. Bradycardia is controlled by orbitofrontal, cingulate, and insula cortices (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 the 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."

This pattern of dis-organization, which also occurs in "posttraumatic stress disorders and the consequences of child abuse," is described by Porges (2000):

> [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)

I have cited several clinical studies that indicate that parasympathetic emotional bradycardia is a psychobiological marker of pathological dissociation. Peritraumatic dissociation associated with low heart rate has been reported by Griffin, Resick, and Mechanic (1997), Lanius et al. (2002), Koopman et al. (2004), and Williams, Haines, and Sale (2003). In a clinical study, Schmahl and colleagues (2002) documented a heart rate decline while a PTSD patient with a history of childhood abuse was dissociating (Figure 8.4).

Very recent studies have shown that the human right insula is activated by perceptual awareness of threat (Critchley, Mathias, & Dolan, 2002), anticipation of emotionally aversive visual stimuli (Simmons et al., 2004) and harm avoidance (Paulus et al., 2003). In normal functioning, the right insula supports a representation of visceral responses accessible to awareness (Critchley et al., 2004). On the other hand, neurological damage of the right insula in infancy is associated with abnormal bradycardia (Seeck et al., 2003). Increased right insula activity is also found in adult subjects with bradycardia (Volkow et al., 2000). These studies suggest that the right insula may play a key role in somatoform dissociation.

In short, developmental and neurobiological data suggest that DSM-V can specify a neuropsychobiological marker for somatic dissociation—namely, heart rate deceleration in response to intersubjective contexts that are associated with nonconsciously perceived survival threat.

8.8.4 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 Spiegel and Cardeña (1991) returned to the work of Janet, proposing that dissociation is a response to "overwhelming" emotional experience, particularly in childhood. At the end of the last decade, several major theoreticians in traumatology echoed this conclusion. 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). Van der Kolk and 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

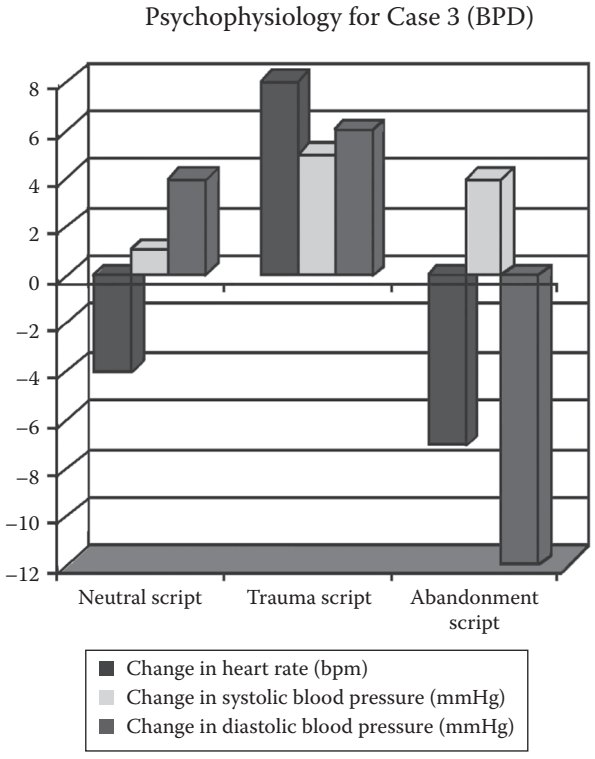

FIGURE 8.4 As this patient diagnosed with PTSD and borderline personality disorder heard her trauma script, she displayed an intense emotional reaction and her heart rate rose by 11 bpm. While listening to an abandonment script she dissociated. She had the impression that things were moving in slow motion, that things seemed unreal, and that she was watching the situtation as an observer. She felt disconnected from her own body and the sense of her body felt changed. Duing this period her heart rate fell by 14 bpm. After the interview the dissociative state lasted for a few more minutes. From Schmahl et al. 2002.

& 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.

Although these studies are convincing, the precise psychopathogenetic mechanism by which early trauma produces a later predisposition to pathological dissociation has not been identified. The more general developmental question of how early traumatic psychological experience generates deficits of later adaptive functioning is, in fact, the central issue of psychopathogenesis. Here, an interdisciplinary perspective can provide more detailed and complex models. Developmental psychopathology 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). Developmental psychologists have demonstrated a strong link between early attachment trauma and dissociation (Ogawa et al., 1997; Carlson, Yates, & Sroufe, this volume; Dutra, Bianchi, Lyons-Ruth, & Siegel, this volume). Neuropsychiatrists have established that "the overwhelming stress of maltreatment in childhood is associated with adverse influences on brain development" (De Bellis, 1999, p. 1281).

My work in developmental psychopathology integrates attachment theory, psychiatry, and developmental affective neuroscience in order to explore how attachment trauma alters the developmental trajectory of the right brain (Schore, 1994, 2003a, 2003b). From a developmental viewpoint, early abuse and neglect generates disorganized-disoriented attachment which endures into 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; a predisposition to later psychiatric disorders is the negative long-term effect. From a developmental neuroscience viewpoint, early abuse and neglect have immediate impact on the developing right brain during a critical growth period; this produces an immature right brain that has a limited capacity to regulate intense affective states. These perspectives converge on a basic developmental principle: early trauma is critical to the genesis of an enduring predisposition to pathological dissociation.

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 firmly supports the following principles: (1) "early adverse developmental experiences may leave behind a permanent physiological reactivity in limbic areas of the brain" (Post, Weiss, & Leverich, 1994); (2) emotional and social deprivation interfere with the normal development of the synaptic architecture and lead to "neurological scars" which underlie "subsequent behavioral and cognitive deficits" (Poeggel & Braun, 1996; Poeggel et al., 1999); and (3) "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 PTSD, in other works I have shown that this same developmental neurobiological description applies to the ontogeny of pathological dissociation in borderline personality disorder (Schore, 2003b, 2003c).

In the introduction, I cited Brown and Trimble's (2000) call 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 definition must include a developmental model of dissociative phenomena. In total, the interdisciplinary data cited here indicate 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 via emotional neglect and abuse and interpersonal threat to the body predict somatoform dissociation. The model also strongly supports Putnam's (1995) assertion that dissociation offers "very rich models for understanding the ontogeny of environmentally produced psychiatric conditions."

Although dissociation has been quite controversial, there is now solid convergent evidence from different disciplines that there is a direct relationship between early trauma and pathological dissociation. The next DSM should reflect this advance in our knowledge.

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