

## Healing the Body-Mind in Heart-Centered Therapies

David Hartman, MSW and Diane Zimberoff, M.A. \*

**Abstract:** Some of the most profound influences on human behavior may be found within the deep evolutionary streams of human nature, flowing through the hormonal and nervous systems, regulated by the instinctual “reptilian brain” (limbic system). These archaic, archetypal patterns, when denied or thwarted or undischarged, split off from the whole self and become trapped in the body. That is where we find them, and how we heal them. We assess the damaging effects of traumatic response in the womb and in childhood. If a person tends toward hyperarousal (fight/flight) response that is not effectively discharged, his/her body will tend to utilize *parasympathetic dissociation* as a defensive effort to achieve the semblance of homeostasis. If a person tends toward hyporarousal (freeze) response that is not effectively discharged, his/her body will tend to utilize *sympathetic dissociation* to achieve the semblance of homeostasis. The area of the body that is *not* feeling (*parasympathetic dissociation*) can be equally as important an indicator of stored trauma as body parts that *do* feel (*sympathetic dissociation*). We review the Theory of Structural Dissociation proposed by Nijenhuis as a way to understand the common alternation between re-experiencing trauma and detachment from or unawareness of the trauma. The primary emotions are regulated neurally.

The emotional operating systems proposed by Panksepp can be divided into the primordial set (FEAR, RAGE, and SEEKING) basic to survival; and the social set (LUST, PANIC, CARE and PLAY) characteristic of mammals, which depend on the creation and maintenance of social bonds for survival. Before directly addressing the memory of a traumatic event in therapy, three areas must first be strengthened: containment of what feels overwhelming, positive body awareness to promote a sense of the body and its sensations as friend, not foe, and dual time awareness to separate the past from what is occurring now. We establish the value of this neurophysiology as it elucidates the process of spiritual awakening. Finally, we address the implications of these observations for trauma resolution in a clinical setting.

Some of the most profound influences on human behavior may be found within the deep evolutionary streams of human nature, flowing through the hormonal and nervous systems, regulated by the instinctual “reptilian brain” (limbic system). That part of the brain controls emotional thought, including rage, fear, aggression, and arousal. The influence of these systems is especially direct as imminent threat increases. And yet the “new brain,” the prefrontal cortex and especially the hippocampus, are capable of moderating that influence, putting into context the reality of the threat, and making mindful conscious choices of behavior.

---

\*The Wellness Institute, 3716 - 274<sup>th</sup> Avenue SE, Issaquah, WA 98029 USA 425-391-9716

These influences operate in a number of different layers of depth within the continuum of conscious/ unconscious, yet are accessible through the body. These archaic, archetypal patterns, when denied or thwarted or undischarged, split off from the whole self and become trapped in the body. That is where we find them, and how we heal them. “According to Jung, bodily experiences relating to instinctual discharges constitute the most deeply unconscious psychic elements, which can never become completely conscious” (Sidoli & Blakemore, 2000, p. 91).

We will present a detailed discussion of the somatic response to stress and trauma, then of dissociation and embodiment, and finally a summary of treatment guidelines.

### *Nervous system response to threat*

The *sympathetic* nervous system is primarily aroused in states of stress, both positive (excitement) and negative (fear). It responds to extreme traumatic threat by telling the body to prepare for defensive action (fight or flight) through increasing respiration and heart rate to provide more oxygen, sending blood away from the skin and into the muscles for quick movement. Signs of hyperarousal include cold pale skin, dilated pupils, raised blood pressure. There is an upsurge of the feelings that are experienced as coming UP, i.e., anger, fear, excitement, desire, hatred. These feelings, if expressed, involve outward movement (toward or away from) (Carroll, 2001).

The *parasympathetic* nervous system is primarily activated in states of rest and relaxation. However, when death may be imminent or the traumatic threat is prolonged, the parasympathetic system creates a state of passive defense (freeze). In this state of freeze, the victim of trauma enters an altered reality; it is a passive form of dissociation. Time slows down and there is no awareness of fear or pain. Signs of hypoarousal include decreased heart rate and respiration, warm and flushed skin, normally reactive pupils (not dilated), and lowered blood pressure. Here the feelings are of coming DOWN – disappointment, grief, shame, guilt, despair, or else contentment, peacefulness, satisfaction. These feelings, if expressed, draw the energy inward.

Ideally the sympathetic and parasympathetic systems counterbalance each other, providing a timely antidote to swing the pendulum back toward center from either extreme. Wilhelm Reich perceived their reciprocal action as part of a four-beat cycle: tension-charge-discharge-relaxation, which offers a holistic model embracing both psychological and

physiological functioning. Tension is created by the awareness of an unmet need or desire; charge by activation of the sympathetic system; discharge by resolution of the conflict; and relaxation by activation of the parasympathetic system. When the discharge phase of the cycle is blocked or interrupted, the charge continues to build and becomes at first uncomfortable, then frenzied, and finally unbearable. The individual must develop defenses against such toxic tension, and those defenses of splitting, dissociation and suppression soon enough become deeply embedded patterns.

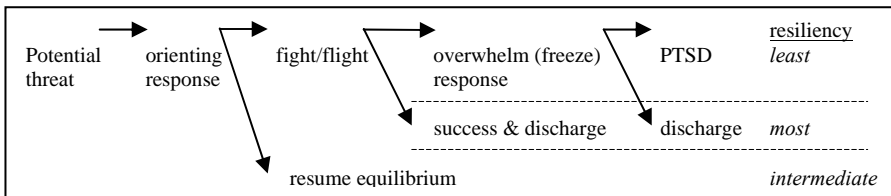


Figure 1.

### *Orienting response*

Engaged in daily activity, the prey animal’s “early warning system” senses a potential threat. The animal enters a state of “alert immobility,” becoming almost entirely motionless, and focuses its attention on sensory cues regarding the threat (primarily sight, sound and smell). If no danger is detected, the animal resumes normal activity without wasting energy on a fight/flight response. Mammals (unlike reptiles) may voluntarily continue to sustain their attention out of curiosity or hopes of a reward, or they may continue with facial expressions and vocalizations to foster communication. We shall see that this option creates *voluntary* attention and complex social interaction. These voluntary options are available, however, only if the animal feels safe enough to override the “reptilian” reflexive response. Obviously, if danger is imminent, then fight or flight is initiated.

Actually, this “early warning system” is continually evaluating risk by processing information from the environment through the senses. This pertains to both the external environment (e.g., a dangerous person or situation) or the internal environment (e.g., fever, pain, or physical illness). Porges (2004) uses the term *neuroception* to describe how neural circuits distinguish whether situations or people are safe, dangerous, or life

threatening. Neuroception takes place in primitive parts of the brain, without our conscious awareness. This is important to know because if that primitive autonomic system has become confused as a result of repeated threats, anxiety, abuse or neglect, it may either signal imminent danger when there is none or fail to recognize danger when it is in fact present, or both. An example is maltreated and institutionalized children with Reactive Attachment Disorder, who tend to be either inhibited (emotionally withdrawn and unresponsive) or uninhibited (indiscriminate in their attachment behavior) (Porges, 2004; Zeanah, 2000).

Certain neuropeptide “messenger molecules”, such as oxytocin and serotonin, are released in the brain during activities that help establish social bonds. Thus, when people sense that their environment is safe, the release of oxytocin allows them to enjoy the comfort of an embrace without fear, interpreting the immobility as a nurturing containment. But if their nervous system identifies someone as dangerous, no oxytocin is released and they struggle against the attempted embrace, interpreting the immobility as a threatening restraint. Oxytocin allows one to self-soothe and calm the internal state, and is also intricately involved in emotionally healing chronic states of stress, anxiety, and depression, as well as spiritually awakening. The implications of the neurophysiological responses to stress for spiritual experience will be evaluated later in this paper.

There are, incidentally, gender differences that will be discussed later in this paper. A woman who was sexually abused as a girl, and whose neuroception is therefore skewed toward hypervigilance, might react to the friendly intimacy of an embrace by her husband with fear, perhaps becoming immobile and dissociated, or anxious and feel like fleeing. She knows cognitively that she is safe, but her nervous system is unconsciously reacting and she has no ability to override that “hardwired” response. Both the woman and her husband are now frustrated, and both may blame her for being cold, unresponsive, or frigid. What needs to happen for her to gradually be able to relax and accept intimacy is to have the experience of safety without pressure. In other words, a neuroception of safety is necessary before social engagement behaviors can occur.

We shall discover that the process for repairing faulty neuroception involves (1) ensuring that the context elicits an assurance of safety; (2) achieving the ability for immobilization without fear (required for true intimacy); (3) repairing the lost temporal perspective of past, present, and future by reactivating the hippocampus within the brain during re-

experiences of previous traumas; and (4) stimulating the social engagement system, which is neurophysiologically intact, by stimulating the neural circuits that regulate the muscles of the face and head, e.g., through eye contact and verbal attentiveness.

### *Fight/flight response*

When the instinctual fight/flight response is activated, stress hormones are pumped into the body by the *sympathetic* nervous system to put all available energy into survival. Norepinephrine immediately calls into action certain centers in the brain to respond, including the *amygdala* (the center for memory for emotionally laden information) and the *hippocampus* (the nerve center that provides a conscious structure and context for declarative memory, the form of memory that relates to facts and events in cooperation with the cerebral cortex).

Hanoch & Vitouch (2004, p. 440) explain this dual system very clearly:

information processing can occur via two different channels. The first, the emotional channel, is faster though less precise ('quick but dirty', as LeDoux terms it); the second, the reflective channel, is slower, though more precise. Evolution has, in a sense, given us two options: (i) act fast (based on as little information as possible), with the risk of higher rates of false positives; or (ii) act slowly (integrating a larger set of data), though with more precision and flexibility. There are advantages, of course, to each option, but in cases of danger, as Zajonc (1980) points out, "the decision to run must be made on the basis of minimal cognitive engagement" (p. 156). The 'decision' reached on which path to take is executed automatically without conscious involvement (Ekman, 1992; Panksepp, 1982). That is, if the information carries with it a particular signal such as danger, then an emotional arousal schema is activated that automatically produces a reaction; but if the information does not correspond to a pre-programmed criterion, a more elaborate deliberation can occur. In a way, the emotional arousal systems are designed to react to stimuli (e.g. predators) in a schematic fashion with "the most-likely-to-succeed behavior" (LeDoux, 1996, p. 175).

The emotional arousal can be positive or negative, of course. Richard Davidson, director of the Laboratory for Affective Neuroscience at the University of Wisconsin, has used functional M.R.I. (fMRI) and advanced EEG analysis to study the difference in brain activity when people are happy or distressed (Goleman, 2003). The fMRI images reveal that when people are emotionally distressed (anxious, angry, depressed), the most active sites in the brain are circuitry converging on the amygdala, part of the brain's emotional center, and the right prefrontal cortex, a brain region important for the hypervigilance typical of people under stress. By contrast, when people are in positive moods (upbeat, enthusiastic and

energized) those sites are quiet, with the heightened activity in the left prefrontal cortex.

Activation of the amygdala is a manifestation of a neurobiological fear reaction, and enhanced amygdala activation is found in individuals with Borderline Personality Disorder (Herpertz et al., 2001), posttraumatic stress disorder (Rauch et al., 1996), and obsessive-compulsive disorder (Breiter et al., 1996a; Breiter et al., 1996b) during fMRI scanning of their provoked symptoms. Further, the amygdala is activated by perseverative thoughts and memories (Aggleton, 1992).

These two systems represent separable memory systems as well as separable processing systems: the amygdala as a “hot” memory system, and the hippocampus as a “cool” memory system. Psychopathology develops when there are dissociations between these memory systems. Elevated cortisol levels for chronic periods are associated with increased activity in anxiety-related brain regions, especially the amygdala (Drevets et al., 2002; Erickson, Drevets, & Schulkin, 2003; Makino, Gold, & Schulkin, 1994; Schulkin, Gold, & McEwen, 1998). Chronically elevated cortisol levels are also damaging to hippocampal neurons (McEwen, Gould, & Sakai, 1992).

Under traumatic stress, emotional memories are encoded without a significant contribution of context from the hippocampal system. “This produces a pool of stimulus-bound emotional memories that have been encoded without a coherent event-specific spatiotemporal frame to organize them. This pool is, essentially, a population of traumatic memory fragments. Upon retrieval, traumatic memories cannot be experienced as a memorial event with a beginning, end, and internal spatiotemporal structure. Instead, each emotional memory is experienced as fragmented, disorganized, and intrusive (Thomas et al., 1995)” (Jacobs et al, 1996).

Adrenaline surges, and glucose is mobilized from the liver to provide energy to activate the heart and lungs, the arms and legs, to optimize impending fight or flight. Endorphins are released in the brain to create stress-induced analgesia. If the animal survives the threat on its life, it discharges the stress hormones in its body and then resumes its normal daily activities. The discharge is primordial, reflexive, and totally somatic, and ranges from twitching movements to violent shaking. And a record of the successful survival experience is etched in the hippocampus.

