CHAPTER 1

Relational Trauma, Brain Development, and Dissociation

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Over the past two decades I have integrated findings from scientific studies and clinical data in order to construct regulation theory, a neuropsychoanalytic model of the development, psychopathogenesis, and treatment of the implicit self (Schore, 2001, 2002). In these expositions I have suggested that in contrast to single-incident trauma from the physical environment, the intense social stressor of early relational trauma is typically ambient, cumulative, and derived from the interpersonal environment. In light of the fact that the infant's first interactions with the interpersonal environment take place within the emotional transactions of the attachment relationship, I equated developmental relational trauma with attachment trauma. Using the perspective of interpersonal neurobiology, I then described the impact of two common expressions of attachment trauma, abuse and neglect, on brain development, especially during the brain growth spurt from the last trimester through the second year of human infancy. Relational trauma thus can be understood as the quintessential expression of "complex trauma," which has been defined as "repeated interpersonal trauma occurring during crucial developmental periods" (Lanius et al., 2011, p. 2).

More recently I have expanded the model of the developmental and

biological disruptions that result from relational trauma (Schore, 2009a, 2009b, 2010a, 2012). In the present chapter I continue to build on those works and provide very recent interdisciplinary data from developmental neuroscience and interpersonal neurobiology that allow for a deeper understanding of the psychological *and* biological effects of early relational trauma, especially caregiver maltreatment in the form of abuse and neglect.

Over this time period the field of developmental neuroscience has experienced "phenomenal progress" (Leckman & March, 2011, p. 333). In a recent issue of the *Journal of Child Psychology and Psychiatry* devoted to the direct relevance of these advances for both research and clinical practice, Roth and Sweatt (2011, p. 400) articulate the currently accepted view of the long-term effect of early caregiver maltreatment in psychopathogenesis:

> Abusive and neglectful experiences from the caregiver are known to leave a child particularly susceptible to cognitive and mental dysfunction. Indeed, there is a significant association of reported childhood maltreatment and the later diagnosis of adolescent and adulthood schizophrenia, borderline personality disorder, posttraumatic stress disorder, and major depression.

Leckman and March (2011) conclude: "A complex, dynamic story is unfolding of evolutionarily conserved genetic programs that guide mammalian brain development and how our in utero and our early postnatal *interpersonal worlds* shape and mold the individuals (infants, children, adolescents, adults and caregivers) we are to become" (p. 333, emphasis added).

The shaping of brain development by our early interpersonal worlds is an essential focus of the fields of interpersonal neurobiology and developmental neuropsychoanalysis. Recall that Bowlby's (1969) formulation of attachment theory attempted to integrate biology and psychoanalysis. For the past two decades a growing body of research indicates that the right hemisphere of the brain is the biological substrate of the human unconscious. This conception is echoed in recent neuroscientific writings by Tucker and Moller (2007): "The right hemisphere's specialization for emotional communication through nonverbal channels seems to suggest a domain of the mind that is close to the motivationally charged psychoanalytic unconscious" (p. 91). This chapter focuses on current studies of the enduring impact of relational trauma on the early maturing right hemisphere of the brain, and thereby on the early development of the human unconscious mind that forms an internal representation of our early interpersonal worlds.

This *interpersonal neurobiological model* explicates the mechanisms by which attachment shapes, for better of worse, the survival functions regulated by the right hemisphere of the brain. It is now clear that the developing brain is not "resilient" but "malleable" (Schore, 2012). In the following sections I offer an overview of studies on the developmental

interpersonal neurobiology of secure attachment, and then on how relational trauma negatively impacts the developmental trajectory of the brain's right hemisphere and the mind and body over the course of the lifespan. Also discussed is the etiology of pathological dissociation, the bottom-line defense of all early-forming severe developmental psychopathologies.

Developmental Interpersonal Biology of Secure Attachment

The essential task of the first year of human life is the creation of a secure attachment bond of emotional communication and interactive regulation between the infant and primary caregiver (Schore & Schore, 2008). Secure attachment depends upon the caregiver's psychobiological attunement with the infant's dynamic alterations of arousal and affective states. Through nonverbal visual–facial, tactile–gestural, and auditory–prosodic communications, the caregiver and infant learn the rhythmic structure of the other and modify their behavior to fit that structure, thereby co-creating a specifically fitted interaction. During the affective communications of mutual gaze, the empathically attuned mother synchronizes the spatial–temporal 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 back to the infant. To accomplish this sequence, the sensitive mother must successfully modulate nonoptimal high *or* excessively low levels of stimulation that would induce extremely heightened or lowered levels of arousal in the infant.

