Chapter 2

Relational trauma and the developing right brain

The neurobiology of broken attachment bonds

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Over the past two decades I have integrated ongoing scientific studies and clinical data in order to construct regulation theory, a neuropsychoanalytic model of the development, psychopathogenesis, and treatment of the implicit self. Towards that end, in 2001 I edited an issue of the *Infant Mental Health Journal*, and in it I offered an article, 'The effects of early relational trauma on right brain development, affect regulation, and infant mental health'. In this chapter I build on that work and provide very recent interdisciplinary developmental data that allow for a deeper understanding of the psychological *and* biological effects of early relational trauma. A particular focus will be on current studies of the early developing right brain, the biological substrate of the human unconscious and the site of the highest cortical–subcortical regulatory centers. This interpersonal neurobiological model explicates the mechanisms by which attachment trauma negatively impacts the developmental trajectory of the right brain/mind/body system 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. Pierre Janet (1889) defined pathological dissociation as a phobia of memories, expressed as excessive or inappropriate physical responses to thought or memories of 'old traumas'. It is now clear that these 'old traumas' specifically refer not just to childhood traumas but also to relational trauma occurring in infancy, the critical period of attachment. This theoretical perspective has direct clinical applications for models of both treatment and prevention.

Developmental interpersonal neurobiology 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. There is now agreement that 'learning how to communicate represents perhaps the most important developmental process to take place during infancy' (Papousek & Papousek, 1997),

and that 'In one sense we can consider the whole of child development to be the enhancement of self-regulation' (Fonagy & Target, 2002). In line with the essential attachment elements of affect communication and affect regulation, Schore and Schore (2008: 10) now suggest that:

> In line with Bowlby's fundamental goal of the integration of psychological and biological models of human development, the current clinical and experimental focus on how affective bodily-based processes are nonconsciously interactively regulated . . . has shifted attachment theory to a regulation theory.

Secure attachment depends not on the mother's psychobiological attunement with the infant's cognition or behavior, but rather on her regulation of the infant's internal states of arousal, the energetic dimension of the child's affective state. Through nonverbal visual–facial, tactile–gestural, and auditory–prosodic communication, 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 bodily based affective communications of mutual gaze, the attuned mother synchronizes the spatio-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, the sensitive mother must successfully modulate nonoptimal high *or* nonoptimal low levels of stimulation that would induce supra-heightened or extremely low levels of arousal in the infant.

In order to enter into this communication, the mother must be psychobiologically attuned to the dynamic crescendos and decrescendos of the infant's bodily based internal states of autonomic nervous system (ANS) peripheral arousal. Indeed, the intersubjective dialogue between mother and infant consist of signals produced by the autonomic, involuntary nervous system in both parties. The attachment relationship mediates the dyadic regulation of emotion, wherein the mother co-regulates the infant's postnatally developing ANS, and thereby its internal homeostatic state. Also known as the vegetative nervous system (from the Latin *vegetare*, to animate or bring to life), it is responsible for the generation of what Stern (1985) calls vitality affects.

Research now clearly demonstrates that the primary caregiver is not always attuned and optimally mirroring, that there are frequent moments of misattunement in the dyad, ruptures of the attachment bond. The disruption of attachment transactions leads to a regulatory failure and an impaired autonomic homeostasis. In this pattern of 'interactive repair' following dyadic misattunement (Tronick, 1989) or 'disruption and repair' (Beebe & Lachmann, 1994), the 'good-enough' caregiver, who induces a stress response

through misattunement, in a timely fashion reinvokes a reattunement, a regulation of the infant's negatively charged arousal.

If attachment is the regulation of interactive synchrony, then attachment stress is an asynchrony of psychobiological attunement. In optimal interpersonal contexts, following such stress, a period of re-established synchrony allows the child to recover his/her regulatory equilibrium. Resilience in the face of stress is an ultimate indicator of attachment security. 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. These affectively synchronized experiences trigger homeostatic alterations of neuropeptides (oxytocin), neuromodulators (catecholamines), and neurosteroids (cortisol), which are critical to the establishment of social bonds and to brain development (Schore, 1994, 2005; Wismer Fries et al., 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). Thus, the evolutionary mechanism of attachment represents the regulation of biological synchronicity between and within organisms (Schore, 1994; Bradshaw & Schore, 2007).

A large body of studies now support the proposal that the long-enduring regulatory effects of attachment are due to its impact on brain development (Schore, 1994, 2003b, 2009b). 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). This growth, especially of white matter, is experience-dependent. Fonagy and Target (2005: 334) point out that:

> If the attachment relationship is indeed a major organizer of brain development, as many have accepted and suggested (e.g., Schore, 1994, 2003[a]), then the determinants of attachment relationships are important far beyond the provision of a fundamental sense of safety or security (Bowlby, 1988).

Echoing this in the neuroscience literature, Ziabreva and colleagues (2003: 5334) conclude that:

> the mother functions as a regulator of the socio-emotional environment during early stages of postnatal development . . . subtle emotional regulatory interactions, which obviously can transiently or permanently alter brain activity levels . . . may play a critical role during the establishment and maintenance of limbic system circuits.

Because the human limbic system myelinates in the first year and a half (Kinney et al., 1988) and the early-maturing right hemisphere (Geschwind

& Galaburda, 1987; Schore, 1994) – which is deeply connected into the limbic system (Tucker, 1992; Gainotti, 2000) – is undergoing a growth spurt at this time, attachment communications specifically impact limbic and cortical areas of the developing right cerebral brain (Cozolino, 2002; Henry, 1993; Schore, 1994, 2000, 2005; Siegel, 1999).

Indeed, in 1997 Chiron and her colleagues published a developmental neurobiological study entitled 'The right brain hemisphere is dominant in human infants'. In subsequent neuropsychological research on emotional lateralization in the second year of life, Schuetze and Reid (2005: 207) stated, 'Although the infant brain was historically reported to be undifferentiated in terms of cerebral lateralisation until 2 years of age, evidence has accumulated indicating that lateralised functions are present much earlier in development'. They further observe 'lateralisation of negative emotional production to the right hemisphere in infants as young as 12 months of age', and 'a developmental enhancement of right hemisphere control of negative emotional expression that is evident by 24 months'. More recently, Howard and Reggia (2007: 112) conclude, 'Earlier maturation of the right hemisphere is supported by both anatomical and imaging evidence'.

In my ongoing work I continue to offer data which indicate that the attachment mechanism is embedded in infant–caregiver right-hemisphere-to-right-hemisphere affective transactions, and that this interpersonal neurobiological model is supported by a large body of recent developmental research (Schore, 1994, 2000, 2003b, in press). With respect to visual–facial attachment communications, it is now established that 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). Developmental neuroscience documents that 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., 2000), infants show right hemispheric activation when exposed to a woman's face (Tzourio-Mazoyer et al., 2002). Recent near-infrared spectroscopy research (perhaps the most suitable of all neuroscience techniques applicable to human infants) reveals that specifically the five-month-olds' right hemisphere responds to images of adult female faces (Nakato et al., 2009; Otsuka et al., 2007).

Closer to an interpersonal face-to-face perspective, an electroencephalography (EEG) study by Grossmann et al. (2007) reports that four-month-old infants presented with images of a female face gazing directly ahead show enhanced gamma electrical activity over right prefrontal areas. These authors conclude that the brain mechanisms underlying eye gaze perception show a high degree of specialization early in ontogeny, recruiting areas in the right hemisphere. Other researchers have established that mutual gaze activates face-processing areas of the right hemisphere (Pelphrey et al., 2004).