If the animal is *not* functioning optimally, then the stress hormones in its body do not discharge even though it resumes its daily activities. This is often the case with humans, and one common example of it is chronic

vigilance (or hypervigilance). Sterling (2004) proposes an explanation of this phenomena: hypertension emerges as the accumulated response to multiple experiences of a need for vigilance. Sterling explains in detail (pp. 18-19):

Vigilance starts when a child is delivered from its mother's protection to the care of strangers. Thirty years ago this occurred when US children first entered school at around age 6. Correspondingly, blood pressures were constant from the first year of life until age six (median systolic level ~100 mm Hg). Then commenced a steady rise, so that by age 17 half of all boys showed systolic pressures above 130 mm Hg, and about 20 percent showed pressures above 140 mm Hg (hypertensive). The rise for girls was similar, though slightly milder (Figure 8). But now blood pressures begin to rise in the first year of life (NIH Report, 1997). This startling change might be associated with the rise of "day care" and the shift of mothers away from their infants and into the workforce. Consistent with this hypothesis, rat pups detached from their mothers show an eight-fold rise of corticosterone over 24 hours, and human toddlers detached from their parents show increased cortisol (Schulkin, 1999).

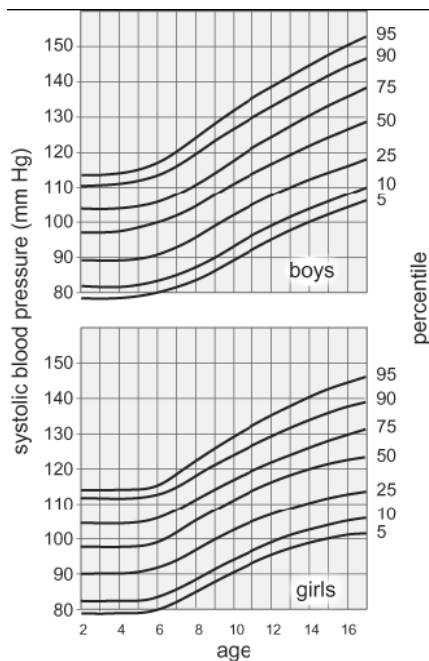


Figure 2.

**Systolic blood pressures were steady until school age and then rise continuously. [Figure 8, Sterling, 2004, p. 19]**

Diastolic pressures also rise. Recent data show pressures rising in the first year, perhaps associated with the increase of "day care" (NIH report, 1997). Redrawn from Blumenthal et al., 1977.

### *Freeze response*

If the fight or flight is not successful, then at the point of recognizing defeat and impending death, the animal goes into a state of helplessness and hopelessness, physiologically the freeze response. It appears as “feigning death” because of the sudden and extreme immobility. The stress hormones are not discharged but are rather counteracted by a new cocktail of hormones activated by the *parasympathetic* nervous system. The pulse and blood pressure, elevated by stress hormones, are now forced to drop precipitously. The endorphins released in response to threat now persist during freeze/immobility, rendering the animal analgesic in the face of the potential injury from the attack. The animal is in a state of passive dissociation, not unconsciousness but an altered state of “suspended animation.”

If the animal survives, it enters a discharge sequence similar to the discharge of the stress hormones. The experience of escape is assigned high significance by the hippocampus and is stored in procedural memory, increasing confidence and resilience for future threatening experiences, in fact creating greater resiliency than an animal never challenged (Figure 1).

If that discharge is blocked, the animal suffers chronic physical symptoms of immune deficiency and a shortened lifespan, and behaviorally suffers depression and neurosis. An example is animals in cages whose freedom of movement is severely restricted. Their resignation to “inescapable” trauma is called the defeat response, or learned helplessness, and represents a state of adaptation to living indefinitely with life-threatening trauma without further attempts to escape (fight or flight). Learned helplessness is the chronic persistence of the undischarged parasympathetic freeze response. We know this because blocking the parasympathetic nervous system (with the drug atropine) blocks the development of learned helplessness (Seligman, 1975). Here, unable to discharge and complete the process of recovery from the freeze response, the experience of inescapable threat is consolidated in somatic memory, reinforcing the tendency to freeze in the future.

Such prolonged exposure to elevated levels of stress hormones within the context of the undischarged freeze response creates damage to the brain’s hippocampus. That damage consists of a loss of neurons and synapses (a loss of up to 18%), and results in corruption of thought process and learning, particularly deficits of encoding short-term into long-term memory (Squire, 1992). There is contrasting evidence, however, that indicates that “smaller hippocampi in PTSD represent a pre-existing,



familial vulnerability factor rather than the neurotoxic product of trauma exposure per se” (Gilbertson et al, 2002). In either case, traumatic experience damages the contextual memory and processing function of the human brain.

Functioning properly, the hippocampus is a center for comparing input with stored data, and, as such, a center to filter out irrelevant (that is, distracting) stimuli that might lead to maladaptive arousal responses (Sprick, 1995). The hippocampal circuitry locks onto only one set of inputs at a time, thereby facilitating selective attention to only one set. However, when dysfunctional, the hippocampus, rather than filtering out irrelevant stimuli, becomes fixated on them. Abarbanel (1995) reviews studies of damage to the prefrontal cortex and hippocampus which reveal symptoms reminiscent of ADHD, i.e., hyperactivity, distractibility, and a tendency toward preoccupation with certain activities that verges on pathological “undistractibility-distractibility” reminiscent of ADHD children’s tendency to become mesmerized by television or video games.

Greatrex (2002, p. 189) notes that “when we feel an emotion consciously or unconsciously, our whole being is engaged in the experience. Furthermore, the more powerful the emotion, the more our subjective sense of time is altered.” The traumatic experiences etched in procedural memory without accompanying resolution experiences interfere with current working memory. Past threats are perceived to be present threats, suggested by intrusive thoughts, flashbacks, and hypervigilance. Not only is the ability to distinguish between past and present lost, but the **“repertoire of survival skills remains confined to those skills that were acquired up to the time of the trauma, and they lack the resilience to learn new strategies”** (Scaer, 2005, p. 67, emphasis added). An aspect of this individual is frozen in the past – a “primitively organized alternative self” (Davies & Frawley, 1992, p. 16).

Vickers (2005, p. 220) puts this time confusion regarding past and present into perspective:

Ehlers and Clark (2000) point out that PTSD is, at first sight, a paradoxical disorder. It is a disorder characterized by extreme anxiety and classified as an anxiety disorder, yet these disorders by definition involve a fear of a threat in the *future* (Beck, 1976); for PTSD sufferers, the terrifying event is in the past. These authors propose three reasons for this paradox. First, in the mind of the PTSD sufferer, the past event has been processed (or rather, has failed to be normally processed) so that the sense of threat remains active, as if it were now. Second, to the sufferer, the event is wrongly attributed to have a lasting significance so that its *meaning remains current*. Third, the patient engages in strategies intended to control the threat, such as various types of avoidance or ‘safety behaviours’. As

in other anxiety disorders, these have the opposite to the desired effect of maintaining the fear.

A very crucial element of effective treatment follows from these facts, i.e., that we can override the primitive “hot” response to threat (amygdala) and intervene with the “cool” response (hippocampus), but the latter is only capable of being different from the former when the environment is perceived as *safe and non-threatening*. Only then is the urgency of sympathetic response with primary emotion and reflex action dampened, allowing a “cooler head to prevail.” We will see later in detail how crucial is the creation of a *safe and non-threatening* environment, and many of the actual techniques that are so useful in doing so in the therapeutic milieu.

The sympathetic response is most easily understood as an upsurge – those feelings which are experienced as coming UP – anger, fear, excitement, desire, hatred – and which if expressed involve movement out, or towards, or in the case of fear, away from, an object. Conversely the parasympathetic action is a concomitant of coming DOWN – disappointment, grief, shame, guilt, despair; and contentment, peacefulness, satisfaction - feelings which involve a decrease in tension, withdrawal of energy inward and tend more towards introspection. Laughter and tears are both usually a sign of parasympathetic activity (Carroll, 2001).

These two propensities, the fight/flight sympathetic response and the freeze parasympathetic response, establish the basis for panic on the one hand and depression on the other, respectively. Stanley Keleman (1989, n.d.) has worked on this formulation for many years, expressing that the first response to any stress or challenge is always arousal. When that arousal is not containable, if arousal is of such intensity that it becomes unboundaried, he/she will become overwhelmed. There’s too much excitation and not enough form, structure, context, or containment. At the extreme this is panic. To protect oneself from being too overwhelmed or panicked, a person sometimes responds by holding it in, waiting, stiffening, squeezing, and constricting: the freeze response, which is headed in the direction of depression.

“The life process takes place in a constant alternation of expansion and contraction” (Reich, 1967, p. 257). When there’s too much excitation without form or containment, there is panic. When there’s too much constriction and containment without a flow of activation, there is numbing, in which nothing can be aroused; there is depression.

And these extremes can actually blend into each other. Buhl (n.d.) explains:

There is another remarkable particularity about the ANS [autonomic nervous system]: not only can it fluctuate rhythmically between sympathetic and parasympathetic disposition, but the respective extremes can also blend into one another. An extreme and chronic sympathicotonus can turn into an extreme and chronic parasympathicotonus and vice versa. This is the reason why in situations of extreme sympathetic activity, like agitation before an exam, stress-induced diarrhea can occur (which actually indicates strong parasympathetic stimulation). Accordingly, one could say that permanent readiness for fight can turn into resignation, but also regression can turn into readiness for fight under increasing stress. This mechanism explains why an illness can be induced not only by persistence of one chronic state, but also by an irregular, uncoordinated “furious back and forth” between sympathicotonic and parasympathicotonic conditions.

Such a pattern of cycling between sympathetic and parasympathetic activation, or “furious back and forth”, is commonly seen in children with disorganized attachment and many other survivors of chaotic and distressed childhood.

The autonomic nervous system of the trauma victim, however, is characterized by a state of instability, sensitivity, and a cyclical abnormality of its normal state of regulation. Homeostasis may be disrupted by excessive and disproportionate stimulation by one limb of the autonomic nervous system, a state that is characteristic of the trauma victim. Assaulted by internal cues of threat, the victim experiences repetitive episodes of **sympathetic arousal that in turn reflexively trigger deep parasympathetic dissociation**. The trauma victim lives in a state of involuntary and disruptive autonomic instability and cycling (Scaer, 2005, p. 195, emphasis added).

The psychopathology labeled *catatonia* is an obvious extreme example of the unresolved freeze response (Moskowitz, 2004). The *catatonic stupor* symptoms of immobility, rigid and waxy mask-like face, fixed and unfocused gaze or stare, and lack of reaction to stimuli nevertheless accompany evidence of alertness: despite their apparent unresponsiveness, they often have a surprising level of mental functioning with awareness of events going on around them (Rosebush & Mazurek, 1999). No longer considered solely as a subtype of schizophrenia, these symptoms present in about 10% of psychiatric inpatients (Taylor & Fink, 2003), most often following situations in which the individual felt him/herself to be under profound threat from internal or external perceived sources, experienced as unbearable anxiety.

Depression is much more common than catatonia, of course, and Dixon (1998) linked the immobility response (which he termed *arrested*

*flight*) with such depression symptoms as social withdrawal, reduced eye contact, and psychomotor retardation. Thus depression is conceptualized as a last resort defense strategy triggered by imminent inescapable threat. Indeed, Gilbert and Allan (1998) consider such a blocked motivation for flight, the arrested flight, to result specifically from feelings of *entrapment* and *defeat*. Moskowitz (2004) suggests that this pattern of anxious depression follows a specific cluster of experiences: “a perception of inescapable but at the same time amorphous danger, a sense of defeat and entrapment, and a sensation of imminent doom” (p. 995). These are circumstances that Panksepp (1998) indicates generate *rage*. When release (discharge) of the rage is blocked or suppressed, they become the key elements in the complex PTSD experience which we are calling *shock*.

An alternate form of catatonia (*hyperkinetic* catatonia) appears with *catatonic excitement* symptoms of apparently purposeless agitation not influenced by external stimuli (American Psychiatric Association, 1994, p. 765). This is an extreme example of the unresolved sympathetic fight-flight response which typically precedes or follows the freeze response (Moskowitz, 2004). It is especially relevant that the “purposeless” agitation and “undirected” combativeness associated with catatonic excitement results from an inability to localize the source of danger, i.e., there is no clearly recognizable threat to attack or from which to flee. The individual is reacting to a perceived threat which does not exist in the present; it is a memory from the traumatic past. This inability to differentiate past from present leaves the person feeling threatened from all directions, and from nowhere.

When the catatonic state becomes lethal it is called *malignant catatonia*. In such cases there is generally an extended period of frenetic activity (sympathetic response) followed by stupor (parasympathetic response) and then death. In most cases (80%) there is no medical cause of the death (Mann et al., 2003). Gurrera (1999) argued that a hyperactive and unregulated sympathetic response, released from inhibition by the frontal cortex (via the hypothalamus), accounts for the death process, and suggests that “acute psychic distress” may be responsible for that disinhibition. The person has literally been “scared to death.”