Primary caregivers are not always able to attune to, and optimally mirror, their infants, leading to frequent moments of misattunement in the dyad and ruptures of the attachment bond. The disruption of attachment transactions leads to a transient regulatory failure and an impaired autonomic homeostasis. In the pattern of "interactive repair" or "disruption and repair" following dyadic misattunement, the "good-enough" caregiver who induces a stress response through misattunement reinvokes, in a timely fashion, a reattunement—that is, a regulation of the infant's negatively charged arousal. This repair process allows the infant to cope with stressful negatively charged affects and ultimately allows the individual to gain self-regulatory skills in the form of maintaining persistent efforts to overcome interactive stress.

In a secure attachment relationship the regulatory processes of affect synchrony that co-create positive arousal and interactive repair of negative arousal allow for the emergence of efficient self-regulation (Bradshaw & Schore, 2007). These affectively synchronized experiences trigger homeostatic alterations of neuropeptides (oxytocin, endorphins, corticotropin-releasing factor, growth factors, etc.), neuromodulators (catecholamines),

and neurosteroids (cortisol) that are critical to the establishment of social bonds and to brain development (Schore, 1994, 2005; Wismer Fries, Ziegler, Kurian, Jacoris, & Pollak, 2005). Protective and growth-facilitating attachment experiences have long-term effects on the developing hypothalamic–pituitary–adrenocortical (HPA) axis, which plays a central role in the regulation of stress reactivity (Gunnar, 2000).

In this manner, the optimal interactively regulated affective communications embedded in secure attachment experiences directly imprint the postnatally maturing central nervous system (CNS) limbic system that processes and regulates social–emotional stimuli and the autonomic nervous system (ANS) that generates the somatic aspects of emotion. The limbic system derives subjective information in terms of emotional feelings that guide behavior and allow the brain to adapt to a rapidly changing environment and organize new learning. The higher regulatory systems of the right hemisphere form extensive reciprocal connections with the ANS and the limbic system. Both the ANS and the CNS continue to develop postnatally, and the assembly of these limbic–autonomic circuits (Rinaman, Levitt, & Card, 2000) in the right hemisphere, which is dominant for the human stress response (Wittling, 1997), is directly influenced by the attachment relationship.

A large body of studies now supports the proposal that the long-enduring regulatory effects of attachment are due to their impact on brain development (Schore, 1994, 2003b, 2009a, 2012). The right hemisphere is in a critical growth period from the last trimester of pregnancy through the second year (Chiron et al., 1997; Mento, Suppiej, Altoe, & Bisiacchi, 2010). Attachment transactions in the first year are occurring when total brain volume is increasing by 101% and the volume of the subcortical areas by 130% (Knickmeyer et al., 2008). Because the human limbic system myelinates in the first year and a half, and the early-maturing right hemisphere (Gupta et al., 2005; Schore, 1994; Sun et al., 2005)—which is deeply connected into the limbic system (Gainotti, 2000)—is undergoing a growth spurt at this time, attachment communications specifically impact limbic and cortical areas of the developing right brain (Ammaniti & Trentini, 2009; Cozolino, 2002; Henry, 1993; Schore, 1994, 2000, 2005; Siegel, 1999). Howard and Reggia (2007) state, "Earlier maturation of the right hemisphere is supported by both anatomical and imaging evidence" (p. 112).

In my ongoing work, I continue to present data indicating that the attachment mechanism is embedded in infant–caregiver right-brain to right-brain affective transactions (Schore, 1994, 2000, 2003b, 2009c). Consistent with this interpersonal neurobiological model, researchers in a near-infrared spectroscopy study of infant–mother attachment conclude, "our results are in agreement with that of Schore (2000) who addressed the importance of the right hemisphere in the attachment system" (Minagawa-Kawai et al., 2009, p. 289).