In terms of tactile–gestural attachment communications, Nagy (2006: 227)

demonstrates 'lateralized system for neonatal imitation' and concludes, 'The early advantage of the right hemisphere (Chiron et al., 1997; Schore, 2000; Trevarthen, 2001) in the first few months of life may affect the lateralized appearance of the first imitative gestures'. Sieratzki and Woll (1996) describe the effects of touch on the developing right hemisphere, and assert that the emotional impact of touch is more direct and immediate if an infant is held to the left side of the body. Studies also demonstrate that spontaneous gestures that express feeling states communicated within a dyad activate right hemispheric structures (Gallagher & Frith, 2004). And mirror neuron researchers now contend that developing children rely on a 'right hemisphere-mirroring mechanism – interfacing with the limbic system that processes the meaning of observed or imitated emotion' (Dapretto et al., 2006).

As for auditory–prosodic attachment communications, prosodic processing in three-month-old infants activates the right temporoparietal region (Homae et al., 2006). At 11 months the voice of a woman's child-directed speech (i.e. with somewhat exaggerated prosody) elicits a right-lateralized event-related potential (ERP) (Thierry et al., 2003). According to Bogolepova and Malofeeva (2001: 353):

> The right hemisphere of the neonate is actively involved in the perception of speech melody and the intonations of the voices of mother and surrounding people. The pre-speech stage of child development is characterized by interactions of the descriptive and emotional components due mainly to mechanisms operating within the hemispheres on the principle of non-verbal communication.

And on the other side of the attachment dyad, researchers now describe the mother's processing capacities: 'A number of functions located within the right hemisphere work together to aid monitoring of a baby. As well as emotion and face processing the right hemisphere is also specialized in auditory perception, the perception of intonation, attention, and tactile information' (Bourne & Todd, 2004: 22–23).

This right lateralized system stores a vocabulary of nonverbal affective facial expressions, gestures, and prosody, right brain signals used in implicit attachment communications (see Schore's model of affect regulation and right brain development in Table 2.1). The output of the right hemisphere, 'the emotional brain', is a conscious affect. The highest centers of this hemisphere, especially the orbitofrontal (ventromedial) cortex, the locus of Bowlby's attachment system, act as the brain's most complex affect and stress regulatory system (Schore, 1994, 2003a, 2003b; Sullivan & Gratton, 2002). The organization of dendritic and synaptic networks in the orbitofrontal (and anterior cingulate) cortex, including its connections into the limbic system, are thus dramatically shaped by early relational emotional experience (Schore, 1994; Bock et al., 2008).

Table 2.1 Schore's model of affect regulation and right-brain development

	Infant context	*Mother context*	*Interactive right brain to right brain*
RIGHT BRAIN COMMUNICATION PROCESSES			
Visual/Facial			
Regulated response	• Orients, explores, gazes at face of mother and others, seeks eye contact. • Displays bright, wide-eyed facial expressions. • Uses a wide range of affective expressions. • Resting quiet–alert state of pleasant facial expressions.	• Responds (attunes) to infant's cues with variety of affectively expressive facial expressions (eye contact, smiling, pleasant facial expressions).	• Dyadic visual–affective arousal regulation. • Each member of dyad focuses gaze upon the other, engaging in mutual eye contact, smiling, bright facial expressions. • Interpersonal resonance amplifies positive states in both.
Stress response	• During relational stress, transiently avoids orienting, exploring, or gazing at mother's face or engaging in eye contact.	• Flat, absent, fear-inducing, or incongruent facial expressions (laughing when infant is distressed).	• One breaks off mutual gaze and/or eye contact. • Dyad transiently out of sync (misattuned): acute dyadic stress. • *Absence or avoidance of eye contact by either mother or infant may be a significant indicator requiring further investigation.*
Vocal tone and rhythm			
Regulated response	• Turns towards mother's voice. • Uses inviting/playful tone in response (cooing, babbling).	• Vocalizes soothing responses with varied tones and rhythms. • Modulates tones and rhythms of voice to infant's psychobiological state.	• Dyadic auditory–affective arousal regulation. • Matches or imitates each other's vocal tones and rhythms.

Stress response	• During relational stress, transiently turns away from mother's voice. • Uses distressed tone (crying) in response or is nonresponsive.	• Uses discordant, harsh, loud, or unmodulated tone and rhythm of voice or does not use vocalizations in response to infant's emotional communication. • Does not vocalize or mirror (match) infant's vocalizations.	• One uses discordant tone while the other is silent or both are using distressed or discordant tones. • *Nonresponsivity or turning away from mother's voice may be a significant indicator requiring further investigation.*
Gestural/Postural Regulated response	• Moves limbs and body evenly and fluidly, relaxed posture, reaches and turns toward other or novel social stimulus.	• Approaches to soothe, manipulate, or manoeuver infant gently and cautiously. • Responds to and interprets social bodily based gestures.	• In intimate physical context, dyad's rhythmic matching allows bodies to cradle/mold into other. • In social referencing late in first year, gestures become purposeful and synchronized, promoting intersubjective engagement.
Stress response	• In socially stressed contexts, moves limbs unevenly and/or frantically. • Fails to reach out, averts head, turns body away, stiffens or arches body to mother's touch.	• Approaches infant too quickly or responds to infant in threatening or fearful manner. • Handles awkwardly or roughly. • Misinterprets infant's gestures or does not attempt to soothe, respond, or interpret gestures and body movements.	• Infant continues or increases distressed gestures and postures and is unresponsive to mother's efforts. • Mother increases rough/awkward gestures/postures. • Mother continues to misinterpret infant's gestures/body movements. • Dyad becomes frustrated or ceases/fails to attempt to soothe and comfort interactively.

(*Continued overleaf*)

Table 2.1 Continued

	Infant context	*Mother context*	*Interactive right brain to right brain*
RIGHT BRAIN AFFECT PROCESSING			
Positive affect processing			
Regulated response	• High, positive arousal. • Enjoyment–joy, interest–excitement. • Vitality expressed freely.	• Happy demeanor; responsive to, supportive of, and matching of infant's affect and positive arousal.	• Mutual delight. • Mother or infant leads affective interaction while other follows. • Non-overwhelming and turn-taking behaviors. • Dyadic amplification of positive arousal in relational play.
Stress response	• Hyperaroused/overstimulated or hypoaroused/understimulated.	• Incongruent happy demeanor to infant's distressed cues or sad demeanor to infant's positive cues. • Continues to fail to create regulated, positive arousal stimuli for infant. • Low frequency of play behavior.	• Mismatched (misattuned) arousal states. • One or both hyperaroused/ overstimulated or one is in positive arousal state while the other is hypoaroused/understimulated or hyperaroused/overstimulated. • Overwhelmed dyad.
Negative affect processing			
Regulated response	• Fussy, moody affect expressed freely. • Resilience.	• Able to tolerate and express sadness, anger, fear in self and infant while seeking to interact appropriately. • Participates in interactive repair.	• Mutual attuning to disquieting stimuli or condition.
Stress response	• Withdraws or is nonresponsive or becomes agitated, frustrated, or fearful when experiencing sensations of distress (dysregulated states). • Increasing intensity and duration of either state precludes infant's quick response to soothing attempts and return to regulated state.	• Unable to tolerate own negative feelings and responds inappropriately (expresses anger, irritation, or frustration or withdraws and is nonresponsive toward infant). • Poor capacity for interactive repair.	• Mutual frustration. • Mother cannot or does not soothe infant and repair negative affect: dyad remains in distressed state.