We know, too, that people can die of a broken heart. Informally called “broken-heart syndrome,” the formal name for this phenomenon is human stress cardiomyopathy or myocardial stunning (Pavin et al., 1997; Wittstein et al., 2005). While the individual appears to be having a heart attack, there is no blocked artery or other physical reason for it that can be

treated by the emergency room physician. The only recognized treatment is support and the passage of time. It occurs due to exaggerated sympathetic stimulation, or undischarged catecholamine excess, related to intense emotional stress, usually the sudden loss of a loved one.

We can see that death is a very possible outcome of interrupted stress discharge throughout the life cycle, from the unexplained death of “unwanted” infants (to be discussed later) to the “broken-heart syndrome” or being “scared to death.”

### *The nervous system and the chakras*

The Polyvagal Theory (Porges, 2003) is a recent scientific explanation of how the brain and the autonomic nervous system switch the body on and off in order to regulate physiological response, including behaviors and emotions. This theory is based on the interaction of the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). The PNS is further divided into two components, representing different stages in human evolution: respectively, the recent *Ventral Vagal Complex* (VVC) and the ancient *Dorsal Vagal Complex* (DVC). The VVC is activated by perceptions of safety and trust and initiates the Social Engagement System which enables social interaction, fostering pro-social behaviors, communication and engagement with others. The VVC allows rapid and precise activation and de-activation of metabolic responses in order to carefully match external requirements. The SNS, activated by perceptions of danger and threat, promotes defense and emergency responses, mobilizing resources and promoting responses such as fight and flight, namely aggression or active avoidance. This system is powerful but difficult to switch off once activated. The DVC, the most ancient structure, is activated by the perception of a severe danger or life threat and promotes the preservation of resources through passive avoidance and immobilization. From a behavioral perspective, excessive activation of the DVC leads to isolation, demotivation, immobilization, and potentially death.

The activation or deactivation of somatic energies is intricately related to the chakra system. The newer (ventral) branch of the vagus is involved in more conscious, voluntary, flexible and often social activities, and the older (dorsal) branch of the vagus deals with unconscious reflexive functions. The old reptilian nervous system is built around the dorsal (sub-diaphragmatic and unmyelinated) branch of the vagus nerve, which fosters digestion and responds to threat by depressing metabolic activity (the

freeze response). This system is associated with passive reflexive regulation of visceral functions: sweating, lungs, diaphragm, stomach and intestines. These organs and the dorsal vagus nerve are interconnected with the first three chakras, i.e., survival (thriving), procreation (passion for life), and power (internal locus of control).

The newer branch of the vagus nervous system, unique to mammals, is the ventral (myelinated) vagus complex, which regulates the functions of the larynx, pharynx, and esophagus (speech and eating). The ventral vagus system is associated with cranial nerves that regulate sociability via facial expression and vocalization, and thereby is related to processes associated with attention, motion, emotion and communication, referred to as the Social Engagement System. This system is interconnected with the heart and throat chakras.

Both vagal circuits share in regulating the heart: the dorsal (reptilian) nerve controls heart rate, dilation of blood vessels and blood pressure, while the ventral (mammalian) vagus regulates the heart and the bronchi to promote calm and self-soothing states, producing a cardiorespiratory rhythm. And since the two vagal systems are programmed with different response strategies, they can sometimes respond in a contradictory manner which causes physical and psychological distress. When the two vagal circuits are out of balance, excessive sympathetic or excessive parasympathetic activity leaves the body stressed and out of sync.

### *Heart rate variability (HRV)*

When one is stressed, the entire body is out of sync. Typical negative emotions that accompany stress, like anger, frustration, anxiety and worry, lead to increased disorder in the heart's rhythms and in the nervous system. In contrast, positive emotions like joy, appreciation, care and kindness create harmony in the heart's rhythms and the nervous system. Other bodily systems, i.e., neural, hormonal and biochemical, sync to this heart rhythm. It is the master switch setting the pace for the implementation of the autonomic nervous system, and is in turn regulated by both branches of that system. As such, one's breath provides a bridge between conscious and voluntary regulation and unconscious, involuntary regulation.

Heart rate variability (HRV) is the natural rise and fall of the heart rate in response to one's breathing, blood pressure, hormones, stress and emotions. It refers to the degree of fluctuation in the length of the intervals between heart beats, and reflects the degree of balance between sympathetic and parasympathetic activity. When one sleeps or feels

appreciation or happiness, it falls; that is to say the body/brain experiences order and coherence. When one experiences hostility or frustration, it increases; the body/brain's experience is disordered and erratic. A higher HRV reflects an ability to adapt to changes in the environment; it shows stress resiliency. A lower HRV implies decreased ability to respond to changes in the environment; it shows stress vulnerability.

The significance of HRV was first discovered when unborn infants were attached to cardiac sensors in utero, and it was found that very even intervals between fetal heart beats were a precursor to death (Hon & Lee, 1963). It was further observed in this group that the beat-to-beat intervals would become progressively more even until death was reached. The same pattern of less variability in approaching death has also been observed in adults (Nakagawa, Saikawa & Ito, 1994).

Studies have now shown a link between stressful emotional conditions and reduced HRV: panic or anxiety disorders (Friedman & Thayer, 1998a; Friedman & Thayer, 1998b; Kawachi et al., 1995; Middleton, 1990; Offerhaus, 1980; Watkins, Grossman, Krishnan & Blumenthal, 1999; Yeragani et al., 1990; 1993); hostility (McCraty, Atkinson, Tiller, Rein & Watkins, 1995; Sloan et al., 1994); depression (Carney, et al., 1995; Krittayaphong, et al., 1997); PTSD (Cohen, et al., 1998). Feeling appreciation improves HRV (McCraty, Atkinson, Tiller, Rein & Watkins, 1995). Refer to Figure 3.

People who are socially unsupported and are socially isolated have lower HRV than those with more social connections (Horsten, et al., 1999).

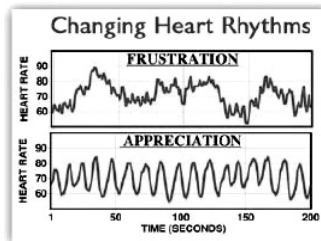


Figure 3.

HeartMath is a registered trademark of Institute of HeartMath  
[http://www.heartmath.com/health/professional/stress\\_e-book.pdf](http://www.heartmath.com/health/professional/stress_e-book.pdf)

One of the best ways known to positively affect the HRV is through breathing consciously. Conscious breathing can be defined as *intentional* breathing, breathing purposefully at a particular rate, depth or pattern. Most conscious breathing involves increasing the depth of the breath,

slowing or speeding up the breathing from the average 12 to 18 breaths per minute, and either holding the breath between inhale and exhale or connecting the inhale and exhale as one fluid motion. The following two charts (from the website of The Institute for Applied Meditation) show the HRV of a subject in distress (Figure 3), and secondly the HRV of someone meditating with a quiet rhythmic breathing pattern (Figure 4).



Figure 4.

Notice four peaks where the heart rate shoots up to 95 or more than 100 beats/minute. These indicate short moments of emotional distress, while the subject thinks of his finances. The subject has had no physical movement during this time. Note also that there is no pattern to the heart rate. There is no heart rhythm.



Figure 5.

Notice the regularity of this heart rate, and that the average heart rate is decreased. This pattern shows a person meditating, with a rhythmic breath.

*Prenatal trauma*

The effects of traumatic cortisol and endorphins lingering in the system are even more damaging when occurring in the womb. Research documents that newborns of depressed mothers “show a biochemical/physiological profile that mimics their mothers’ prenatal biochemical/



physiological profile including elevated cortisol, lower levels of dopamine and serotonin, greater relative right frontal EEG activation and lower vagal tone. Elevated prenatal maternal cortisol is the strongest predictor of these neonatal outcomes [delayed prenatal growth, prematurity and low birthweight]" (Field et al., 2006, p. 445). The mother's depression itself is not the most telling factor contributing to her infant's vulnerability, however. One study compared *withdrawn* depressed mothers and *intrusive* depressed mothers with nondepressed mothers on their prenatal cortisol and catecholamine levels and on fetal activity and neonatal outcome variables. The infants of withdrawn mothers had the highest cortisol levels and the lowest dopamine and serotonin levels as well as the most asymmetrical EEG patterns (Field et al., 2001). This would seem to substantiate the common belief that neglect may be even more detrimental than abuse.

The maternal in utero imprint of stress response, even more so than after birth, can become a lifelong template. A dramatic example of this difference is the fact that, in comparing the incidence of psychiatric disorders later in life, children born to mothers who were widowed during pregnancy are significantly higher than for infants whose father died in their first year of life (Huttunen et al., 1994). In response to invasive threat, the prenatate's levels of plasma cortisol and endorphins are dramatically elevated, not through massive direct transport of cortisol across the placenta but rather due to the way that the mother's stress hormones trigger the fetus to manufacture its own (Huizink et al., 2004). These stress hormones persist longer in the fetus' system than in a child or adult (Giannakouloupoulos et al., 1994). The memories, and the learned helplessness, are embedded deeper, at a pre-verbal, cellular level. After all, the repertoire of survival skills remains confined to those skills that were acquired up to the time of the trauma. In fact, psychological and motor development can be arrested in child victims of trauma, leading to increasingly negative impact on their lives if they continue to mature without intervention to restore lost or undeveloped resources and skills (Pynoos, 1993).

The effects of such trauma can, in fact, be fatal. Dissociation is utilized at the earliest stages of development to defend against intolerably painful experiences (Spitz, 1961; Fraiberg, 1982). Fraiberg (1982) observed freezing in seriously disturbed infants as young as 5 months, in which the infant remains immobile and glassy-eyed for as long as 20 minutes. The passive defense of parasympathetic dissociation in prenates and newborns

may even, at the extreme, account for death. Infants born of unwanted pregnancies are more than twice as likely to die within a month of birth than wanted infants (Bustan & Coker, 1994).

There is growing evidence that stress affects the fetus differently depending on when during the pregnancy it occurs. DiPietro et al. (2006) have published research challenging the assumption that stress in mothers during pregnancy (specifically during the period from 24 to 32 weeks gestation) is always harmful. Their work indicates that babies born of mothers who reported elevated stress during this part of their pregnancy showed no restricted growth, birth defects, temperament differences, or behavior differences. In fact, during this period elevated levels of cortisol “could be enhancing the development of organs before birth.” Other research (Van den Bergh & Marcoen, 2004) indicates that maternal stress between 12 and 22 weeks gestation may indeed be related to a variety of negative outcomes for the baby, including greater likelihood of developing ADHD, whereas anxiety at 32 to 40 weeks is not.

High risk preterm neonates have problems coordinating breathing, sucking, and swallowing, and have irregularly regulated heart rate, apnea, and a drop in available oxygen (i.e., processes [not] regulated by the mammalian vagus). This response, to deal with decreased resources, is adaptive for reptiles, but potentially lethal for the human. This also is observed during fetal distress, when there is severe hypoxia, and later in life a similar mechanism may produce bronchial asthma (Porges, 1995).

By three months, several areas of the limbic system, including the hippocampus, show activity on PET scans. The period from 10 to 13.5 months appears to be a critical time window for the rapid growth of the energy mobilizing the *sympathetic* part of the ANS. By 18 months, Malatesta et al. (1989) feel that the infant is able to have one set of *inner feelings privately* held and display another set of feelings publicly. This aged infant learns to anticipate reward, punishment, approval and disapproval, has an initial capacity for ambivalence (love and hate) and object constancy, and has an initial image of him/herself and others (Greatrex, 2002). Also at eighteen months the infant begins to mentally integrate time as past, present and future (Wheeler et al., 1997). Into the second year, as voluntary activity increases in motoric, emotional, and cognitive spheres, the inhibiting, energy conserving *parasympathetic* system begins to mature, and along with it, the dynamic unconscious, and the hypometabolic emotion, shame.

### *Benefits of surviving trauma*

It is not difficult to understand, however counterintuitive it may be, that some stress and anxiety is *helpful* to the growth and development process. Remember that survival of stress, which includes successful discharge of the excess stress hormones, results in increased confidence and resilience for future stressful experiences (Figure 1).

It is exciting that the discharge of freeze response can be facilitated therapeutically, and the damage to the brain and memory can be repaired. The hippocampus is part of the limbic system and is especially susceptible to hypoxic and ischaemic damage (obstruction of the blood supply). Fortunately, however, the hippocampus is the only known region of the human brain which can replicate new neurons (Ehling et al., 2003; Vermetten et al., 2003). Selective serotonin reuptake inhibitors (e.g., Prozac) may actually promote the regeneration of neurons in the hippocampus (Harvard Mental Health Letter, 2005).

Long-term potentiation (LTP) is defined as an increase in strength of synaptic transmission with repetitive use that lasts for more than a few minutes. In the hippocampus, LTP can be triggered by less than 1 second of intense synaptic activity and lasts for hours or more. This may account for the memory re-acquisition and re-contextualization necessary for healing trauma. Through techniques such as catharsis, titration of threat cues, unwinding of bound undischarged energy, and reprogramming the conditioned perpetuation of the freeze response, effective facilitation can indeed reverse the damage done and return the individual to a state of resilience and homeostasis.