An essential tenet of the interpersonal neurobiological perspective of regulation theory is that affective attachment transactions shape the cortical–subcortical emotion- and stress-regulating circuits of the developing right brain during early critical periods. Indeed, basic research now establishes that optimal stress regulation is dependent on "right hemispheric specialization in regulating stress- and emotion-related processes" (Sullivan & Dufresne, 2006, p. 55). Over the first 2 years of life, a hierarchy of regulatory centers emerges in the developing right brain (Schore, 2003a, 2010b, 2012). Specifically, the subcortical amygdala, with its connections into the insula and hypothalamus, and thereby into the ANS and the HPA axis, is functional at birth. At 3–9 months of age, the anterior cingulate (medial-frontal cortex), a cortical–limbic structure that is associated with responsivity to social cues, comes online, giving the infant even greater self-regulatory capacities. From 10–12 months of age, the regulatory center in the orbital–frontal cortex begins its developmental growth period. This ventral-medial prefrontal cortex, especially in the right hemisphere, is the locus of Bowlby's attachment control system and contains the brain's most complex affect- and stress-regulating mechanisms (Schore, 1994, 2003a, 2003b). Supporting this model, developmental neurobiological research now confirms that the dendritic and synaptic maturation of the anterior cingulate and orbital-frontal cortices is specifically influenced by the social environment (Bock, Murmu, Ferdman, Leshem, & Braun, 2008). Indeed, a recent review of the functional neuroanatomy of the parent–infant relationship by Parsons, Young, Murray, Stein, and Kringelbach (2010, p. 235) concludes, "The same adult brain networks involved in emotional and social interactions are already present in immature and incomplete forms in the infant," specifically mentioning the amygdala, hypothalamus, insula, cingulate cortex, and orbitofrontal cortex.

With optimal attachment experiences, the vertical axis that connects the orbital-frontal and medial prefrontal cortices with multiple cortical and subcortical areas is well developed. This allows the right orbital-frontal cortex to efficiently regulate the subcortical amygdala, which has been shown to be centrally involved in the generation of attachment security (Lemche et al., 2006). Moreover, developmental neurobiological research reveals that coping with early life stress increases the myelination of the ventral-medial cortex, a prefrontal region that controls arousal regulation and resilience (Katz et al., 2009). For the rest of the lifespan the *right* lateralized prefrontal regions are responsible for the regulation of affects, especially stressful affects (Cerqueira, Almeida, & Sousa, 2008; Czeh, Perez-Cruz, Fuchs, & Flugge, 2008; Schore, 1994; Sullivan & Gratton, 2002; Wang et al., 2005). Attachment histories appear to be imprinted into right cortical–subcortical circuits in implicit procedural memory, thus generating an internal working model of attachment that encodes strategies of affect regulation that nonconsciously guide the individual through interpersonal contexts. These adaptive capacities are central to the dual processes of self-regulation:

interactive regulation, the ability to flexibly regulate psychobiological states of emotions with other humans in interconnected contexts, and autoregulation, which occurs apart from other humans in autonomous contexts.

Developmental Interpersonal Biology of Relational Trauma and Dissociation

In contrast to caregivers who foster secure attachment, attachment trauma occurs when the caregiver is either hyperintrusive or emotionally inaccessible and disengaged, given to inappropriate and/or rejecting responses to the infant's expressions of emotions and stress. Such responses provide minimal or unpredictable regulation of the infant's states of over- or underarousal. Instead, the caregiver induces extreme levels of stimulation and arousal (i.e., the very high stimulation of abuse and/or the very low stimulation of neglect). Due to the fact that no interactive repair of frequent significant affective ruptures of the attachment relationship is provided, the caregiver leaves the infant to endure extremely stressful and intense negative states for long periods of time. In an immature organism with undeveloped and restricted coping capacities, stress regulation, and therefore a sense of safety, must be provided by the primary caregiver. When not safety but danger emanates from the attachment relationship, the homeostatic assaults have significant short- and long-term consequences on the maturing psyche and soma.