RIGHT BRAIN REGULATION			
Interactive regulation	• Expresses and recognizes affective facial expressions, vocalizations, and gestures. • Infant seeks out mother to co-regulate inner state of being.	• Responds with arousal/regulating facial expressions, vocalizations, and gestures. • Mother seeks to affect infant's inner state of being.	• Each member of dyad contingently responds to other's facial expressions, vocalizations, and gestures (right brain to right brain) • Mother and infant interactively seek attunement. • Frequent episodes of interactive play.
Autoregulation	• Self-soothing behaviors (sucks finger/pacifier, rocks body, holds soft object). • Self-created solutions for regulating inner state of being.	• Self-calming behaviors (deep breaths, self-talk). • Mother lets infant struggle with distress briefly and then regulates (assists in autoregulation).	• Each member of dyad remains calm in presence of other. • Each regulates own state of being autonomously.
RIGHT BRAIN DYSREGULATION			
Interactive dysregulation	• Averts gaze, becomes agitated by sounds and gestures. • Startles to parent. • Habitually disconnects from mother's attempts to coregulate while inner state escalates. • Sense of safety threatened by interaction.	• Frequent angry, hostile facial expressions, harsh tone and uneven rhythms, threatening gestures. • Does not look at the infant or unresponsive 'dead face.' • Repeatedly fails to respond to infant's affective struggle despite infant's escalating inner distress.	• Mutual arousal dysregulation. • Individually or dyadically ignores cues of other: dyad fails to collaborate in regulating infant's inner need state. • Inconsolable infant may lead to mother's negative feelings toward him/her and diminish mother's confidence in her being a 'good enough' mother.
Autodysregulation	• Crying, arching, flailing, and vomiting; or blank stare, limp, motionless. • Infant repeatedly fails to self-regulate inner state, becoming overwhelmed, eventually exhausted and withdrawn. • Dissociates to maternal stimuli. • Chronic sense of threat or lack of sense of safety.	• Irritable, threatening, intrusive, and rough or flat affect, unresponsive. • Disregards infant's ability to autoregulate by quieting or stimulating self. • Dissociates to infant's stimuli.	• Agitated or withdrawn in presence of other. • Both fail to allow infant to enlarge his/her capacity to self-regulate affect. • No relational or intersubjective context.

Confirming this right-brain-to-right-brain interpersonal neurobiological model, in very recent functional magnetic resonance imaging studies of mother–infant emotional communication Lenzi et al. (2009) offer data 'supporting the theory that the right hemisphere is more involved than the left hemisphere in emotional processing and thus, mothering', and Noriuchi et al. (2008) show activation of the mother's right orbitofrontal cortex during moments of maternal love triggered by viewing a video of her own infant. A near-infrared spectroscopy study of infant–mother attachment at 12 months concludes, '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: 289).

At the end of the first year, right cortical–subcortical circuits imprint in implicit–procedural memory, an internal working model of attachment which encodes strategies of affect regulation that nonconsciously guide the individual through interpersonal contexts. This working model generates unconscious 'procedural expectations' of the emotional availability of others during stress (Cortina & Liotti, 2007). Although these expectations are not experienced as left-brain conscious thoughts, they are consciously experienced as subjective right-brain affectively charged, embodied cognitions ('gut feelings'). At all points of the lifespan attachment communications are expressed not in left-brain secondary process but in right-brain primary process cognitions (Dorpat, 2001; Schore & Schore, 2008; Schore, in press).

Attachment neurobiology of relational trauma

Optimal attachment communications directly affect the maturation of the central nervous system (CNS) limbic system that processes and regulates social–emotional stimuli and the autonomic nervous system (ANS) that generates the somatic aspects of emotion. It is important to stress that a growth-facilitating emotional environment is required for a child to develop an internal system that can adaptively regulate arousal and an array of 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 scaffold and support an expansion of the child's right-brain regulatory coping capacities and underlie the developmental principle that secure attachment is the primary defense against trauma-induced psychopathology.

In contrast to caregivers who foster secure attachment, an abusive or neglectful caregiver not only plays less but also induces enduring negative affect in the child. Such caregivers provide little protection against other environmental impingements, including that of an abusive father. In contexts of relational trauma 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 over-arousal. 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 extremely stressful intense negative states for long periods of time.

There is now extensive evidence that stress is a critical factor that affects social interactions, especially the mother–child interaction (Suter et al., 2007). Overviewing the literature, these researchers report that during stressful life episodes mothers were less sensitive, more irritable, critical and punitive, and showed less warmth and flexibility in interactions with their children. They conclude, 'Overall, stress seems to be a factor that has the power to disrupt parenting practices seriously and results in a lower quality of the mother–child interaction' (Suter et al., 2007: 46). In a review of parenting issues for mothers who manifest chronic stress dysregulation and are diagnosed with borderline personality disorders, Newman and Stevenson (2005: 392) conclude, 'Clearly, this group of women are very fragile and experience high levels of inner turmoil. This distress, often a product of their own experiences of early abuse and attachment disruption in abusive relationships, can be re-enacted with their own infants.'

This re-enactment occurs in episodes of relational trauma (Schore, 2001, 2002, 2009a, 2009b, in press). Interdisciplinary evidence indicates that the infant's psychobiological reaction to severe interpersonal stressors comprises two separate response patterns, hyperarousal and dissociation. Beebe (2000: 436) describes the initial state of '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.

In the earliest stage of threat, the child's sudden alarm or startle reaction indicates activation of the infant's right hemisphere (Bradley et al., 1996). This, in turn, evokes a sudden increase of the sympathetic branch of the ANS, resulting in significantly elevated heart rate (cardiac acceleration), blood pressure, and respiration. Distress is expressed in 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 (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., 1990).

But a second, later forming reaction to relational trauma is dissociation, in which the child disengages from stimuli in the external world – traumatized infants are observed to be 'staring off into space with a glazed look'. Tronick and Weinberg (1997: 66) note that:

> when 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.

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. 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, 2003a, 2003b). In writings on psychic trauma and 'emotional surrender', Anna Freud (1951/1968, 1964/1969) also referred to helplessness, defined as a state of 'disorientation and powerlessness' that the organism experiences in the traumatic moment. 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: 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 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 that allows the infant to maintain homeostasis in the face of the internal state of sympathetic hyperarousal. It is seldom acknowledged that sympathetic energy-expending hyperarousal *and* parasympathetic energy-conserving hypoarousal are both 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 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: 75).

There is now agreement that sympathetic nervous system activity manifests in tight engagement with the external environment and a 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). The traumatized infant's sudden switch from high-energy sympathetic hyperarousal to low-energy parasympathetic dissociation is reflected in Porges' (1997: 75) 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.

Similarly, Krystal (1988: 114–115) describes 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.

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 et al., 1999).

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) and is associated with prenatal and/or postnatal maternal alcohol or cocaine use (Espinosa et al., 2001). Hesse and Main (1999) note that Type D disorganization and disorientation is phenotypically similar to dissociative states. Main and Solomon (1986) conclude 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, these authors assert that frightening parents places infants in an irresolvable bind wherein they cannot approach the mother, shift their attention, or flee. These infants are utterly unable to generate a coherent strategy to actively cope with their frightening parents.