The theta brain wave frequency (4-7 Hz) is a basic ingredient of hippocampal action, dominating the EEG activity recorded from the hippocampus (Steriade et al., 1990). Theta is known to be a predominant pattern when an individual is accessing subconscious material; it is also dominant during REM dream activity during the sleep cycle. "As detailed by Winson (1972, 1990), this process [LTP, or long-term potentiation] is mediated by the theta rhythm. Miller (1991) has calculated that this process involves transit times from hippocampus to neocortex to hippocampus on the order of 200 ms (corresponding to 5 Hz), thereby supporting a resonance at the theta frequency between the two sets of networks" (Abarbanel, 1995).

Dysfunction can be represented as being stuck in too much or too little. The depressed person seems to be stuck in a low arousal pattern. The brain is not producing the activity common in a "normally" functioning person.

The anxious person's brain may be producing too much high frequency brainwave activity and that person may need to learn to make the transition to a more relaxed, lower arousal state from time to time. The child or adult with an attention problem may have trouble making the shift to an alert, focused brainwave state. The person with a substance abuse problem may produce too much high arousal brainwaves and not enough of the brainwaves that make people feel good. Using a substance or a behavior to reverse this is only a temporary fix, while increasing the individual's self-regulation provides a natural and permanent solution.

**Excessive sympathetic branch activity** can lead to increased energy-consuming processes, manifested as increases in heart rate and respiration and as a pounding sensation in the head (Siegel, 1999). Over the long term, such hyperarousal may disrupt cognitive and affective processing as the individual becomes overwhelmed and disorganized by the accelerated pace and amplitude of thoughts and emotions, which may be accompanied by intrusive memories.

**Excessive parasympathetic branch activity** leads to increased energy-conserving processes, manifested as decreases in heart rate and respiration and as a sense of 'numbness' and 'shutting down' within the mind (Siegel, 1999, p.254). Such hypoarousal can manifest as numbing, a dulling of inner body sensation, slowing of muscular/skeletal response and diminished muscular tone, especially in the face. Here cognitive and emotional processing are also disrupted.

Wilbarger and Wilbarger (1997) suggest that when arousal remains within the "optimal arousal zone," a person can contain and experience (i.e., not dissociate from) the affects, sensations, sense perceptions and thoughts that occur, and can process information effectively.

Periods of sympathetic arousal, with high levels of stress hormones, will include symptoms of muscle bracing, bruxism, ocular divergence, tachycardia, diaphoresis, pallor, tremor, startle, hypervigilance, panic, rage and constipation. These states will alternate with parasympathetic dominance, including symptoms of palpitations, nausea, dizziness, indigestion, abdominal cramps, diarrhea and incontinence. The state of low serum cortisol (Mason et al., 1986; Yehuda et al., 1990) is associated with behavioral responses including social isolation and withdrawal, substance abuse, constricted affect, denial, cognitive impairment and dissociation, all relatively parasympathetic states.

People who suffer from PTSD are plagued with frightening body symptoms which are characteristic of hyperarousal: accelerated heart beat,

cold sweating, rapid breathing, heart palpitations, hypervigilance, and hyper startle response (jumpiness). These symptoms lead to sleep disturbances, loss of appetite, sexual dysfunction and difficulties in concentrating, which are further hallmarks of PTSD.

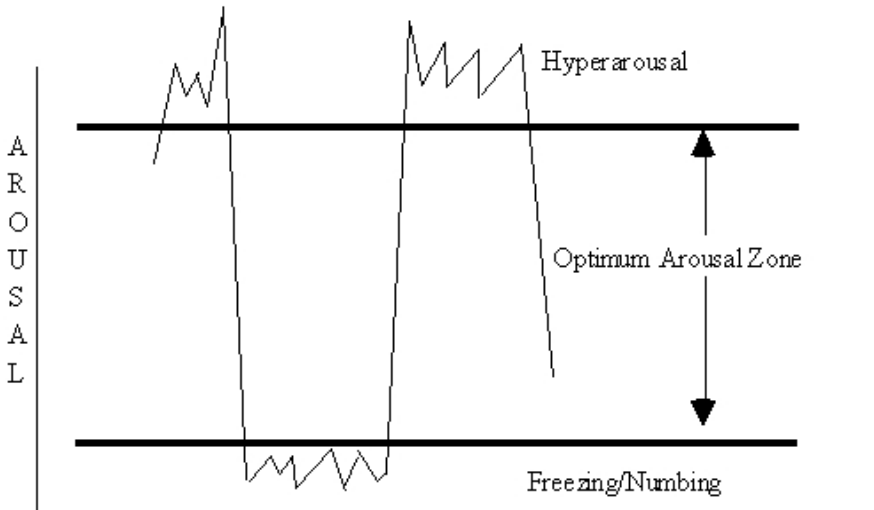


Figure 6.  
The Modulation Model: The Bi-Phasic Response to Trauma  
(Figure 2 from Ogden & Minton, 2000)

### Hyperarousal (fight or flight)

1. rapid breathing and heart rate, heart palpitations
2. cold and pale skin, pallor
3. dilated pupils
4. raised blood pressure
5. endorphins are released to reduce pain
6. hypervigilance, panic, rage, constipation, tremor, muscle spasms, hyperventilation, cold sweats, profound sleep disturbances, loss of appetite, sexual dysfunction
7. accelerated pace and amplitude of thoughts and emotions, intrusive memories, difficulties in concentrating
8. active dissociation: persisting physiological hyperarousal and hyperactivity, busyness, increased muscle tone, increased body temperature, hyper startle response (jumpiness), affect regulation problems and generalized (or specific) anxiety

**Hypoarousal (freeze)**

1. decreased heart rate and respiration
2. warm and flushed skin
3. lowered blood pressure
4. Endorphins are released in the brain to create stress-induced analgesia
5. helplessness and hopelessness
6. sudden and extreme immobility (appears as “feigning death” or “suspended animation”)
7. palpitations, nausea, dizziness, bowel cramps, indigestion, diarrhea, incontinence, generalized weakness
8. numbness, ‘shutting down’ within the mind, dulling of inner body sensation, slowing of muscular/skeletal response, especially in the face
9. social isolation and withdrawal, substance abuse, constricted affect, denial
10. passive dissociation: drifting consciousness, emotional constriction, social isolation, loss of contact with reality, lack of motivation, body numbness, a sense of “leaving” the body, withdrawal, somatic complaints, anxiety, dependence

When abuse is prolonged, severe, involves a family member, and is associated with neglect and lack of social support, the effects are more damaging (Binder et al., 1996; Coffey et al., 1996; Tsai et al., 1979; van der Kolk, 1996; Wyatt & Newcomb, 1990), and likely to lead to an overwhelming level of response, variously called shock, complex PTSD, or Disorders of Extreme Stress, Not Otherwise Specified (DESNOS).

*Structural dissociation*

Many traumatized individuals alternate between re-experiencing their trauma and being detached from, or even relatively unaware of the trauma and its effects. Viewing these two approaches as polar ends of a continuum, e.g., the sympathetic/parasympathetic response, is simplistic. Each of these alternative ways of being can better be conceptualized as a cluster of mental/emotional states, proposed as a Theory of Structural Dissociation by Nijenhuis and associates (2004). Severe threat may provoke a structural dissociation of the premorbid personality (Van der Hart, 2000), creating a split between the defensive system on one hand (re-experiencing trauma), and the systems that involve managing daily life and

survival of the species on the other hand (detachment from trauma). The traumatized individual, then, develops an “emotional” part of the personality (EP) and an “apparently normal” part of the personality (ANP) that engages in matters of daily life (and that has failed to integrate the traumatic experience) (Myers, 1940).

The emotional personality carries memories somatically, and often pre-verbally. “Van der Kolk and Fisler (1995) found that traumatic memories of subjects with PTSD were retrieved, at least initially, in the form of dissociated mental imprints of sensory and affective elements of the traumatic experience with little or no linguistic component. Sexually abused children also ‘remembered’ their traumas in the form of sensory perceptions, and behavioral responses (Burgess, Hartman, & Baker, 1995)” (Nijenhuis et al, 2004).

The apparently normal personality is condemned to live life “on the surface of consciousness” (Appelfeld, 1994, p. 18), dissociated from his body and his emotions.

These dissociative parts of the individual’s personality each have a different sense of self, and respond to trauma memories differently (Nijenhuis et al., 1999; Reinders et al., 2003). This dissociation compromises the development of a coherent sense of personal existence in a framework of the past, the present, and the future. Harvey and Bryant (1999) found that dissociative symptoms of patients with acute stress disorder did not predict PTSD well, whereas re-experiencing trauma was highly predictive. “The emotional personality can also be further structurally dissociated within itself, as evident in complex PTSD” (Nijenhuis et al, 2004). The divided EP encompasses several survival strategies that are often utilized sequentially (flight, freeze, fight, submission), e.g., freezing with analgesia, physically resisting threat with anger, then submitting to threat while being severely anesthetized. Dividedness may also occur within a single moment of time during trauma, i.e., between an observing EP and an experiencing EP.

Traumatized individuals often experience the memories of traumas as personal to their emotional personality but the apparently normal personality may represent them as fragmented aspects of the trauma (Van der Hart & Op den Velde, 1995) or have no access to them at all. Indeed, “when the emotional personality is activated, the patient in that state tends to lose access to a range of memories that are readily available for the apparently normal personality. The lost memories typically involve episodic memories (personified memories of personal experiences), but

may also include semantic memories (factual knowledge) and even procedural memories (e.g., memories for skills and specific kinds of associations among various stimuli as a result of classical conditioning)” (Van der Hart & Nijenhuis, 2001a). The emotional personality, of course, presents with the identity of a child. The emotional part of the personality cannot function in the world like the apparently normal personality, because its brain does not have access to the memories necessary to function in that way.

Perry and his colleagues (Perry, 1999; Perry et al., 1995b) have argued that repeated activation of specific trauma-related states, or emotional personalities, leads to neurobiological “hard-wiring” of the brain, with the first six years of life as a critical period. This hard-wiring promotes state-dependent functioning or functioning that is dependent on dissociative parts of the personality. Markowitsch and his colleagues have demonstrated that trauma-related dissociative amnesia can be associated with reduced blood flow in parts of the brain that are normally activated during retrieval of autobiographical memories (Markowitsch, 1999; Markowitsch et al. 1997a; Markowitsch et al., 1997b; Markowitsch et al., 1998, 2000). Moreover, partial regaining of these memories was correlated with a return to normal brain metabolism in these areas (Markowitsch et al., 2000). There is a switch between these two parts of the personality, each with its own way of operating in the brain; that switch is, however, normally *not* within intentional control of either part.

The theory of structural dissociation correlates with what we know about the autonomic nervous system, suggesting that PTSD individuals who do not respond physiologically to trauma-cues manage to remain in the trauma-avoidant apparently normal personality, and that it could be utilizing a parasympathetic nervous system response, involving a vagal brake on activation of the sympathetic nervous system (Porges, 2003), to effect the suppression. The vagal brake provides a neural mechanism to rapidly change visceral state by slowing or speeding the heart rate. Functionally, by modulating visceral state, the vagal brake enables the individual to rapidly engage or disengage attention, to promote self-soothing and calm behavioral states, and to mobilize communication behaviors. He/she is unconsciously, automatically suppressing (dissociating) a fight/ flight response and substituting a calm/ freeze response. Such an individual could also be a manifestation of an emotional personality that engages in total submission, a response pattern that may be



controlled by the dorsal vagal branch of the parasympathetic nervous system (Porges, 2003).

According to the theory, PTSD individuals who *do* respond physiologically to trauma-cues, fail as an apparently normal personality to avoid cued intrusion by the emotional personality, or else they switch into the emotional personality that engages in fight and flight, i.e., reactions controlled by the sympathetic nervous system.

The theory of structural dissociation allows an explanation of dissociation that stretches from “normal” dissociative experience all the way to Dissociative Identity Disorder. “In this context we have proposed three levels of structural dissociation that mark a range of trauma-related disorders: simple PTSD; complex PTSD, DES and DDNOS; and DID. According to the theory, these various disorders can be situated on a continuum of complexity of structural dissociation of the personality” (Nijenhuis et al., 2004).

Treatment calls for introducing the dissociative parts of the personality, both EP and ANS, and their mental contents including memories, to each other in carefully planned steps that promote integration and preclude re-dissociation. Such treatment should be phase-oriented, initially raising the level of functioning of ANPs by gradually overcoming phobia of mental contents, phobia of ANPs for each other, and phobia of EPs. Also, phobia of attachment is addressed in that at least a working alliance and cooperation between ANPs and EPs among each other, and with the therapist are secured. If and when the integrative level has been sufficiently raised, phobia of traumatic memories can be addressed in the second treatment phase, by stepwise exposure and prevention of re-dissociation. In the third and final phase, integration of the personality, overcoming phobia of intimate attachment, and coping with life in non-dissociative and otherwise non-avoidant ways are the main goals (Nijenhuis et al., 2004).