In terms of the short-term effects, interdisciplinary evidence indicates that the infant's psychobiological reaction to severe interpersonal stressors is comprised of two separate response patterns: hyperarousal and dissociation (Perry, Pollard, Blakley, Baker, & Vigilante, 1995; Schore, 2003a). In the earliest stage of threat, the child's sudden alarm or startle reaction indicates activation of the infant's right hemisphere. This, in turn, evokes a sudden increase of the energy-expending sympathetic branch of the ANS, resulting in significantly elevated heart rate (cardiac acceleration), blood pressure, and respiration. Distress is expressed via crying and then screaming. The infant's state of frantic distress, or 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, and thus noradrenaline, dopamine, and adrenaline levels are significantly elevated, creating a hypermetabolic state within the developing brain. Increased concentrations of cortisol and glutamate (the major excitatory neurotransmitter in the brain) also accompany the state of hyperarousal.

A second later-forming reaction to relational trauma is dissociation, in which the child disengages from stimuli in the external world—traumatized infants often are observed to be "staring off into space with a glazed look." The child's dissociation in the midst of terror involves numbing, avoidance,

compliance, and restricted affect. This energy-conserving 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, 2003a, 2003b). This state of metabolic shutdown and cardiac deceleration is a primary regulatory process that is used throughout the lifespan. 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 has been hypothesized to mediate 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 that of hyperarousal. In this passive state of pain numbing and blunting, endogenous opiates are elevated. Serotonin dysregulation may play an important role (Pieper, Out, Bakermans-Kranenburg, & van IJzendoorn, 2011), consistent with the view that elevated parasympathetic arousal may be viewed as a survival strategy that allows the infant to maintain homeostasis in the face of the internal state of sympathetic hyperarousal. It is important to note that sympathetic energy-expending hyperarousal *and* parasympathetic energy-conserving hypoarousal are both states of "extreme emotional arousal" (Dixon, 1998).

It is now known that there are two parasympathetic vagal systems (Porges, 1997). The late-developing "mammalian" or "smart" ventral vagal system, in the nucleus ambiguus, enables contingent social interactions and secure attachment transactions via the ability to communicate with facial expressions, vocalizations, and gestures. On the other hand, the early-developing "reptilian" or "vegetative" system in the dorsal motor nucleus of the vagus shuts down metabolic activity during intense social stress, generating 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 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).

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). 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 could explain the lengthy "void" states that are associated with pathological dissociative detachment (Allen, Console, & Lewis, 1999).

Disorganized Attachment: The Role of Trauma and Maternal and Child Dissociation

How are the trauma-induced alterations of the developing right brain expressed in the social–emotional 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, Cicchetti, Barnett, & Braunwald, 1989) and that is behaviorally similar to dissociative states (Hesse & Main, 1999). For example:

> 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. (Main & Solomon, 1986, p. 119)

Main and Solomon (1986) document that type D infants often encounter disturbing and dissociative maternal behavior of two kinds: intrusive and apparently angry behavior, or maternal withdrawal and expressions of fear terror. Hesse and Main (2006) hypothesize that when the mother enters a dissociative state, a fear alarm state is triggered in the infant. In their description, the caregiver suddenly completely " 'freezes' with eyes unmoving, half-lidded, despite nearby movement and addresses the infant in an 'altered' tone with simultaneous voicing and devoicing" (2006, p. 320):

> 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. . . . We 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)

Ongoing developmental research now underscores a strong link between dissociative-like maternal behavior and disorganized infant attachment (Schuengel, Bakersmans-Kranenburg, & van IJzendoorn, 1999; MacDonald et al., 2008).

In recent writings Beebe and colleagues (2010) report studies of mothers of 4-month-old infants who later show disorganized attachment. They

observe that the mothers of these infants are overwhelmed by their own unresolved abuse or trauma and therefore cannot bear to intersubjectively engage with their infant's distress. Because these mothers are unable to regulate their own distress, they cannot regulate their infant's distress. Because these mothers are unable to allow themselves to be emotionally affected by their infant's dysregulated state, they shut down emotionally, closing their faces, looking away from the infant's face, and failing to coordinate with the infant's emotional state. Beebe interprets this fearful maternal behavior as a defensive dissociation, a strategy that protects the mother from the facial and visual intimacy that would come from joining the infant's distressed moments. This type of mother thus shows disrupted and contradictory forms of affective communication (abuse intrusiveness hyperarousal *and* neglect disengagement hypoarousal), especially around the infant's need for comfort when distressed.