Main and Solomon detail the uniquely bizarre behaviors of 12-month-old Type D infants in the Strange Situation procedure. These infants displayed brief (frequently only 10–30 s) but significant interruptions of organized behavior. At such times, Type D infants may exhibit a contradictory behavior pattern such as 'backing' towards the parent rather than approaching face to face. Main and Solomon (1986: 117) note that:

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

Maltreated infants exhibit apprehension, confusion, and very rapid shifts of state during the Strange Situation. Main and Solomon (1986: 119) 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.

These behaviors are not restricted to the infant's interactions with the mother. Indeed, 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 'leaned forehead against the wall for several seconds, looking back in apparent terror'. These infants exhibit 'behavioral stilling' – that is, 'dazed' behavior and depressed affect, behavioral manifestations of dissociation. One infant 'became for a moment excessively still, staring into space as though completely out of contact with self, environment, and parent'. Another showed 'a dazed facial appearance . . . accompanied by a stilling of all body movement, and sometimes a freezing of limbs which had been in motion'. Yet another 'fell face-down on the floor in a depressed posture prior to separation, stilling all body movements' Guedeney and Fermanian (2001) offer an alarm distress scale that assesses the sustained withdrawal that is associated with disorganized attachment. This withdrawal state is expressed in 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. In this 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 severe inhibition of nonverbal communication and interactive affect regulation 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 arousal intensification is ultimately followed by bodily collapse, loss of postural control, withdrawal, gaze aversion, sad facial expression, and self-comforting behavior.

Furthermore, 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 American Psychiatric Association's *Diagnostic and Statistical Manual* 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. He 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 Strange Situation and Still-Face biphasic induction of arousal and affect dysregulation occurs in face-to-face communications with the mother. The mother's face is the most potent visual stimulus in the child's world, but it is well known that direct gaze can mediate not only loving but also aggressive messages. Hesse and Main (1999: 511) describe the 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. The image of this aggressive face and the associated alterations in the infant's bodily state are indelibly imprinted into limbic circuits.

Main and Solomon (1986) document 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. Indeed, studies show that the caregiver of Type D infants exhibits dissociated, trancelike, and fearful behavior. Current research underscores a link between frightening maternal behavior, dissociation, and disorganized infant attachment (Schuengel et al., 1999). In recent work, Hesse and Main (2006: 320) 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'. In describing the mother as she submits to the freeze state, they note (321):

> Here the parent appears to have become completely unresponsive to, or even [un]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.

In an early history of traumatic attachment the developing infant/toddler is too frequently exposed to a massively misattuning primary caregiver who triggers and does not repair long-lasting intensely dysregulated states. The growth-inhibiting environment of relational trauma generates dense and prolonged levels of negative affect associated with extremely stressful states of hyperarousal and hypoarousal. And so for self-protective purposes it severely restricts its overt expressions of an attachment need for dyadic regulation. The child thus significantly reduces the output of its right-lateralized emotion-processing, limbic–autonomic attachment system. When one 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, detrimental to the substantial amounts of energy required for critical period biosynthetic processes.

During these episodes, the infant is matching the rhythmic structures of the mother's dysregulated states, and this synchronization is 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. Infants designated as 'very fearful' at seven months show larger ERPs over the right hemisphere when viewing fearful facial expressions (De Haan et al., 2004). An EEG study of five-month-old infants observes increased theta activity over the right posterior temporal area while they are looking at a blank face (Bazhenova et al., 2007).

It is now established that maternal care influences both the infant's reactivity (Menard et al., 2004) and the infant's defensive responses to threat (Parent et al., 2005). These dyadic processes 'serve as the basis for the transmission of individual differences in stress responses from mother to offspring' (Weaver et al., 2004: 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 intersubjective psychopathogenetic mechanism thus mediates the psychobiological intergenerational transmission of both relational trauma and the dissociative defense against overwhelming and dysregulating affective states. In accord with this model, research now indicates that severe early maternal dysfunction is associated with high dissociation in psychiatric patients (Draijer & Langeland, 1999), and that physical abuse and parental dysfunction on the part of the mother – not the father – is associated with somatoform dissociative symptoms (Roelofs et al., 2002).

From a developmental psychology viewpoint, the profound negative psychological effect of relational trauma (early abuse and neglect) is the generation of a disorganized–disoriented attachment that endures over the later stages of childhood, adolescence and adulthood, and acts as a risk factor for later psychiatric disorders (Schore, 2001, 2002, 2003a). From a developmental neuroscience perspective, the immediate detrimental impact is on the altered metabolic processes that poorly sustain critical period growth of the developing right brain, and the lasting impairment is an immature and functionally limited right-brain capacity to regulate later life stressors that generate intense affect states. Relational traumatic experiences are stored in imagistic procedural memory of the visuospatial right hemisphere (Schiffer et al., 1995), the locus of implicit (Hugdahl, 1995) and autobiographical (Markowitsch et al., 2000) memory. 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 characterologically access the auto-regulating, affect-deadening defense of pathological dissociation.

Enduring effect of relational trauma on right brain development: Impaired emotion processing and pathological dissociation

Neuropsychoanalytic authors now contend that 'If children grow up with dominant experiences of separation, distress, fear and rage, then they will go down a bad pathogenic developmental pathway, and it's not just a bad psychological pathway but a bad neurological pathway' (Watt, 2003: 109). Current workers in the field of developmental traumatology now agree that the overwhelming stress of maltreatment in childhood is associated with adverse influences on not just behavior, but also brain development (de Bellis et al., 1999), especially the right brain which is dominant for coping with negative affects (Davidson et al., 1990) and for 'regulating stress- and emotion-related processes' (Sullivan & Dufresne, 2006). Describing the essential survival functions of this lateralized system, Schutz (2005: 15) notes:

> 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.

In states of pathological dissociation the right brain's 'red phone line' is dead. The right brain is fundamentally involved in an avoidant defensive mechanism for coping with emotional stress, including the passive survival strategy of dissociation. These adaptive right brain functions are impaired in histories of early relational trauma. A large body of psychiatric, psychological, and neurological studies supports the link between childhood trauma and pathological dissociation (e.g., Dikel et al., 2003; Diseth, 2005; Liotti, 2004; Merckelbach & Muris, 2001; Macfie et al., 2001).

Recent neurobiological data can also be utilized to create models of the psychopathogenetic mechanism by which attachment trauma negatively impacts right brain development. Adamec and colleagues (2003) report experimental data that 'implicate neuroplasticity in right hemispheric limbic circuitry in mediating long-lasting changes in negative affect following brief but severe stress'. 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'. 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 excitotoxic stress (Schore, 2001, 2002).

Recall that right cortical areas and their connections with right subcortical structures are in a critical period of growth during early human development. The massive psychobiological stress associated with attachment trauma impairs the development of this system, and sets the stage for the characterological use of right-brain pathological dissociation when encountering later stressors. Converging evidence indicates that early abuse negatively impacts limbic 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 leads to self-modulation of painful affect by directing attention away from internal emotional states (Lane et al., 1997). The right brain, dominant for attention (Raz, 2004) and pain processing (Symonds et al., 2006) thus generates dissociation, a defense by which intense negative affects associated with emotional pain are blocked from consciousness.