“The affect ego is simply a channel for archetypal affects” (Kalsched, 2003). The emotional personality is a dissociated aspect of the person, yet it carries a much bigger burden than simply that of the wounded ego. The full force of the denied infant is brought to bear. “Nothing is more forceful than an infant’s distressed cry. Similarly, when an archetypal force is at work, the energetic attraction of the complex draws and imprisons all the energy away from the rest of the personality” (Sidoli & Blakemore, 2000, p. 90). The overwhelming and unbearable tension results in a split to allow it to be contained, finally, in the body.

### *Dissociation and embodiment*

No matter how dissociated a person may be from his/her body, that person is in fact *embodied*. We could accurately say that all the unconscious influences and all the autonomic responses in his/her life are embodied. "Human existence rests upon the fact of embodiment. Embodiment is, in fact, the terms of a person's existence. Somatic reality is how a person embodies himself, how he lives his embodiment" (Keleman, 1989, p. i). Indwelling is the non-dissociated experience of being in the body, being one with the body.

Donald Winnicott (1945, 1965) was a British pediatrician and psychoanalyst who specialized in the lifelong effects of a person's experience as an infant and in the process of children's development of a sense of self. He used the term "indwelling" for that process, and saw it as a core constituent of cohesive selfhood. In his view, infants only become integrated and personalized, or "called into existence," as they come to experience linkages between self and body and body functions. Indwelling provides a limiting membrane between "what is me versus what is not me," a psychic structure providing an internal sense of containment and safety.

When the infant's caregivers are not healthy themselves, they cannot give the child the intimate support he/she needs, and the child begins to disconnect from the unreliable (or reliably hurtful) source of disappointment and pain. This is the process of dissociation. When the infant begins to disconnect from its caregivers, it may become distracted by other things in the environment. Dissociation gets more and more severe depending on how noxious the caregiving is. Distraction progresses to unawareness, as in daydreaming-type states. As the child distances from others (parents), he/she inevitably disconnects from him/herself as well. In a teenager or adult we call this being a "space cadet." This disconnection from self widens to become resistance to living one's life ("I don't want to be here"). The infant has no words, of course, but definitely has the feelings and experience.

With worse treatment or neglect, the infant's resistance to living progresses to more destructive levels and it becomes necessary to make a choice about living or dying. An example is, of course, the high risk of infant death for unwanted infants already mentioned (Bustan & Coker, 1994). Another example is a study of a number of adolescent patients with a history of more than five suicide attempts each, always at the same time of year (Feldmar, 1979). He eventually determined that the suicide dates of

four patients corresponded to the month in which their mothers had tried to abort them. The adolescents had no *conscious* knowledge of the abortion attempts that they were *unconsciously* acting out. After discovering that their suicide attempts were seasonal intrusions of prenatal memory, the patients were free of the suicidal compulsion. They never attempted suicide again, even when their ‘anniversaries’ returned.

Consequences for those babies whose resistance to living progresses to more destructive levels *without actually dying* is to disavow or renounce parts of him/herself (“The part of me that has needs is bad. I want to get rid of it”). The abused or neglected child, like shipwreck survivors in an overcrowded lifeboat, must sacrifice some aspects of the self in order to preserve others. The more overwhelming the assault or neglect, the more essential and closer to the core is that aspect that must be sacrificed. Inner resources such as innocence, trust, spontaneity, courage, and self-esteem were lost, stolen, or abandoned in those early traumatic moments, leaving an immense empty space. What was split off and stashed away for safe-keeping may have been the child’s very essence, his/her “imperishable personal spirit” (Kalsched, 2003). The psychic energy cast off through dissociation, the sacrificed aspects of self, do not simply disappear into thin air, however, but rather are sent into “internal exile” where they continue in split off form as a primitively organized alternative self. Retrieving these inner resources in age regression to those traumatic events reunites the sacrificial alternative self with the immanent embodied person, bringing back to indwelling embodiment what had been stashed away for safe keeping.

This child is straddling the fence between physical death and stopping just short of it in a state of “psychic death.” Infants who have frightened and/or frightening parents may be observed to periodically enter a state of ‘stopping’, (noted by Fraiberg, 1982) in which the child becomes totally still, eyes glazed over, apparently frozen psychically and emotionally. This tendency can persist into childhood and adolescence, and the interesting thing that these individuals say about their ‘stopping’ is that their minds are not blank. To the contrary, they have so many thoughts demanding attention regarding how to find safety that they become overwhelmed, frozen from excess possibilities and an inability to act. This frozen state is as close as they can come to escaping into annihilation without actually dying. But at least they have stashed away the vital part of themselves in an internal “zone of safe retreat.”

These are some of the deepest wounds humans can suffer, and healing them requires “retrieval” efforts. What needs to be reconnected with, i.e., retrieved, may be attributes (such as innocence, self-worth, or spontaneity), or ego states (such as the playful child or the decisive choice-maker), or of the soul itself (that is, the person’s spiritual essence). We are retrieving “lost” aspects of the self from that place deep within where they were originally put for safekeeping, and bringing them back into direct access.

### *Dissociation and somatic dissociation*

Dissociation involves disengaging from the external world and attending to stimuli in the internal world. This can involve distraction, avoidance, numbing, daydreaming, fugue, fantasy, derealization, depersonalization and, in the extreme, fainting or catatonia.

The brain releases endorphins during trauma for analgesia, and the result is mental and somatic dissociation. Endorphins create relief from pain, and also pleasure, even euphoria. Sometimes the traumatized individual has been conditioned to repeat the trauma, or experiences that provoke similar physiological responses, as a means of generating endorphins in the brain. Endorphins create a trance state, dissociated from one’s actual circumstances in the environment. Thus, there is a secondary gain of endorphin release (and the attendant pain relief and dissociation) for re-creating trauma or other self-injurious experiences (Kosten & Krystal, 1988). Think of the thrill-seeker, the long-distance runner, or the sex addict who engages in dangerous behavior. In fact, “plane crash survivors have described getting a new found ‘rush’ from subsequently putting themselves in risky situations such as skydiving or extreme sports” (Farkas, 2004, p. 126).

The hippocampus, in addition to its role in memory formation, is related to the phenomenon of dissociation. For example, electrical stimulation of the hippocampus and adjacent cortex resulted in dissociation-like phenomena (Halgren et al., 1978). Also, the administration of ketamine, which is highly concentrated in the hippocampus, induced a wide range of phenomena that resemble dissociative symptoms in healthy individuals (Krystal et al., 1994).

Hyperarousal (fight/flight) and hypoarousal (freeze) in the face of threat or stress each lead to a different form of dissociation.

**Dissociation in hyperarousal** may occur because the intensity and accelerated pace of sensations and emotions are experienced as overwhelming, and the person cannot stay present with the experience. The

means of dissociation, i.e., of disconnecting from *felt* experience, is generated through the fight/flight response. For example, one may “run at the mouth,” using nonstop verbalization to induce the trance of disconnection, or pick at one’s fingernails incessantly, become lost in compulsive ritualized behaviors, or “perform” for others rather than engage in authentic interactions.

If the child exposed to trauma uses a predominately hyperarousal response rather than a passive dissociative response, the child will be vulnerable to developing persisting hyperarousal related symptoms and related disorders (e.g., PTSD, ADHD, conduct disorder). These children are characterized by persisting physiological hyperarousal and hyperactivity, with increased muscle tone, frequently a low grade increase in temperature, an increased startle response, profound sleep disturbances, affect regulation problems and generalized (or specific) anxiety.

**Dissociation in hypoarousal** utilizes variations of the freeze response, such as drifting consciousness, emotional constriction, social isolation, loss of contact with reality, derealization or depersonalization. Dissociation manifests as an inability to sense or feel even significant events, a sense of estrangement, an inability to accurately evaluate dangerous situations or think clearly, and a lack of motivation. The body, or a part of the body, may become numb, and the victim may experience a sense of “leaving” the body.

“The kind of dissociation described by those with PTSD - altered sense of time, reduced sensations of pain, absence of terror or horror - resembles the characteristics of those who have responded with freezing to a traumatic threat” (Rothschild, 1998). If a child dissociates in response to a severe trauma and stays in that dissociative state for a sufficient period of time, she will alter the homeostasis of the sympathetic/parasympathetic systems. She may develop prominent dissociative symptoms (e.g., withdrawal, somatic complaints, dissociation, anxiety, helplessness, dependence) and related disorders (e.g., dissociative disorders, somatoform disorder, anxiety disorders, major depression) (Perry, 2001).

If a person tends toward hyperarousal (fight/flight) response that is not effectively discharged, his/her body will tend to utilize *parasympathetic dissociation* as a defensive effort to achieve the semblance of homeostasis. If a person tends toward hypoarousal (freeze) response that is not effectively discharged, his/her body will tend to utilize *sympathetic dissociation* to achieve the semblance of homeostasis.

Dissociation may be accompanied by split or altered perceptions not

only of *self* and reality, but also of parts or regions of the *body*. The body carries memories of early trauma and abuse in the form of sensations specific to the site of invasive trauma (e.g., sensitivity in the temple area of the head caused by use of forceps at birth, or tendency to gag or experience choking or suffocation caused by oral sexual abuse). In the same way, the body or body parts carry dissociation, disconnection from feeling, numbness, withdrawal, or hyperactivity. Either way, whether dissociated or not, somatic memory, frozen in place, determines subsequent traumatic stress response: one's shadow behavior is embodied at the time of the overwhelming trauma.

Dissociation, by this model, is a neurophysiological syndrome of central nervous system origin. It is initiated by a failed attempt at defensive/escape efforts at the moment of a life threat, and is perpetuated if spontaneous recovery from the resulting freeze response is blocked or truncated. Lack of recovery from this freeze response results in conditioned association of all sensorimotor information assimilated at the time of the traumatic event into procedural memory, to be resurrected at times of subsequent perceived threat as a primitive conditioned survival reflex (Scaer, 2001).

Based on the concept of somatic dissociation, the region of the body manifesting the symptom (the area of regional somatic dissociation) is capable of communicating to the perceptive therapist what the original trauma was, and provides access to the resolution of that trauma, however belated, in the same way that body memories do. The area of the body that is *not* feeling (*parasympathetic dissociation*) can be equally as important an indicator of stored trauma as body parts that *do* feel (*sympathetic dissociation*).

### *Primary emotions and emotional operating systems*

Darwin (1872) suggested that certain emotions have as their substrate an innate neural basis and, because these emotions are neurally based, they are universally expressed and understood across cultures. These primary emotions include anger, fear, panic, sadness, surprise, interest, happiness (ecstasy), and disgust (Ross et al., 1994). "Thus, the Ventral Vagal Complex . . . is clearly related to the expression and experience of emotion" or more simply "Primary emotions are related to autonomic function" (Porges, 1995).

The emotional operating systems (Panksepp, 1998) can be divided into the primordial set (FEAR, RAGE, and SEEKING) basic to survival; and the social set (LUST, PANIC, CARE and PLAY) characteristic of mammals, which depend on the creation and maintenance of social bonds

for survival. At least four of these primal emotional circuits mature soon after birth, as displayed by all mammals including humans. “The four most well studied systems are (1) an appetitive motivation SEEKING system, which helps elaborate energetic search and goal-directed behaviors in behalf of any of a variety of distinct goal objects; (2) a RAGE system, which is especially easily aroused by thwarting and frustrations; (3) a FEAR system, which is designed to minimize the probability of bodily destruction; and (4) a separation distress PANIC system, which is especially important in the elaboration of social emotional processes related to attachment” (Panksepp, 1998, p. 52).

The SEEKING system is critical for survival; when new resources are needed, it provides the motivation and force to keep going, to move forward, to follow the scent. This system makes animals intensely interested in exploring their world and leads them to become excited when they are about to get what they desire. It engages the frontal cortex, which is involved with planning and expectancy. It generates and sustains curiosity, even for intellectual pursuits, and facilitates learning and the search for higher meaning. The SEEKING system contributes in a fundamental way to our active engagement with the world.

But the over-activation of the SEEKING system can be a trap. When the system becomes spontaneously overactive, which can happen as a result of stress, one’s behavior becomes excessive and manic which is ungrounded and unstable. The shadow side of seeking is frustration, disappointment, and lack, and can descend into self-indulgence. It can act in the service of denial, simply by re-orienting attention away from what is painful. In this way it might be an effective way to fool oneself into believing one’s self-deceptions.

When this brain system becomes underactive, as is common with aging, complacency, or anxiety, depression is the result. There is a lack of hope; a flat feeling, or stuck in a process of mourning which knows no end, Jung’s *abaissement du niveau mental*. Depression is the extreme expression of the constrictive parasympathetic nervous system when the individual feels suffocated, trapped, as if imploding. The constricted SEEKING system, instead of activating a passionate and vital engagement with the world, chokes it off, leading the descent deeper into an unconscious death wish, a lack of enthusiasm for life, or what we call resistance to life.

Its chemistry is characterized by dopamine, a neurotransmitter described as the ‘power switch’ because it turns on, energizing and

invigorating the individual in relation to his/her environment. Dopamine is similar in chemistry to cocaine, and it has the same effect on the individual: creating states of high arousal and focus, and can promote states of eagerness and directed purpose in both humans and animals.