Over an ongoing period of relational trauma, the mother's disengagement and "detachment from an unbearable situation" is matched by the infant's disengagement, detachment, and withdrawal. Milne, Greenway, Guedeney, and Larroque (2009) describe the long-term negative developmental impact of social withdrawal and depression in 6-month-old infants. They conclude:

> A withdrawal response in infancy is problematic behavior .. . not because it leads to later withdrawal per se, but because of the compounding effects on development of not being present in the interpersonal space—the space upon which much of infant development depends. (p. 165)

Guedeney, Foucault, Bougen, Larroque, and Mentre (2008) report a study of relational withdrawal in infants ages 14–18 months. This withdrawal reaction reflects inadequate parent–infant interactions and is a feature of disorganized attachment. Guedeney et al. (2008) note: "Sustained withdrawal behavior may be viewed as a chronic diminution of the attachment system, which is gradually generalized into a diminished engagement and lowered reactivity to the environment as a whole" (p. 151). They conclude:

> Withdrawn social behavior from as early as 2 months of age, indicated by a lack of either positive (e.g., smiling, eye contact) or negative (e.g., vocal protestations) behavior, is more akin to a state of learned helplessness and should alert the clinician to the possibility that the infant is not displaying age-appropriate emotional/social behavior. (p. 151)

The developing infant/toddler who has an early history of traumatic attachment is too frequently exposed to a massively misattuning primary caregiver who triggers and does not repair long-lasting, intensely dysregulated states. The growth-inhibiting environment of relational trauma may generate dense and prolonged levels of negative affect associated with extremely stressful states of hyper- and hypoarousal. For self-protective

purposes the child severely restricts her or his overt expressions of an attachment need for dyadic regulation. As discussed above, this restricted expression would be consistent with a reduction in the output of the right-lateralized emotion-processing, limbic–autonomic attachment system. When stressed, defensive functions may be rapidly initiated that quickly shift the brain from interactive regulatory modes into long-enduring, less complex autoregulatory modes that may result in dissociation.

During these episodes, the child appears to be matching the rhythmic structures of the mother's dysregulated states; this synchronization can be registered in the firing patterns of the stress-sensitive cortical and limbic regions of the infant's brain, especially in the right brain, which is in a critical period of growth (Bazhenova, Stroganova, Doussard-Roosevelt, & Posikera, 2007; Buss et al., 2003; de Haan, Belsky, Reid, Volein, & Johnson, 2004). Thus the chaotic and dysregulated alterations of state induced by relational trauma may become imprinted into the developing right brain self-system of the child.

From a developmental psychopathological viewpoint, a profound negative psychological effect of relational trauma (early abuse and neglect) is the generation of a disorganized attachment that endures over the later stages of childhood, adolescence, and adulthood and acts as a risk factor for later psychiatric and personality disorders (Schore, 2001, 2002, 2003a). From a developmental neuroscience perspective, the immediate detrimental psychobiological impact is an alteration in metabolic processes that can now only poorly sustain the critical growth period of the developing right brain, and the lasting impairment is an immature and functionally limited right-brain capacity to regulate later life stressors that, unregulated, generate intense affect states (Schore, 2003b). Montirosso, Borgatti, and Tronick (2010) conclude: "Infants cope with the emotional distress caused by unresponsive mothers through self-regulation behaviors associated with a greater activation of the right hemisphere. This finding supports the view that during a stressful condition there is a state-dependent activation of the right hemisphere" (p. 108).

Relational traumatic experiences are believed to be stored in the form of visual and procedural memories associated with the visual–spatial right hemisphere (Schiffer, Teicher, & Papanicolaou, 1995), which is dominant for the processing of unconscious emotions (Gainotti, 2012) and is the locus of implicit (Hugdahl, 1995) and autobiographical (Daselaar et al., 2007; Markowitsch, Reinkemeier, Kessler, Koyuncu, & Heiss, 2000) memory. Recent models of early-life trauma thus shift the focus from deficits in later-maturing conscious, verbal, explicit, and voluntary behavior to impairments in early-maturing nonconscious, nonverbal, implicit, and automatic adaptive social–emotional functions (Schore, 2010c). These psychological and biological perspectives converge on a basic developmental principle of regulation theory: that early traumatic sundering of attachment bonds is critical to the genesis of an enduring predisposition to a variety

of early-forming severe psychopathologies that involve the autoregulating, affect-deadening defense of pathological dissociation.