Congruent with this developmental model, Spitzer et al. report a transcranial magnetic stimulation study of adults and 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' (2004: 168). In functional magnetic resonance imaging research, Lanius et al. (2005) show predominantly right hemispheric activation in psychiatric patients while they are dissociating, and conclude that dissociation, an escape from the overwhelming emotions associated with the traumatic memory, can be interpreted as representing a nonverbal response to the traumatic memory. In the clinical literature, 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'. Dissociation is then automatically and immediately triggered as the fundamental defense to the arousal dysregulation of overwhelming affective states.

Both researchers and clinicians are now exploring the evolution of a developmentally impaired regulatory system and provide 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, in press). This hemisphere, more than the left, is densely reciprocally interconnected with emotion-processing limbic regions, as well as with subcortical areas that generate both the arousal and autonomic bodily-based aspects of emotions. Recall, SNS activity is manifest in tight engagement with the external environment and a high level of energy mobilization, while the parasympathetic component drives disengagement from the external environment and utilizes low levels of internal energy. These ANS components are 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 expressed in bodily based visceral–somatic disturbances.

Pathological dissociative detachment represents a bottom-line defensive state driven by fear–terror, in which the stressed individual copes by pervasively and diffusely disengaging attention 'from both the *outer and inner* worlds' (Allen et al., 1999: 164, emphasis added). I have suggested that the 'inner world' is more than cognitions, the realm of bodily processes, central components of emotional states (Schore, 1994). 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'. 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 – their body, their actions, and their sense of identity (Allen et al., 1999). This 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'.

In a number of works I have offered interdisciplinary evidence that the implicit self, equated with Freud's system *Ucs*, is located in the right brain (Schore, 1994, 2003a, 2007). The lower subcortical levels of the right brain (the deep unconscious) contain all the major motivational systems (including attachment, fear, sexuality, aggression, etc.) and generate the somatic autonomic expressions and arousal intensities of all emotional states. On the other hand, higher orbitofrontal–limbic levels of the right hemisphere generate a conscious emotional state that expresses the affective output of these motivational systems. In an optimal attachment scenario, this right lateralized hierarchical prefrontal system, the system *Pcs* performs an essential adaptive motivational function – the relatively fluid switching of internal bodily 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; it negatively impacts the early organization of right brain survival mechanisms that operate beneath levels of conscious awareness. Pathological dissociation is manifest in a maladaptive highly defensive rigid, closed self system, one that responds to even low levels of intersubjective stress with parasympathetic dorsal vagal parasympathetic hypoarousal, heart rate deceleration, and passive disengagement. This fragile unconscious system is susceptible to relational stress-induced mind–body metabolic collapse and thereby a loss of

energy-dependent synaptic connectivity within the right brain, expressed in a sudden implosion of the implicit self and a rupture of self-continuity. This collapse of the implicit self is signaled by the amplification of the parasympathetic affects of shame and disgust, and by the cognitions of hopelessness and helplessness. Because the right hemisphere mediates the communication and regulation of emotional states, the rupture of intersubjectivity is accompanied by an instant dissipation of safety and trust.

Dissociation thus reflects the 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 on a moment-to-moment basis integrate them with internal stimuli (interoceptive information from the body, somatic markers, the 'felt experience'). This failure of integration of the higher right hemisphere with the lower right brain induces an instant collapse of both subjectivity and intersubjectivity. Stressful affects, especially those associated with emotional pain, are thus not experienced in consciousness. Dissociated affect is thus unconscious affect, described by Freud: 'Unconscious ideas continue to exist after repression as actual structures in the system *Ucs*, whereas all that corresponds in that system to unconscious affects is a potential beginning which is prevented from developing' (1915: 178).

At all points of the lifespan, although dissociation represents an effective short-term strategy, it is detrimental to long-term functioning, specifically by preventing exposure to potential right-brain socioemotional attachment object learning experiences embedded in intimate intersubjective contexts that are necessary for emotional growth. 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. Clinical research shows pathological dissociation, a primitive defense against overwhelming affects, is a key feature of not only reactive attachment disorder of infants and pediatric maltreatment disorder, but also dissociative identity disorder, posttraumatic stress disorder, psychotic disorders, eating disorders, substance abuse and alcoholism, somatoform disorders and sociopathic and borderline personality disorders.

Psychotherapy with such patients needs to attend to more than the significant dysregulation of affect that characterizes these severe self pathologies. It must also address the early forming defense that blocks these overwhelming affects from reaching consciousness, thereby denying the possibility of interactive regulation and the organization of more complex right-brain stress regulation. Bromberg (2006) observes that in the clinical encounter pathological dissociation acts as an 'early warning system' that anticipates potential affect dysregulation before the trauma arrives. The current paradigm shift from cognition to affect also includes a shift from repression to the survival strategy of dissociation as the major mechanism of

psychopathogenesis. It thus represents a major obstacle to the intersubjective change process in all affectively focused psychotherapies (Schore, 2007, in press), and to the effectiveness of early intervention programs, a major theme of this book.

References

Adamec, R.E., Blundell, J. & Burton, P. (2003) Phosphorylated cyclic AMP response element bonding protein expression induced in the periaqueductal gray by predator stress: Its relationship to the stress experience, behaviour, and limbic neural plasticity. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, 27: 1243–1267.

Allen, J.G., Console, D.A. & Lewis, L. (1999) Dissociative detachment and memory impairment: Reversible amnesia or encoding failure? *Comprehensive Psychiatry*, 40: 160–171.

Barach, P.M.M. (1991) Multiple personality disorder as an attachment disorder. *Dissociation*, 4: 117–123.

Bazhenova, O.V., Stroganova, T.A., Doussard-Roosevelt, J.A., Posikera, I.A. & Porges, S.W. (2007) Physiological responses of 5-month-old infants to smiling and blank faces. *International Journal of Psychophysiology*, 63: 64–76.

Beebe, B. (2000) Coconstructing mother–infant distress: The microsychrony of maternal impingement and infant avoidance in the face-to-face encounter. *Psychoanalytic Inquiry*, 20: 412–440.

Beebe, B. & Lachmann, F.M. (1994) Representations and internalization in infancy: Three principles of salience. *Psychoanalytic Psychology*, 11: 127–165.

Bion, W.R. (1962) *Learning from experience*. London: Heinemann.

Bock, J., Murmu, R.P., Fredman, N., Leshem, M., & Braun, K. (2008) Refinement of dendritic and synaptic networks in the rodent anterior cingulate and orbitofrontal cortex: Critical impact of early and late social experience. *Developmental Neurobiology*, 68: 685–695.

Bogolepova, I.N. & Malofeeva, L.I. (2001) Characteristics of the development of speech motor areas 44 and 45 in the left and right hemispheres of the human brain in early post-natal ontogenesis, *Neuroscience and Behavioural Physiology*, 31: 349–354.

Bourne, V.J. & Todd, B.K. (2004) When left means right: An explanation of the left cradling bias in terms of right hemisphere specializations. *Developmental Science*, 7: 19–24.

Bowlby, J. (1988) *A secure base: Parent–child attachment and healthy human development*. New York: Basic Books.

Bradley, M., Cuthbert, B. N. & Lang, P. J. (1996) Lateralized startle probes in the study of emotion. *Psychophysiology*, 33: 156–161.

Bradshaw, G.A. & Schore, A.N. (2007) How elephants are opening doors: developmental neuroethology, attachment and social context. *Ethology*, 113: 426–436.

Bromberg, P.M. (2006) *Awakening the dreamer: Clinical journeys*. Mahwah, NJ: Analytic Press.