Dopamine appears to be discharged during REM sleep and dreaming, suggesting that dreams are linked with the SEEKING function. In addition, the hippocampus exhibits highly synchronous rhythmic theta activity during REM.

During waking, this type of hippocampal synchronization (also known as *theta rhythm*, which is common when animals are exploring their environment) usually indicates that the circuits are systematically encoding information (i.e., translating recent experiences into long-term memories) (Vanderwolf, 1981; Vertes, 1982). . . . Although we do not presently know exactly how memories are consolidated during REM, we can anticipate that the hippocampus will be in the middle of the neuronal action. After all, the hippocampus is the brain area that is well established to be a mediator between short- and long-term memories, and it goes into a characteristic *theta* state during REM. Also, it is now clear that the types of information processing that were undertaken by the hippocampus during waking are rerepresented during the endogenous dynamics of the REM state (Pavlides & Winson, 1989; Wilson & McNaughton, 1994; Winson, 1993)" (Panksepp, 1998, p. 129).

The RAGE emotional operating system works in opposition to SEEKING, energizing the body to angrily defend its territory and resources. RAGE is aroused by frustration and attempts to curtail an animal's freedom of action. As a general principle, anything that restricts our freedom will stimulate the neural circuitry of RAGE within the brain. For instance, a human baby typically becomes enraged if its freedom of action is restricted simply by holding its arms to its sides (Campos et al., 1994). Other common experiences that stimulate RAGE are when one does not receive expected rewards, i.e., when one is frustrated, and when one is "cheated" out of something promised or expected, i.e., when one feels betrayed. The RAGE system can, of course, provide the ignition for behaviors that may not look like anger and aggression. An example is addictions, provided by Sterling (2004, pp. 22-23):

Cortisol and related signals are elevated, not only during hypervigilance, but also during states of hyposatisfaction – when outcomes prove less than expectations. Because satisfaction cannot be stored, it must be continuously renewed. So if its potential sources become constricted, the brain must inevitably rely on those that remain: people needing a pulse of satisfaction will try to find it somehow (Figure 7). For those of higher socioeconomic status there are opportunities for satisfaction in work, achievement, and money. Mono-pursuit of such opportunities tends to spiral out of control ("workaholism", "type A" behavior, etc). This may occur especially when expectations inculcated by the family as "prior knowledge" are so high as to be intrinsically unsatisfiable. Another likely



factor is that a stimulus which initially releases dopamine adapts, limiting the satisfaction obtainable from its repetition.

For people of lower socioeconomic status potential sources of satisfaction are less available, but food is abundant and cheap. So the allostasis model suggests that the brain overrides local negative feedback (metabolic satiety signals) – just as it overrides the negative feedback that would counter commands to raise blood pressure – and people eat. For the reasons just cited, satisfaction is fleeting – so people eat even more (Saper et al., 2002; Schultz, 2002).

Alcohol and drug addictions follow a similar pattern and apparently share many of the same mechanisms (Wise, 2003).

The FEAR system is essentially the ‘fight, flight or freeze’ reflex. There are two circuits for fear, a hot one and a cold one (the same as for traumatic memory): the cold one takes information about stimuli and associates it to a time and place (active involvement of the hippocampus) and also makes it available for conscious reflection (activated cortex); the hot circuit goes directly from stimuli to response (reflexive amygdala action) bypassing the sense-making structures. The FEAR system is active in sudden terror and in persistent feelings of anxiety. It interacts with the RAGE circuits, which contribute to the choice of or balance between fight and flight reactions.

It makes good evolutionary sense for FEAR and RAGE circuits to be intimately related, for one of the functions of anger is to provoke fear in competitors, and one of the functions of fear is to reduce the impact of angry behaviors from threatening opponents. Although it has not been empirically demonstrated, it is reasonable to suppose that at low levels of arousal, the two systems are mutually inhibitory (Panksepp, 1998, p. 208).

PANIC is the name given to separation-distress which is part of the attachment system. The undesired loss of a lover or the unexpected death of a loved one can plunge us into one of the deepest and most troubling emotional pains that we, as social creatures, can experience. This response is sorrow or grief, and it can verge on panic in its most intense and precipitous forms. At a less acute but more persistent level, the same essential feeling is called loneliness or sadness. As Panksepp puts it, we are wired to cry and wail when we feel abandoned. The PANIC system is fundamentally bound up with abandonment, mourning and loneliness. It is closely linked to the perception of pain as well; contact comfort releases opiates which soften pain. Separation, on the other hand, rapidly diminishes the supply of opiates, leaving the individual with very real ‘withdrawal’ symptoms facing the agonizing feeling of abandonment and loss. Oxytocin and serotonin are important in quelling this emotion.

We know that panic is the extreme expression of the expansive sympathetic nervous system when the individual feels uncontained, unsupported, or unboundaried. During separation distress as well as during panic attacks, the victims feel as if their center of comfort and stability has been abruptly removed, leading them to actively solicit help and social support. Both are commonly accompanied by such autonomic symptoms as a feeling of weakness, difficulty in getting one's breath, and a feeling of having a lump in the throat (Uhde & Tancer, 1988). People who suffer from repeated panic attacks typically have had childhood histories characterized by separation anxiety problems (Torgersen, 1986).



Figure 7.

The four most primal emotional systems (Panksepp, 1998, p. 53)

The additional systems for sexual, maternal, and playful feelings will be called LUST, CARE, and rough-and-tumble PLAY systems.

The PLAY system points first towards active physical engagement and improvisation and only secondly towards symbolic or fantasy play. The rough and tumble play exhibited by all healthy young mammals facilitates the learning of physical and social skills. It engages the parts of the brain linked to somatosensory information processing and convergence. It enables us to find the limits and possibilities in our behavioural repertoire, and I suspect that it plays a critical role in helping establish good clear flexible boundaries. . . . PLAY comes into operation only when basic needs have been met – until that point other instincts predominate (Carroll, n.d.).

PLAY and SEEKING are complementary systems. Whereas REM sleep (related to the SEEKING system) assists in organizing affective information, PLAY seems to utilize the same information through trying it out in action. In other words, Panksepp suggests that PLAY may be the daytime version of dreaming.

All these emotional operating systems have prototypical physical gestures, stances, and facial expressions, which eventually after extensive habitual use become the muscular armour discussed by Reich. Psychotherapists familiar with the somatic expression and suppression of emotion can, through observing the body, quickly identify which system has been activated (*sympathetic dissociation*) or is chronically inhibited (*parasympathetic dissociation*). A basic premise of somatic therapies is that support to enter fully into a gesture, a stance, a facial expression, a body movement, or a sound can bring the individual into a conscious experience of the underlying dissociated emotional pattern. .

### *Subtle bodies*

The opposite of dissociation is embodiment, or indwelling, which involves connection and identification with the physical body, but also with one's subtle energy field, or subtle body.

Jung suggested that the Eastern idea of the subtle body could be compared to his idea of the *somatic unconscious* defined to be the unconscious as perceived in the body (1988, p. 441). This state requires a free flow of energy that is felt as a wave moving up and down the body; then, one begins to feel that one inhabits the body. In this state, the body is a container. The condition of being embodied is an experience of a medium that exists between one's material body and mind. Thus, our unconscious thoughts, feelings, and beliefs exist in the subtle body, and to the extent they remain unconscious they manifest as physical structure and physical symptoms. Thus the unconscious feelings and beliefs are *embodied*. *Indwelling* is the process of embodiment of the higher energies, making the soma conscious. The seven major chakras are the intersections of contact between subtle bodies and the physical body, and are also gatekeepers to monitor the passage of energies between bodies. If the incoming flow of energy, from the subtle level to the physical, is weak or blocked (i.e., distressed indwelling), the person suffers physically, emotionally, or cognitively. If the outgoing flow of energy, from the

physical level to the subtle, is weak or blocked, the person's ability to discharge is impaired.

The subtle energy bodies also provide the vehicle for autonomic and unconscious communication between people, e.g., transference, countertransference, synchronizing of hormonal stimulation, projection, introjection. This can occur through either harmonizing or dissonance. When the two people's frequencies are compatibly similar to each other, there is what is called synchrony, sympathetic vibration, resonance, or rhythm entrainment. This has the effect of amplifying the pattern within both. It is through this process that the hypnotic and NLP techniques of modelling and pacing create their effect. It is through this process that the therapist provides containment for the other's experience which is too activated for him/her to tolerate. It is through this process that shock is so easily contagious among people. If on the other hand the frequencies are dissonant, an imbalance is created in the charge/ discharge sequence for each person.

### *Gender differences*

There are marked gender differences in the response to traumatic violence (Perry et al., 1995a; Perry et al., 1995b). Females are more likely to dissociate and males more likely to display a classic "fight or flight" response. As a result, more males will tend to develop the aggressive, impulsive, reactive and hyperactive symptom presentation (more externalizing), while females will tend to be more anxious, dissociative and dysphoric (more internalizing). In fact, studies by Carmen et al. (1984) and Jaffe et al. (1986) indicate that abused men and boys tend to identify with the aggressor and later victimize others whereas abused women are prone to become attached to abusive men who allow themselves and their offspring to be victimized further. Brain metabolic evidence in humans validates these tendencies: temporal lobe areas (where aggression circuitry is concentrated) are more active in males, while cingulate areas (where nurturance and other social emotional circuitries are concentrated) are more active in females (Gur et al., 1995). Of course, identification with the abuser victimizes not only others but also oneself, just as collaborating with the abuser does.

Some of the gender differences are accounted for by oxytocin, a hormone which prompts labor as well as milk production in the mother. Oxytocin is also released during stressful events, prompting the uniquely female stress response of *tend and befriend* (Taylor, 2002): tending to

dependent children, and seeking out social support. A complication is that oxytocin's effects are enhanced by estrogen, but are antagonized by androgens, male hormones. In an interesting experiment recently, human subjects who inhaled oxytocin in a nasal spray showed increased trust in social interactions with unfamiliar individuals (Kosfeld et al., 2005).

### *Spiritual depression, trauma re-enactment, attachment, and Borderline Personality Disorder*

Survivors can be drawn to re-enactment of the traumatic abuse, and can actually come to *psychological bypass* (the inverse of *spiritual bypass*, in which a person focuses on spiritual experience as a means of avoiding their unresolved emotional issues). Psychological bypass is a strategy of avoiding growth into one's highest potential, including spiritual development, by remaining preoccupied with the effects of early trauma in one's life. We recognize this condition of psychological bypass by another name as well: *spiritual depression*. Van der Kolk (1989) discusses this:

Some traumatized people remain preoccupied with the trauma at the expense of other life experiences (van der Kolk, 1987; van der Kolk, 1988) and continue to re-create it in some form for themselves or for others. War veterans may enlist as mercenaries (Solursh, 1987), victims of incest may become prostitutes (Gelinas, 1983; Russell, 1986; Silbert, 1981), and victims of childhood physical abuse seemingly provoke subsequent abuse in foster families (Green, 1980) or become self-mutilators (van der Kolk et al., 1988). Still others identify with the aggressor and do to others what was done to them (Burgstein, 1985; Fish Murray, 1987). Clinically, these people are observed to have a vague sense of apprehension, emptiness, boredom, and anxiety when not involved in activities reminiscent of the trauma.

The mechanism for trauma re-enactment for the person whose attachment style is *preoccupied* is to focus on "what is missing, what might have been, what never can be." He/she will idealize the pre-trauma past and feel bitter resentment at its having been taken away. This person experiences deep loss of self-confidence, and a timidity about attempting to overcome the loss.

For the person whose attachment style is *avoidant* it is to focus on "doing what I have to do, getting on with it. I will deal with this on my own." He/she will dismiss or discount the value of what has been lost (innocence, spirituality, or personal expression) and inflate a positive self image.

For the person whose attachment style is *disorganized* it is to be highly anxious about what is missing, but yet fearfully avoidant about it. He/she

will dismiss its value, and at the same time diminish his own. This resembles the intrusion-avoidance chaos of PTSD, with negative beliefs about what has been lost *and* about the self. Remember that 80% of traumatized children have disorganized attachment patterns (van der Kolk, 2001).

Borderline personality disorder (BPD) is a severe, chronic disorder characterized by affective, impulsive, and interpersonal symptoms. Fear of abandonment, a diagnostic criteria for BPD, is considered a core feature that best discriminates BPD from a range of other psychiatric disorders (Gunderson, 1996), along with a sense of inability to regulate negative affect (Mikulincer and Florian, 1998) and poor distress tolerance. We will examine this cluster of symptoms in light of the preceding discussion of neuroception, failure of self-regulation, and faulty functioning of the social engagement system.

Individuals with BPD exhibit disturbed adult social attachment along two fundamental dimensions, and the pattern of childhood maltreatment common to all those with BPD differs along those same two dimensions.

Individuals with BPD are often depicted as exhibiting a “preoccupied” type of insecure attachment (Fonagy et al., 2000), characterized by hypervigilance in attending to and expressing distress. However, two dimensions of adult attachment disturbance have recently emerged from empirical studies (Brennan et al., 1998): one dimension, *attachment-anxiety*, refers to extreme emotional reactivity and negative affect (e.g., fear of rejection and abandonment) in relationships; a second dimension, *attachment-avoidance*, refers to emotional and behavioral strategies for defensively creating interpersonal distance.