A Model of the Enduring Effects of Relational Trauma: Impaired Right-Brain Emotion Processing and Stress Regulation and Pathological Dissociation

Describing the capacities of the right-lateralized "social brain," Brancucci, Lucci, Mazzatenta, and Tommasi (2009) conclude: "The neural substrates of the perception of voices, faces, gestures, smells and pheromones, as evidenced by modern neuroimaging techniques, are characterized by a general pattern of right-hemispheric functional asymmetry" (p. 895). Over all stages of the lifespan, this hemisphere is dominant not only for the processing of social interactions (Decety & Lamm, 2007; Semrud-Clikeman, Fine, & Zhu, 2011), but also for coping with negative affects (Davidson, Ekman, Saron, Senulis, & Friesen, 1990), the organization of the human stress response (Wittling, 1997), and stress regulation (Cerqueira et al., 2008; Schore, 1994; Stevenson, Halliday, Marsden, & Mason, 2008; Thayer & Lane, 2009; Wang et al., 2005). Emphasizing the essential survival functions of this (and not the left) system, Schutz (2005) noted:

> The right hemisphere operates a distributed network for rapid responding to danger and other urgent problems. It preferentially processes environmental challenge, stress and pain and manages self-protective responses such as avoidance and escape. Emotionality is thus the right brain's "red phone," compelling the mind to handle urgent matters without delay. (p. 15)

These adaptive right-brain functions often are impaired in individuals with histories of early relational trauma.

Recent neurobiological data support the proposed model of the psychopathogenetic mechanism by which attachment trauma negatively impacts right-brain development. Adamec, Blundell, and Burton (2003) reported experimental data that "implicate neuroplasticity in right-hemispheric limbic circuitry in mediating long-lasting changes in negative affect following brief but severe stress" (p. 1264). According to Gadea, Gomez, Gonzalez-Bono, and Salvador (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). I suggest that this right-brain "damage" is most operative during dysregulating experiences of attachment-related hyper- and hypoarousal.

Consistent with these findings, dissociative defensive functions are initiated in attachment contexts that generate too frequent, intense, unrepaired, and enduring relational trauma. These dissociative functions may

reflect a very rapid shift in 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 significantly diminishes the substantial amounts of energy required for critical-period biosynthetic processes. The dysregulating events of abuse and neglect thus could create severe, chaotic biochemical alterations in the infant brain. This disruption of energy resources for the biosynthesis of right-lateralized limbic connections would be expressed in a critical period of developmental overpruning of the cortical–limbic system (see Schore, 1994, 2002, 2003a, 2009a).

It is now accepted that "psychological factors" "prune" or "sculpt" neural networks in, specifically, the postnatal frontal, limbic, and temporal cortices. Excessive pruning of cortical–subcortical limbic–autonomic circuits occurs in early histories of trauma and neglect. This severe growth impairment represents a possible mechanism of the genesis of a developmental structural defect in the "emotional" right brain. Because this defect involves limbic and autonomic circuits, the resulting functional deficit is likely to specifically affect the individual's ability to cope with intense affects. In this manner the traumatic context in which disorganized attachment arises thus could act as a growth-inhibiting environment for the experience-dependent maturation of right-lateralized CNS–ANS circuits.

The psychobiological context of disorganized attachment during the brain growth spurt of the first 2 years of life thus may alter the developmental trajectory of the right brain. The massive psychobiological stress associated with attachment trauma may not only impair the development of this system but may also set the stage for the characterological use of right-brain defensive pathological dissociation when encountering later social–emotional stressors. As noted above, converging evidence indicates that early abuse negatively impacts limbic and autonomic nervous system maturation, producing enduring neurobiological alterations that underlie affective instability, inefficient stress tolerance, memory impairment, *and* dissociative disturbances. In this manner, traumatic stress in childhood may lead to self-modulation of painful affect by directing attention away from internal emotional states (Lane, Ahern, Schwartz, & Kaszniak, 1997). The right hemisphere is dominant not only for the regulation of affects, but also for maintaining a coherent sense of one's body (Tsakiris, Costantini, & Haggard, 2008), for sustaining attention (Raz, 2004), and for pain processing (Symonds, Gordon, Bixby, & Mande, 2006). Thus, the right brain–related strategy of dissociation may represent the ultimate defense for blocking emotional body-based pain from consciousness.