Brown, M.R., Fisher, L.A., Spiess, J., Rivier, C., Rivier, J. & Vale, W. (1982) Corticotropin-releasing factor: Actions on the sympathetic nervous system and metabolism. *Endocrinology*, 111: 928–931.

Carlson, V., Cicchetti, D., Barnett, D. & Braunwald, K. (1989) Disorganized/disoriented attachment relationships in maltreated infants. *Developmental Psychology*, 25: 525–531.

Chiron C., Jambaque, I., Nabbout, R., Lounes, R., Syrota, A. & Dulac, O. (1997) The right brain hemisphere is dominant in human infants. *Brain*, 120: 1057–1065.

Cortina, M. & Liotti, G. (2007) New approaches to understanding unconscious processes: Implicit and explicit memory systems. *International Forum of Psychoanalysis*, 16: 204–212.

Cozolino, L. (2002) *The neuroscience of psychotherapy*. New York: Norton.

Crucian, G.P., Hughes, J.D., Barrett, A.M., Williamson, D.J.G., Bauer, R.M., Bowres, D. et al. (2000) Emotional and physiological responses to false feedback. *Cortex*, 36: 623–647.

Dapretto, M., Davies, M.S., Pfeifer, J.H., Scott, A.A., Sigman, M., Bookheimer, S.Y. et al. (2006) Understanding emotions in others: Mirror neuron dysfunction in children with autism spectrum disorders. *Nature Neuroscience*, 9: 28–31.

Davidson, R.J., Ekman, P., Saron, C., Senulis, J. & Friesen, W.V. (1990) Approach/withdrawal and cerebral asymmetry: I. Emotional expression and brain physiology. *Journal of Personality and Social Psychology*, 58: 330–341.

de Bellis, M.D., Baum, A.S., Birmaher, B., Keshavan, M.S., Eccard, C.H., Boring, A.M. et al. (1999) Developmental traumatology Part I: Biological stress systems. *Biological Psychiatry*, 45: 1259–1270.

De Haan, M., Belsky, J., Reid, V., Volein, A. & Johnson, M.H. (2004) Maternal personality and infants' neural and visual responsivity to facial expressions of emotion. *Journal of Child Psychology and Psychiatry*, 45: 1209–1218.

Devinsky, O. (2000) Right cerebral hemisphere dominance for a sense of corporeal and emotional self. *Epilepsy & Behaviour*, 1: 60–73.

Dikel, T.N., Fennell, E. & Gilmore, R.L. (2003) Post traumatic stress disorder, dissociation, and sexual abuse history in epileptic nonepileptic seizure patients. *Epilepsy and Behaviour*, 4: 644–650.

Diseth, T.H. (2005) Dissociation in children and adolescents as reaction to trauma – an overview of conceptual issues and neurobiological factors. *Nordic Journal of Psychiatry*, 59: 79–91.

Dixon, A. K. (1998) Ethological strategies for defense in animals and humans: Their role in some psychiatric disorders. *British Journal of Medical Psychology*, 71: 417–445.

Dorpat, T.L. (2001) Primary process communication. *Psychoanalytic Inquiry*, 3: 448–463.

Draijer, N. & Langeland, W. (1999) Childhood trauma and perceived parental dysfunction in the etiology of dissociative symptoms in psychiatric inpatients. *American Journal of Psychiatry*, 156: 379–385.

Espinosa, M., Beckwith, L., Howard, J., Tyler, R. & Swanson, K. (2001) Maternal psychopathology and attachment in toddlers of heavy cocaine-using mothers. *Infant Mental Health Journal*, 22: 316–333.

Field, T., Pickens, J., Fox, N.A., Nawrocki, T. & Gonzalez, J. (1995) Vagal tone in infants of depressed mothers. *Development and Psychopathology*, 7: 227–231.

Fonagy, P. & Target, M. (1997) Attachment and reflective function: Their role in self-organization. *Development and Psychopathology*, 9: 679–700.

Fonagy, P. & Target, M. (2002) Early intervention and the development of self-regulation. *Psychoanalytic Inquiry*, 22: 307–335.

Fonagy, P. & Target, M. (2005) Bridging the transmission gap: An end to an important mystery of attachment research? *Attachment & Human Development*, 7: 333–343.

Freud, A. (1951/1968) Notes on the connection between the states of negativism and psychic surrender. In *The writings of Anna Freud* (Vol. 4, pp. 256–259). New York: International Universities Press.

Freud, A. (1964/1969) Comments on psychic trauma. In *The writings of Anna Freud*, (Vol. 4, pp. 221–241). New York: International Universiites Press.

Freud, S. (1915) The unconscious. *Standard Edition* (Vol. 14, pp. 159–205). London: Hogarth Press, 1957.

Gadea, M., Gomez, C., Gonzalez-Bono, R.E. & Salvador, A. (2005) Increased cortisol and decreased right ear advantage (REA) in dichotic listening following a negative mood induction. *Psychoneuroendocrinology*, 30: 129–138.

Gainotti, G. (2000) Neuropsychological theories of emotion. In J. Borod (Ed.), *The neuropsychology of emotion*. New York: Oxford University Press.

Gallagher, H.L. & Frith, C.D. (2004) Dissociable neural pathways for the perception and recognition of expressive and instrumental gestures. *Neuropsychologia*, 42: 1725–1736.

Geschwind, N. & Galaburda, A. M. (1987) *Cerebral lateralization: Biological mechanisms, associations, and pathology*. Boston: MIT Press.

Grossman, T., Johnson, M.H., Farroni, T. & Csibra, G. (2007) Social perception in the infant brain: Gamma oscillatory activity in response to eye gaze. *Social Cognitive and Affective Neuroscience*, 2: 284–291.

Guedeney, A. & Fermanian, J. (2001) A validity and reliability study of assessment and screening for sustained withdrawal in infancy: The alarm distress scale. *Infant Mental Health Journal*, 22: 559–575.

Gunnar, M.R. (2000) Early adversity and the development of stress reactivity and regulation. In C.A. Nelson (Ed.), *The Minnesota Symposium on Child Psychology: Vol. 31, The effects of early adversity on neurobehavioural development* (pp. 163–200). Mahwah, NJ: Lawrence Erlbaum Associates, Inc.

Henry, J.P. (1993) Psychological and physiological responses to stress: The right hemisphere and the hypothalamo–pituitary–adrenal axis, an inquiry into problems of human bonding. *Integrative Physiological & Behavioural Science*, 28: 369–387.

Hesse, E. & Main, M.M. (1999) Second-generation effects of unresolved trauma in nonmaltreating parents: dissociated, frightened, and threatening parental behaviour. *Psychoanalytic Inquiry*, 19: 481–540.

Hesse, E. & Main, M. (2006) Frightened, threatening, and dissociative parental behavior in low-risk samples: Description, discussion, and interpretations. *Development and Psychopathology*, 18(2): 309–343.

Homae, F., Watanabe, H., Nakano, T., Asakawa, K. & Taga, G. (2006) The right hemisphere of sleeping infants perceives sentential prosody. *Neuroscience Research*, 54: 276–280.

Howard, M.F. & Reggia, J.A. (2007) A theory of the visual system biology underlying development of spatial frequency lateralization. *Brain and Cognition*, 64: 111–123.

Hugdahl, K. (1995) Classical conditioning and implicit learning: The right hemisphere hypothesis. In R. J. Davidson & K. Hugdahl (Eds.), *Brain asymmetry* (pp. 235–267). Cambridge, MA: MIT Press.