Minzenberg, Poole, and Vinogradov (2006) examine the particular form of approach-avoidance conflict exhibited by persons with BPD that has been described as “oscillations of attachment” (Melges & Swartz, 1989), and postulate specific adult symptoms exhibited by individuals with BPD in each of the two dimensions. Their sample of clinically stable adults with BPD was characterized by elevated scores on each of two dimensions; however, they found a divergent pattern for the two attachment dimensions. Attachment-anxiety was specifically related to reported sexual abuse, whereas attachment-avoidance was generally related to all types of childhood maltreatment.

The two dimensions were also associated with different current symptom profiles. Elevated attachment-anxiety was associated with hostility and antagonistic behavior in relationships (both assaultive and

self-injurious), as well as attentional and motor impulsivity. In contrast, elevated attachment-avoidance scores were related to depressive symptoms, as well as the relational withdrawal and active disengagement that patients use to manage distressing affective symptoms that they are unable to regulate internally.

We will apply our newly derived general principle: If a person tends toward hyperarousal (fight/flight) response that is not effectively discharged, his/her body will tend to utilize *parasympathetic dissociation* as a defensive effort to achieve the semblance of homeostasis. If a person tends toward hyporarousal (freeze) response that is not effectively discharged, his/her body will tend to utilize *sympathetic dissociation* to achieve the semblance of homeostasis.

These two dimensions exhibit symptoms with a clear bias toward sympathetic and parasympathetic response patterns, respectively. The *attachment-anxiety* dimension, with its tendency toward extreme emotional reactivity and negative affect, hostility and antagonism, is an expression of a *sympathetic dissociation* defense. Those who exhibited this pattern were predominantly sexually abused as children, and it is logical that their initial response to that abuse was parasympathetic in nature (lay still and pretend to be asleep; dissociate somatically; feel shame and self-blame). The *attachment-avoidance* dimension, with its tendency toward depressive symptoms and defensively creating interpersonal distance through withdrawal and disengagement, is an expression of a *parasympathetic dissociation* defense. Those who employ this strategy were subjected to other types of childhood maltreatment, which may have triggered a more active fight or flight response (run away from home; attempt to placate the caregiver's hostility or to be peacemaker between mother and father).

This perspective on BPD offers a clear example of the long-term ill effects of child maltreatment in hypervigilant neuroception, failure of self-regulation, and faulty functioning of the social engagement system.

### *Spiritual awakening*

Just as the body's central nervous system has two branches, the sympathetic and the parasympathetic, to provide balance and counterbalance in responding to the environment, the body's subtle energy system has two branches, seemingly opposites yet neither of which can exist without the other. We have discussed at length the interactions between the two primary psychic forces at work (or, perhaps, at play) in human lives: Eros (*libido* energy of satisfaction, passion, attraction and

openness to life and growth) and Thanatos (*mortido* energy of withdrawal, disintegration, oblivion, and resistance to life and growth). We conceptualize many conditions of pathology as an imbalance between these two forces in a person's life (Hartman & Zimberoff, 2004).

Libido energy is carried by the sympathetic system: the UP, energy-consuming experiences. In extreme, the effect is frenzied. Mortido energy is carried by the parasympathetic system: DOWN, energy-conserving experiences, which in hypoarousal lead to a sense of numbness, shutting down, social isolation and withdrawal, constricted affect, denial, cognitive impairment and dissociation. In extreme, the effect is depression and ultimately death.

Spiritually, the two systems combine to create conscious energization. The kundalini energy lies dormant in the base of the spine, in the lowest levels of physical instinctual being. And it is in the instinctual unconscious (Jung's *somatic unconscious*) that the primordial force is initially discovered. The symbol in the east is that of a lotus flower, which grows up from roots in the thickest slimiest mud at the bottom of the swamp. Kundalini is not the same as sexual energy, but they are both carried in the same channels: the *pingalā* (sympathetic) and *idā* (parasympathetic). Before the awakening of kundalini a benign form of this energy (*prāna*) flows through these two energy channels, usually unnoticed. With the kundalini awakening, the primal force rises up and overactivates the autonomic nervous system, often producing the type of physical symptoms common to overstimulation of one branch over the other: sensations of heat or cold, experiences of hyperactivity or fatigue, racing or slowing of the heart, etc. In the course of rising up through higher levels of refinement, that energy is "liberated from its instinctual organization as libido" (Washburn, 2003, p. 163), and clarifies to reveal itself as the free and awakened expression of Shakti herself.

Libido energy alone cannot entice her to awaken and rise up from potentiality; libido energy alone too quickly becomes frenzied in its expansiveness. Mortido inertia alone cannot quell her destiny to rise up and dance. The two forces together, holding each other in check, create the necessary conditions for her triumphant ascension. "Holding each other in check" is co-operation, not to be confused with the impasse of stalemate. When there is impasse in the struggle between libido and mortido, when neither gains advantage, then movement ceases and paralysis sets in: stalemate. In stalemate, the battle rages on, usurping all available energy. This is the condition of such rigidity (unvarying intervals between heart



beats), measured as low heart rate variability, that death is the outcome. To the contrary, the co-operation of holding each other in check allows each full uninhibited expression, producing the zenith of resilience.

Both vagal circuits (sympathetic and parasympathetic) share in regulating the heart, and it is here in the heart that their co-operation or competition produces rhythmic coherence or erratic disorder. When the two vagal circuits are out of balance, or are competing, the heart rate variability is low which means excessive sympathetic activity or excessive parasympathetic activity, and which in the extreme signals impending death. Remember, heart rate variability (HRV) reflects the degree of balance between sympathetic and parasympathetic activity, and a higher HRV reflects an ability to adapt to changes in the environment; it shows stress resiliency.

Spiritually, too, it is in the heart that these two primal forces must achieve co-operation, or suffer the fate of spiritual depression and psychic death. Once that primeval energy reaches the heart, home of both passion and compassion and therefore of spiritual striving, it has the possibility of becoming liberated from its instinctual organization as libido, not just intermittently but permanently, as the clarified spiritual energy that is its essence. The heart is the halfway point between the mud and the lotus blossom. That primeval energy journeys from its inception in potentiality, coiled at the base of the spine, up to the heart within the confines of the channels of instinctuality, i.e., libido. However, liberation from that confinement, allowing free and unfettered movement up to the highest level of organization spiritually, allows it to “jump the tracks” and access the primary and central energy conduit connecting the lower regions of the body to the head. No longer confined to the central nervous system channels, the *pingalā* and *idā*, it now is free to ascend in its true form through the *sushumnā*.

Another parallel in the eastern formulation of spiritual development and the western formulation of autonomic nervous system balance is the vital importance and central role of the breath. Science is coming to a clear understanding of the role of the breath in balancing the HRV, and of the fundamental role HRV has in overall health and extending mortality. The three energy channels, the *pingalā*, *idā*, and *sushumnā*, begin together at the base of the spine, in the first chakra, where their meeting is known as *Yukta Triveni* (*Yukta* means “combined”, *tri* means “three”, *veni* means “streams”). The dormant kundalini, symbolized by a serpent, lies blocking the entrance to *sushumnā*, the central channel. *Sushumnā* remains closed at

its lower end as long as kundalini is not awakened. The three channels follow separate paths in their upward journey, crossing paths at each chakra but not meeting until the third eye. The meeting of the three is called *Mukta Triveni* (*Mukta* means “liberated”). Continuing beyond the third eye chakra, *idā* and *pingalā* end in the left and right nostrils, respectively, where they provide access to direct management of the body’s energy through regulated breathing. The ancient formulas for breath patterns to balance the male/ female energies of the *pingalā/ idā* energies are remarkably similar to those found to be most effective in regulating the HRV.

The *sushumnā* continues upward to the crown chakra, where Shakti, the feminine, dynamic, energetic and creative force, meets Shiva, the masculine, unmanifested consciousness. Shiva has the power to be but not the power to become or change. It is from the Great Mother Shakti that all form is born and all change is possible. This union creates a resolution of duality into unity once again.

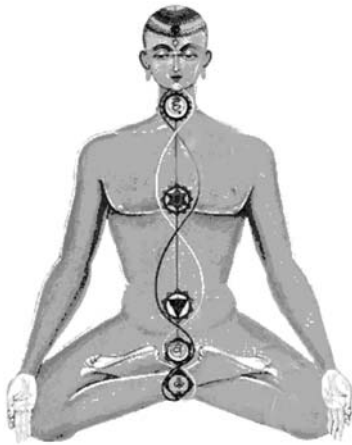


Figure 8.

The Chakra drawing on this page is from the book: *Chakras: Energy Centers of Transformation* by Harish Johari, Destiny Books, a division of Inner Traditions International, Rochester, VT 05767 Copyright © 2000 by Pratibha Johari.

*Idā* is the left channel. *Idā* is white, feminine, cold, represents the moon and is associated with the river Ganga (Ganges). Originating in Muladhara, *Ida* ends up in the left nostril.

*Pingalā* is the right channel. *Pingalā* is red, masculine, hot, represents the sun and is associated with the river Yamuna. Originating in Muladhara, *Pingala* ends up in the right nostril.

*Sushumnā* is the central channel and is associated with the river Saraswati. Running up the body from just below Muladhara chakra to Sahasrara chakra at the crown of the head.

The technique of Kundalini Yoga consists in using Prana, guiding its circulatory movement through *Idā* and *Pingalā* down to the base of the spine into the space where Kundalini lies coiled. **Shakti Kundalini will then awaken and rise up *Sushumnā*, energizing the seven chakras.**

### *Treatment implications*

Steven Porges' therapies for healing trauma, based on his poly-vagal theory, confirm the principle that *less stimulation is more effective for a challenged nervous system*. A second principle is that *the intervention must take place in a safe environment*. A third principle is that *brainstem regulation is the foundation of the self-regulation process*.

Onno van der Hart has identified a similar acknowledgement of the necessity of safety. *The work must largely be done without awakening the defenses, and without tossing the individual back into the maelstrom of his own dysregulations. Work with trauma, to the degree possible, must be done without reawakening it.* van der Hart suggests that it is neural networks that organize integration and dissociation, social engagement and immobilization. And that the organization of the momentary engagement and disengagement of neuronal networks is the heart of self-regulation. The object of therapy for the traumatized individual is to move beyond the "constricted life lived at the surface of consciousness," the zone of provisional safety carved out within the maelstrom of his/her own dysregulation, and to build healthy self-regulation that allows responsiveness to the momentary environment, inner and outer.

Richard Kluft agrees that in healing traumatized individuals, "the slower you go, the faster you get there."

Bessel van der Kolk talks about the necessity, in the process of healing trauma, of helping people gain awareness of their own physical selves and function here in the present. He suggests that PTSD is all about the integrity of brainstem functioning, through its management of autonomic arousal. An individual's action patterns and hormonal responses are mobilized unconsciously and automatically by the emotional brain, impervious to conscious control, and healing means to bring them within conscious control (self-regulation). He provides this startling analogy for the lack of pathways between the rational brain and the emotional brain in the traumatized individual: the worst form of PTSD may be waking in the course of a surgical procedure and being unable to tell the surgeon that you are awake. When a therapist facilitates the accessing of traumatic memories with a client without appropriate attention to coordinating the hard-wired defenses, re-traumatization is likely to occur, and your client may not even be able to communicate to you that it is happening.

Allan Schore elaborates the theme of the "embodiment" of trauma in dysfunction of the right hemisphere of the brain. For the traumatized person, not only is the rational domain somewhat disconnected from the

emotional, but the right hemisphere encodes a very different emotional life experience than the left. Because the right hemisphere can only be accessed and addressed non-verbally, successful psychotherapy must be an inherently *embodied* process. When the right hemisphere develops incompletely, the person develops an apparently normal personality to live in the self-sufficient, detached world of the left hemisphere, “on the surface of consciousness,” in the zone of provisional safety. Living in the refuge of left-hemisphere functioning allowed the traumatized individual to survive and navigate through life, of course, but it now stands as a formidable obstacle to ultimate healing.

Siegfried Othmer, reflecting a neurofeedback treatment approach to trauma, suggests that it is best to start with where the person already functions competently (rational left brain), and proceed from there to enlarge the scope of functioning incrementally and gently. “Engaging the trauma experience head-on . . . may be a mistake.” Rather, “a stealth strategy is needed that builds self-regulation from the ground up, a kind of recapitulation of the developmental processes that is available even at a relatively late stage. All the neuronal pathways are available for the learning or re-learning of self-regulation in the affective and arousal domains” (Othmer, n.d.). He suggests, in a slightly revised manner from van der Hart, that “the trauma itself need never to be touched until it is already transformed.” An even more explicit statement of this principle is offered by Rothschild (1998):

“It is these three areas - containment, positive body awareness, dual time awareness - that must first be strengthened, before addressing the memory of a traumatic event can be done productively. Containment of out-of-control emotions and thinking processes will help restore a feeling of control over the psychological self. Positive body-awareness will help restore a sense of the body and its sensations as friend, not foe. Dual time awareness will help to separate that the trauma occurred in the past even though it feels as if it is occurring now (Rothschild 1996, Rothschild 1997).”