Dutra, Bureau, Holmes, Lyubchik, and Lyons-Ruth (2009) observe that disrupted maternal affective communications and lack of involvement in the regulation of stressful arousal are associated with the child's use of dissociation, "one of the few available means for achieving a modicum of

relief from fearful arousal." This in turn can lead to a child who does "not . . . acknowledge pain and distress within a set of caregiving relationships that are vital for survival" (p. 388). In clinical writings, Bromberg (2006) links right-brain trauma to autonomic hyperarousal, a chaotic and terrifying flooding of affect that can threaten to overwhelm sanity and imperil psychological survival. He observes that dissociation is then automatically and immediately triggered as the fundamental defense to the arousal dysregulation of overwhelming affective states.

Echoing this perspective in the neuroscience literature, Lanius and her colleagues (2005), in a functional magnetic resonance imaging (fMRI) study of traumatized patients with posttraumatic stress disorder (PTSD), show right-hemispheric activation during dissociation. These authors conclude that patients dissociate in order to escape from the overwhelming emotions associated with the traumatic memory. Using transcranial magnetic stimulation, Spitzer, Wilert, Grabe, Rizos, and Freyberger (2004) similarly report that dissociation is associated with right-hemispheric dysfunction in the form of lack of integration in the presence of emotionally distressing or threatening stimuli. Enriquez and Bernabeu (2008) also offer research showing that "dissociation is associated with dysfunctional changes in the right hemisphere which impair its characteristic dominance over emotional processing" (pp. 272–273). They document that "high dissociators" retain an ability for processing left-hemispheric verbal stimuli but show deficits in right-hemispheric perception of the emotional tone of voice (prosody).

More recently, Helton, Dorahy, and Russell (2011) report that "high dissociators" have difficulty in specifically coordinating activity within the right hemisphere and that such deficits become evident when this hemisphere is "loaded with the combined effects of a sustained attention task and negative emotional stimuli. . . . Thus, the integration of experiences, which rely heavily on right-hemispheric activation (e.g., negative emotion, sense of self with reference to the experience), may be compromised in high dissociators" (p. 700). These findings are echoed in current neurological research. Brand et al. (2009) and Stanilou, Markowitsch, and Brand (2010) document right-temporal-frontal hypometabolism in cases of dissociative amnesia, which is clinically expressed as an inability to recall important personal information of a traumatic nature: a failure to integrate consciousness, emotion, and cognition, resulting in a "constricted self" (p. 793).

Thus, both researchers and clinicians are now exploring the evolution of a developmentally impaired regulatory system and providing evidence that prefrontal cortical and limbic areas of the right hemisphere are centrally involved in the deficits in mind and body associated with a pathological dissociative response (Schore, 2002, 2009a, 2009b, 2009c). This hemisphere, more than the left, is densely and reciprocally interconnected with emotion-processing limbic regions, as well as with subcortical areas that generate both the arousal and autonomic body-based aspect of emotions.

Sympathetic ANS activity is manifest in tight engagement with the external environment and a high level of energy mobilization, whereas the parasympathetic component drives disengagement from the external environment and utilizes low levels of internal energy (Recordati, 2003). These ANS components are too frequently uncoupled for long periods of time in stressful interpersonal experiences in infants, children, adolescents, and adults who have histories of attachment trauma, and thus they are likely to be expressed in body-based visceral–somatic disturbances.

Kalsched (2005) describes operations of defensive dissociative processes used by the child during traumatic experience by which "affect in the body is severed from its corresponding images in the mind and thereby an unbearably painful meaning is obliterated" (p. 174). Nijenhuis (2000) asserts that "somatoform dissociation" is an outcome of early-onset traumatization expressed as a lack of integration of sensorimotor experiences, reactions, and functions of the individual's self-representation. Dissociatively detached individuals are not only detached from the environment, but also from the self—from their body, their actions, and their sense of identity (Allen et al., 1999). This detachment is expressed as a deficit in the right-hemispheric "corporeal self" (Devinsky, 2000). Crucian et al. (2000) describe "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" (p. 643).