Janet, P. (1889) *L'automatisme psychologique*. Paris: Alcan.

Kalsched, D. (2005) Hope versus hopelessness in the psychoanalytic situation and Dante's *Divine Comedy*. *Spring*, 72: 167–187.

Kestenberg, J. (1985) The flow of empathy and trust between mother and child. In E.J. Anthony & G.H. Pollack (Eds.), *Parental influences in health and disease* (pp. 137–163). Boston: Little, Brown.

Kinney, H.C., Brody, B.A., Kloman, A.S. & Gilles, F.H. (1988) Sequence of central nervous system myelination in human infancy. II. Patterns of myelination in autopsied infants. *Journal of Neuropathology and Experimental Neurology*, 47: 217–234.

Knickmeyer, R.C., Gouttard, S., Kang, C., Evans, D., Wilber, K., Smith, J.K., et al. (2008) A structural MRI study of human brain development from birth to 2 years. *Journal of Neuroscience*, 28: 12176–12182.

Krystal, H. (1988) *Integration and self-healing: Affect–trauma–alexithymia*. Hillsdale, NJ: Analytic Press.

Kvetnansky, R., Jezova, D., Oprsalova, Z., Foldes, O., Michjlovskij, N., Dobrakovova, M. et al. (1990) Regulation of the sympathetic nervous system by circulating vasopressin. In J.C. Porter & D. Jezova (Eds.), *Circulating regulatory factors and neuroendocrine function* (pp. 113–134). New York: Plenum Press.

Lane, R.D., Ahern, G.L., Schwartz, G.E. & Kaszniak, A.W. (1997) Is alexithymia the emotional equivalent of blindsight? *Biological Psychiatry*, 42: 834–844.

Lanius, R.A., Williamson, P.C., Bluhm, R.L., Densmore, M., Boksman, K., Neufeld, R.W.J. et al. (2005) Functional connectivity of dissociative responses in post-traumatic stress disorder: A functional magnetic resonance imaging investigation. *Biological Psychiatry*, 57: 873–884.

Le Grand, R., Mondloch, C., Maurer, D. & Brent, H. P. (2003) Expert face processing requires visual input to the right hemisphere during infancy. *Nature Neuroscience*, 6: 1108–1112.

Lenzi, D., Trentini, C., Pantano, P., Macaluso, E., Iacaboni, M., Lenzi, G.I. et al. (2009) Neural basis of maternal communication and emotional expression processing during infant preverbal stage. *Cerebral Cortex*, 19(5): 1124–1133.

Liotti, G. (2004) Trauma, dissociation, and disorganized attachment: Three strands of a single braid. *Psychotherapy: Theory, Research, Training*, 41: 472–486.

Loewenstein, R.J. (1996) Dissociative amnesia and dissociative fugue. In L.K. Michaelson & W.J. Ray (Eds.), *Handbook of dissociation: Theoretical, empirical, and clinical perspectives* (pp. 307–336) New York: Plenum.

Macfie, J., Cicchetti, D. & Toth, S.L. (2001) Dissociation in maltreated versus nonmaltreated preschool-age children. *Child Abuse & Neglect*, 25: 1253–1267.

Main, M. & Solomon, J. (1986) Discovery of an insecure–disorganized/disoriented attachment pattern: Procedures, findings and implications for the classification of behaviour. In T.B. Brazelton & M.W. Yogman (Eds.), *Affective development in infancy* (pp. 95–124). Norwood, NJ: Ablex.

Markowitsch, H.J., Reinkemeier, A., Kessler, J., Koyuncu, A. & Heiss, W.-D. (2000) Right amygdalar and temperofrontal activation during autobiographical, but not fictitious memory retrieval. *Behavioural Neurology*, 12: 181–190.

Menard, J.L., Champagne, D.L. & Meaney, M.J.P. (2004) Variations of maternal care differentially influence 'fear' reactivity in response to the shock-probe burying test. *Neuroscience*, 129: 297–308.

Merckelbach, H. & Muris, P. (2001) The causal link between self-reported trauma and dissociation: A critical review. *Behaviour Research and Therapy*, 39: 245–254.

Minagawa-Kawai, Y., Matsuoka, S., Dan, I., Naoi, N., Nakamura, K. & Kojima, S. (2009) Prefrontal activation associated with social attachment: Facial–emotion recognition in mothers and infants. *Cerebral Cortex*, 19: 284–292.

Mollon, P. (1996) *Multiple selves, multiple voices: Working with trauma, violation and dissociation*. Chichester, UK: Wiley.

Nagy, E. (2006) From imitation to conversation: The first dialogues with human neonates. *Infant and Child Development*, 15: 223–232.

Nakato, E., Otsuka, Y., Kanazawa, S., Yamaguchi, M.K., Watanabe, S. & Kakiga, R. (2009) When do infants differentiate profile face from frontal face? A near-infrared spectroscopic study. *Human Brain Mapping*, 30: 462–472.

Newman, L. & Stevenson, C. (2005) Parenting and borderline personality disorder. *Clinical Child Psychology and Psychiatry*, 10: 385–394.

Nijenhuis, E.R.S. (2000) Somatoform dissociation: Major symptoms of dissociative disorders. *Journal of Trauma & Dissociation*, 1: 7–32.

Noriuchi, M., Kikuchi, Y. & Senoo, A. (2008) The functional neuroanatomy of maternal love: Mother's response to infant's attachment behaviours. *Biological Psychiatry*, 63: 415–423.

Otsuka, Y., Nakato, E., Kanazawa, S., Yamaguchi, M.K., Watanabe, S. & Kakigi, R. (2007) Neural activation to upright and inverted faces in infants measured by near infrared spectroscopy. *NeuroImage*, 34: 399–406.

Papousek, H. & Papousek, M. (1997) Fragile aspects of early social integration. In L. Murray & P.J. Cooper (Eds.), *Postpartum depression and child development* (pp. 35–53). New York: Guilford Press.

Parent, C., Zhang, T.-Y., Caldji, C., Bagot, R., Champagne, F.A., Pruessner, J. et al. (2005) Maternal care and individual differences in defensive responses. *Current Directions in Psychological Science*, 12: 229–233.

Pelphrey, K.A., Viola, R.J. & McCarthy, G. (2004) When strangers pass: Processing of mutual and averted social gaze in the superior temporal sulcus. *Psychological Science*, 15: 598–603.

Porges, S.W. (1997) Emotion: An evolutionary by-product of the neural regulation of the autonomic nervous system. *Annals of the New York Academy of Sciences*, 807: 62–77.

Powles, W.E. (1992) *Human development and homeostasis*. Madison, CT: International Universities Press.

Putnam, F. W. (1997) *Dissociation in children and adolescents: a developmental perspective*. New York: Guilford Press.

Raz, A. (2004) Anatomy of attentional networks. *Anatomical Record*, 281B: 21–36.

Recordati, G. (2003) A thermodynamic model of the sympathetic and parasympathetic nervous systems. *Autonomic Neuroscience: Basic and Clinical*, 103: 1–12.

Roelofs, K., Keijers, G.P.J., Hoogduin, K.A.L., Naring, G.W.B. & Moene, F.C. (2002) Childhood abuse in patients with conversion disorder. *American Journal of Psychiatry*, 159: 1908–1913.

Schiffer, F., Teicher, M. & Papanicolaou, A. (1995) Evoked potentials evidence for right brain activity during recall of traumatic memories. *Journal of Neuropsychiatry and Clinical Neuroscience*, 7: 169–175.