Conclusion

In an optimal attachment scenario, a right-lateralized hierarchical prefrontal system performs an essential adaptive motivational function: the relatively fluid switching of internal body-based states in response to changes in the external environment that are nonconsciously appraised to be personally meaningful. In contrast, relational trauma elicits more than a disruption of conscious cognition and a disorganization of overt behavior, but rather, it negatively impacts the early organization of evolutionary-based right-brain mechanisms that operate beneath levels of conscious awareness. Pathological dissociation creates a maladaptive, highly defensive, rigid, closed self system, such that even low levels of intersubjective stress may lead to parasympathetic dorsal vagal hypoarousal, heart rate deceleration, and passive disengagement. This fragile unconscious system is susceptible to relational stress-induced mind–body collapse and thereby to a sudden implosion of the implicit self and a rupture of self-continuity. This collapse of the implicit self often involves the amplification of the parasympathetic-related affects of shame and disgust, and cognitions of hopelessness and helplessness. In addition, the collapse of the implicit self tends to be accompanied by an instant dissipation of a sense of safety and trust, consistent with the hypothesis that it originates in a failure of right-brain–related regulation of attachment security.

Dissociation thus may reflect an inability of the right-brain cortical–subcortical implicit self system to adaptively recognize and process external stimuli (exteroceptive information coming from the relational environment) and to integrate them, on a moment-to-moment basis, with internal stimuli (interoceptive information from the body, somatic markers, the "felt experience"). This integration failure of the higher right hemisphere with the lower right brain could thus induce an instant collapse of both subjectivity and intersubjectivity. Stressful affects, especially those associated with emotional pain, are thus not experienced in consciousness, and the individual's sense of self and of relation to others may become dissociated. The endpoint of chronically experiencing catastrophic states of relational trauma in early life is a progressive impairment of the ability to adjust, take defensive action, or act on one's own behalf, and a blocking of the capacity to register affect and pain—all critical to survival.

Psychotherapy with dissociative patients therefore needs to attend to the severe dysregulation of affect that characterizes the developmental self pathologies associated with histories of relational trauma. Experiences of relational trauma and attachment dysregulation are expressed in the therapeutic alliance as affectively stressful enactments (Schore, 2011, 2012). Bromberg (2011) observes that in the clinical encounter, pathological dissociation acts as an "early warning system" that anticipates potential affect dysregulation before the trauma arrives. Clinical work with such patients must address the early-forming dissociative defense that blocks overwhelming affects from reaching consciousness, thereby denying the possibility of interactive regulation and the organization of more complex right-brain stress regulation. With respect to the psychotherapeutic context, the clinical research of Spitzer et al. (2007) demonstrates that insecurely attached patients with dissociative defenses dissociate as a response to negative emotions arising in psychodynamic psychotherapy, leading to a less favorable treatment outcome.

The current paradigm shift from a focus on cognition to one of affect (Schore, 2012) also includes a shift in clinical work from solely a repression-based theoretical foundation to recognition of the survival strategy of dissociation. Distinguishing between early-forming dissociative defenses and later-forming repressive defenses, Diseth (2005) writes:

> As a defense mechanism, dissociation has been described as a phenomenon quite different from repression. Repression has been considered an unconscious mechanism, placing unwanted feelings away from the conscious mind because of shame, guilt or fear. . . . However, in order to repress, you must to some degree have processed the feelings, recognized their nature and the taboos connected to such feelings. Dissociation is about not having processed the inputs at all. (pp. 81, 82)

This bottom-line psychobiological defense of dissociation thus represents a major obstacle to the intersubjective change process in all affectively

focused psychotherapies, but especially in patients with a history of early relational trauma. Dissociated affects are unconscious affects, and so this treatment needs to directly engage the right hemisphere, which is dominant for the processing of unconscious emotions (Gainotti, 2012), especially unconscious negative emotion (Sato & Aoki, 2006).

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