Schore, A.N. (1994) *Affect regulation and the origin of the self*. Mahwah, NJ: Lawrence Erlbaum Associates, Inc.

Schore, A.N. (2000) Attachment and the regulation of the right brain. *Attachment & Human Development*, 2: 23–47.

Schore, A.N. (2001) The effects of relational trauma on right brain development, affect regulation, and infant mental health. *Infant Mental Health Journal*, 22: 201–269.

Schore, A.N. (2002) Dysregulation of the right brain: A fundamental mechanism of traumatic attachment and the psychopathogenesis of posttraumatic stress disorder. *Australian & New Zealand Journal of Psychiatry*, 36: 9–30.

Schore, A.N. (2003a) *Affect regulation and the repair of the self*. New York: W.W. Norton.

Schore, A.N. (2003b) *Affect dysregulation and disorders of the self*. New York: W.W. Norton.

Schore, A.N. (2005) Attachment, affect regulation, and the developing right brain: Linking developmental neuroscience to pediatrics. *Pediatrics in Review*, 26: 204–211.

Schore, A.N. (2007) Review of *Awakening the dreamer: clinical journeys* by Philip M. Bromberg. *Psychoanalytic Dialogues*, 17: 753–767.

Schore, A.N. (2009a) Attachment trauma and the developing right brain: Origins of pathological dissociation. In P.F. Dell & J.A. O'Neil (Eds.), *Dissociation and the dissociative disorders: DSM-V and Beyond* (pp. 107–141). New York: Routledge.

Schore, A.N. (2009b) Relational trauma and the developing right brain: An interface of psychoanalytic self psychology and neuroscience. *Annals of the New York Academy of Sciences*, 1159: 189–203.

Schore, A.N. (in press) Right brain affect regulation: An essential mechanism of development, trauma, dissociation, and psychotherapy. In D. Fosha, D. Siegel & M. Solomon (Eds.), *The healing power of emotion: Affective neuroscience, development, and clinical practice*. New York: Norton.

Schore, J.R. & Schore, A.N. (2008) Modern attachment theory: The central role of affect regulation in development and treatment. *Clinical Social Work Journal*, 36: 9–20.

Schuengel, C., Bakersmans-Kranenburg, M.J. & Van IJzendoorn, M.H. (1999) Frightening maternal behaviour linking unresolved loss and disorganized infant attachment. *Journal of Consulting and Clinical Psychology*, 67: 54–63.

Schuetze, P. & Reid, H.M. (2005) Emotional lateralization in the second year of life: Evidence from oral asymmetries. *Laterality*, 10: 207–217.

Schutz, L.E. (2005) Broad-perspective perceptual disorder of the right hemisphere. *Neuropsychology Review*, 15: 11–27.

Siegel, A.M. (1996) *Heinz Kohut and the psychology of the self*. London and New York: Routledge.

Siegel, A.M. (1999) *The developing mind: Towards a neurobiology of interpersonal experience*. New York: Guilford Press.

Sieratzki, J.S. & Woll, B. (1996) Why do mothers cradle babies on the left? *The Lancet*, 347: 1746–1748.

Spitzer, C., Wilert, C., Grabe, H.-J., Rizos, T. & Freyberger, H. J. (2004) Dissociation, hemispheric asymmetry, and dysfunction of hemispheric interaction: A transcranial magnetic approach. *Journal of Neuropsychiatry and Clinical Neuroscience*, 16: 163–169.

Stern, D.N. (1985) *The interpersonal world of the infant*. New York: Basic Books.

Sullivan, R.M. & Gratton, A. (2002) Prefrontal cortical regulation of hypothalamic–pituitary–adrenal function in the rat and implications for psychopathology: Side matters. *Psychoneuroendocrinology*, 27: 99–114.

Sullivan, R.M. & Dufresne, M. M. (2006) Mesocortical dopamine and HPA axis regulation: Role of laterality and early environment. *Brain Research*, 1076: 49–59.

Suter, S.E., Huggenberger, H.J. & Schachinger, H. (2007) Cold pressor stress reduces left cradling preference in nulliparous human females. *Stress*, 10: 45–51.

Symonds, L.L., Gordon, N.S., Bixby, J.C. & Mande, M.M. (2006) Right-lateralized pain processing in the human cortex: An fMRI study. *Journal of Neurophysiology*, 95: 3823–3830.

Thierry, G., Vihman, M. & Roberts, M. (2003) Familiar words capture the attention of 11-month-olds in less than 250 ms. *NeuroReport*, 14: 2307–2310.

Trevarthen, C. (2001) The neurobiology of early communication: Intersubjective regulations in human brain development. In A.F. Kalverboer & A. Gramsbergen (Eds.), *Handbook on brain and behaviour in human development* (pp. 841–882). Dordrecht, The Netherlands: Kluwer.

Tronick, E.Z. (1989) Emotions and emotional communication in infants. *American Psychologist*, 44: 112–119.

Tronick, E.Z. (2004) Why is connection with others so critical? Dyadic meaning making, messiness and complexity governed selective processes which co-create and expand individuals' states of consciousness. In J. Nadel & D. Muir (Eds.), *Emotional development*. New York: Oxford University Press.

Tronick, E.Z. & Weinberg, M.K. (1997) Depressed mothers and infants: Failure to form dyadic states of consciousness. In L. Murray & P.J. Cooper (Eds.), *Postpartum depression in child development* (pp. 54–81). New York: Guilford Press.

Tucker, D.M. (1992) Developing emotions and cortical networks. In M.R. Gunnar & C.A. Nelson (Eds.), *Minnesota symposium on child psychology, Vol. 24, Developmental behavioural neuroscience* (pp. 75–128). Mahwah, NJ: Lawrence Erlbaum Associates, Inc.

Tzourio-Mazoyer, N., De Schonen, S., Crivello, F., Reutter, B., Aujard, Y. & Mazoyer, B. (2002) Neural correlates of woman face processing by 2-month-old infants. *NeuroImage*, 15: 454–461.

Watt, D.F. (2003) Psychotherapy in an age of neuroscience: Bridges to affective neuroscience. In J. Corrigall & H. Wilkinson (Eds.), *Revolutionary connections: Psychotherapy and neuroscience* (pp. 79–115). London: Karnac.

Weaver, I.C.G., Cervoni, N., Champagne, F.A., D'Alessio, A.C., Sharma, S., Seckl, J.R. et al. (2004) Epigenetic programming by maternal behaviour. *Nature Neuroscience*, 7: 847–854.

Winnicott, D.W. (1958) The capacity to be alone. *International Journal of Psycho-Analysis*, 39: 416–420.

Wismer Fries, A.B., Ziegler, T.E., Kurian, J.R., Jacoris, S. & Pollak, S.D. (2005) Early experience in humans is associated with changes in neuropeptides critical for regulating social behaviour. *Proceedings of the National Academy of Sciences of the United States of America*, 102: 17237–17240.

Yamada, H., Sadato, N., Konishi, Y., Muramoto, S., Kimura, K., Tanaka, M. et al.

(2000) A milestone for normal development of the infantile brain detected by functional MRI. *Neurology*, 55: 218–223.

Ziabreva, I., Poeggel, G., Schnabel, R. & Braun, K. (2003) Separation-induced receptor changes in the hippocampus and amygdala of *Octodon degus*: Influence of maternal vocalizations. *Journal of Neuroscience*, 23: 5329–5336.

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