Ultimately, we are assisting the client to increase self-regulation of internal states, and to increase the ability to tolerate distress, i.e., to decondition the traumatic stress response. This is fundamental to healing trauma, because people’s core sense of self is defined to a very large degree by their capacity to regulate internal states and regulate their response to stress (Cole & Putnam, 1992).

Knowledge of the tendency toward and interaction between sympathetic and parasympathetic nervous system responses leads to invaluable awareness of the differential approaches to treatment. While

accessing the hyperarousal or hypoarousal response, we must facilitate the person to stay present, feel safe, communicate their experience, and feel empowered. Following are some suggested guidelines for treatment of trauma, complex trauma, DESNOS, and shock.

1. **Approach trauma-related material very gradually** to avoid intensification of the affects and physiologic states related to the trauma. Be watchful for the client's overwhelm and over-activation above or below the optimal arousal zone. When that occurs, immediately discontinue working with the content of the trauma memories, and address the need for safety, containment, body awareness, and connection to support. It is helpful to direct the client's attention to events before and after the incident, working gradually to the actual experience itself. Once at the point of accessing traumatic memories, encourage the client to take all the time he/she needs to slow down the experience of the traumatic moment, and to fully experience the data interpreted to be a threat. This helps the client to validate accurate neuroception, locate and assess the threat, and have time and space to respond to it consciously rather than autonomically. It also offers an opportunity to rehearse a threatening triggering event with a sense of advanced warning.

With the advent of distinguishing between trauma and complex trauma, or PTSD and complex PTSD, we now know that certain prerequisites must be in place for the client before proceeding to confront the trauma directly, namely

the capacity of our patients to modulate their affective arousal: whether they are able to be emotionally upset without hurting themselves, becoming aggressive, or dissociating. As long as they cannot do this, addressing the trauma is likely to lead to negative therapeutic outcomes. Similarly, as long as they dissociate when they feel upset they will be unable to take charge of their lives and will be unable to 'process' traumatic experiences (van der Kolk, 2001).

2. **Cultivate an acute awareness of inner body sensations** (bodily feelings of a distinctly physical character, such as trembling lips, heaviness in the chest, twitching in the hands, clamminess in the feet, tightness in the throat, tension in the buttocks, pounding heart, numbness in the extremities, tingling in the right arm, shallow breathing, or vibrating sensations in the solar plexus area, etc.). "Through cultivating such awareness and ability for verbal description, clients learn to distinguish and describe the various and often subtle qualities of sensation. Developing a precise sensation vocabulary helps

clients expand their perception and processing of physical feelings in much the same way that familiarity with a variety of words that describe emotion aids in the perception and processing of emotions” (Ogden & Minton, 2000).

The therapist points out subtle sensorimotor changes and gross muscular movements through a simple “contact” statement such as, “It seems like your neck is tensing,” or “Your hand is changing into a fist,” or “There’s a slight trembling in your left leg,” just as one would notice and point out emotional states (“You seem scared”).

The therapist asks questions that require awareness (mindfulness) to answer, such as, “What do you feel in your body? Where in your body do you experience that? What sensation do you feel in your legs right now? What happens in the rest of your body when your hand makes a fist?” This focusing is especially important when the client is accessing traumatic memories so that he/she can experience the content of that moment consciously embodied. Questions such as these encourage the client to come out of a dissociated state, and future- or past-centered ideation, and be really present in the body, experienced from the perspective of an observing ego whose intention is simply observing rather than controlling or changing the experience, or reacting to it.

3. **Access enough traumatic material to process but not so much as to dissociate.** The therapist must “hold” the client’s arousal within the optimal limits, while encouraging the accessing, exploring, and expressing of the trauma memory. When arousal reaches either the upper or lower limit, ask the client to temporarily suspend attending to her feelings and thoughts and instead focus on observing the physical sensations and movements in detail until these sensations settle and the movements complete themselves. Attending to and tracking these sensations allows them to change or “sequence,” through an observable pattern and eventually back to within optimal limits. “Once you become aware of them, internal sensations almost always transform into something else” (Levine, 1997, p. 82).

Redirect the person’s attention by asking him/her to refocus as well as in other ways. That may be with a touch or specific eye contact, a sip of water or inquiring about level of comfort. In addition to interrupting the dysfunctional sequence, such attending to the personal comfort and safety of the client is nurturing and elicits their

own social engagement activation. This process is one of titrating the traumatic pattern, alternating between small pieces of the traumatic material and one of the client's resources, discharging the activation in the nervous system that emerges as the person slowly works through the traumatic event. Clients thus develop their capacity to self-regulate, limiting the amount of trauma they process at any given moment, learning their own unique predictable pattern or sequence, and preventing themselves from going into overwhelm.

4. **Interrupt a client's unconscious, autonomic patterns** somatically. One of the most common patterns is some form of "bracing", a condition in which a person in stress resorts to a destructive habitual physiological response, such as muscle tension, vaso-constriction or dilation, or breath suppression. A timely reminder to breathe can be immensely empowering to the person's body. The vaso-constriction or dilation in the hands or feet, stomach, neck or forehead, or any particular body part, causes them to be too cold or too hot, respectively. Hence, the application of the reverse effect (heat or cold) brings the constriction to the body's attention (not to conscious awareness) and allows a relaxing of the hyperarousal. Encouragement to yell or scream when the individual is imploding with suppressed fear is permission and an instruction *to the body* to change a very old and deeply embedded habit. One of the most effective means of relaxing muscle tension is laughter. The therapist can insert humor to accomplish this, as long as it is properly timed and not used to dissipate emotion and thus avoid cathartic release.
5. **Work toward cathartic release**, or other physical forms of release such as "unwinding" the paralyzed energy in limbs or head. Levine discusses the concept of "exchanging ...an active response for one of helplessness" (1997, p. 110). Here we are coaxing the person to use will power, and the support of the therapist or group members, to initiate a sympathetic response (fight/flight) to replace the deeply embedded parasympathetic response (freeze). For the person to experience the active defense sequence (pushing with legs or arms, moving out of paralysis into proactive movement, yelling "No!") brings her out of dissociation, into her body, and begins a *kinesthetic reframing* process. The client may begin to experience the somatic *pleasure* of physical resistance and defense, and the emotional delight of a new experience of personal power. The sequence, produced

through will power and trust, becomes a template for the same new response to occur spontaneously in the future.

Somatic and emotional release also lifts spiritual blockages. “The deeper the catharsis, the more likely that core aspects of the psyche will be touched at the level of transformational and/or spiritual energies. By ‘transformational energies’ we refer to the essential energies that connect us to the Self, energies that we can access through meditation, or other spiritual disciplines” (Emerson, 2002, p. 69).

6. **Experience having personal needs acknowledged and responded to** by a caring and safe support person. The therapist may offer a drink of water (which also flushes toxins from the system), offer heat or cold if it would be soothing, offer additional means of protection (such as something to cover the vulnerable umbilicus, the hand to hold of a supportive group member, or bringing into an age regressed scene someone from the person’s life at that time to ameliorate the terror), or establish direct contact nurturing (touch, open eye contact). Included here, too, would be expressions by the therapist of acceptance of the person’s feelings, outrage at the abuser, empathy for the hopeless predicament of the child, or the affirmation that any child would react the same way in order to normalize the child’s behavior.
7. **Discover and develop personal resources**, real or imagined, that would be available so that the client is not facing the threat alone, isolated, in secret, or unsupported. The resources help to initiate a social engagement response and to discharge excess fight/flight or freeze messages. Over time the client will continue to build an inventory of resources as he/she accumulates them in subsequent sessions. As much as possible, encourage the client to develop his or her own resources rather than having the therapist suggest specific resources. The resources should be experienced not just as an imagined creative visualization, but with awareness to the sensations experienced in their bodies. These somatic experiences are then bookmarked as anchors for the underlying state, to be used in the person’s life to better handle triggers as they arise. The therapist must devote time and attention to the NLP technique of anchoring every resource state for the client, making the anchor multisensory, vivid, and memorable.



8. **Speak the experience of victimization** in order to integrate the memory functions and lessen the immobilizing emotional/sensory reflex. The client is literally reconnecting the experience (implicit or body memory) with the cognitive context for it (explicit memory), and in the process is repairing the physical damage done to her hippocampus. Verbal processing of an experience enhances the potential for memory-making (Bowman, 1996). A child's memories of anything are more enduring when they have been talked about reflectively with caregivers at the time of occurrence (Bauer & Wewerka, 1995). Therefore, having no opportunity to verbalize, or being forbidden to tell, at the time of the original trauma inhibits the child from speaking and effectively contributes to memory loss (repression). Verbalizing reflectively in the age-regressed traumatized ego state, in the presence of the caring therapist, begins to repair the damage through regeneration within the hippocampus.
9. **Reconnect memories and emotions to events.** Bucci (2003) states the case succinctly: "One may be aware of the physiological activation, the painful physical arousal, associated with the activated schema of anger or fear, and also aware of aspects of one's history, including the trauma and abuse, *but without connecting the two*. We see patients telling the stories of their history without emotion, feeling the physiological activation without recognizing its emotional connections" (p. 548, italics added). This is dissociation. The individual has an experience in the present that is perceived to be dangerous enough to lead to a central nervous system arousal – butterflies in the stomach, or a gnawing dread sensation in the chest, or painful stimulation in the sexual areas. These are body memories, triggered by the intensity of the arousal. Or the arousal may be cognitive – a sense of familiarity, or of the "other shoe about to drop." The individual is unable to consciously acknowledge the true source (distant past trauma), instead mistakenly attributing the arousal to the current triggering circumstance. That the activating trigger can as easily be in fantasy as in an actual life event makes it no less arousing, and the consequences set in motion no less devastating.

The physiological or cognitive activation is not connected in memory to the source of the trauma because they were not connected at the highly stressful time of originally laying down the memory (LeDoux, 1999).

10. **Reframe basic assumptions** about the self as secure and intrinsically worthy, and about the world as orderly and just. The person's most deeply embedded beliefs are accessible for review and reframing, because in the age regression process you have arrived at the very scene of their inception. These personal conclusions and beliefs about the self and the world (Attachment Theory's internal working models) are held by the immature, developmentally arrested inner child and carried by the physical body (Reich's character armor). They are the unconscious foundation for all the adaptive, dysfunctional behaviors that sabotage an individual's optimal expression of self (Jung's shadows).
11. **Release the anger and blame directed against the self** for the inability to defend against the abuse. A common response among trauma survivors is to interpret her dissociation and freezing as a personal weakness (Nijenhuis & Van der Hart, 1999). This is a golden opportunity for re-establishing a loving, accepting relationship with the inner child, who until now the client has likely reviled and rejected. One of the most important truths that child ego state needs to hear and accept from the adult ego state is a reattribution of responsibility (Hoagwood, 1990; Morgan & Cummings, 1999). "It was not your fault. There was nothing you could do to stop it from happening. The responsibility and blame belong to the perpetrator."
12. **Locate the traumatic experiences in time and place**, to start making distinctions between current life stresses and past trauma. During traumatic threat, it has been shown, the hippocampus becomes suppressed. Its usual function of placing a memory into the past is not active. The traumatic event is prevented from becoming a memory in the past, causing it to seem to float in time, often invading the present (van der Kolk, 1994; Nadel & Jacobs, 1996). In the words of an incest survivor in treatment, "I'm stuck in my past. It is like a never ending past life" (Putnam, 2004, p. 237). We must recognize, too, that traumatic memories are stored as emotions and senses (implicit memory), and are therefore often hazy, impressionistic, or kinesthetic. Understandably, survivors become haunted by feelings and senses they suspect are related to the trauma, but cannot clearly identify as explicit memories.

Sometimes it takes a lot of patience to eventually get to clear memories of what happened at the time of the trauma. Van der Hart

and Nijenhuis (2001b) verified a long-standing observation in the field of psychiatry, referred to as Ribot's law of regression, that the recovery of lost procedural and semantic memories precedes the recovery of episodic memory, particularly in the recovery of trauma memories. It is common in therapy with trauma victims to observe their healing process begin with awareness of "body memories" and other procedural-implicit memories, and then gradually expand to include specific episodic memories of where, when, and how the trauma occurred.

13. **Learn to trust and surrender to a healthy interpersonal relationship** with the therapist, who demonstrates an ability to understand the client's distress and tolerates the description and re-experience of her traumatic experience without as therapist withdrawing or becoming hyperaroused or hypoaroused. This is the level of containment that the client can most directly experience. However, it requires patience because such an individual has a deeply embedded uncertainty about the reliability and predictability of others, especially those who are caregivers or authorities. The therapist must overcome the client's mistrust and suspicion the old fashioned way: he/she must earn it. This growing trust in another becomes a rehearsal for transferring this willingness to other relationships in the client's life.
14. **Recognize that current life stresses tend to be experienced as somatic states**, and accept physical symptoms as ally rather than enemy. Physical symptoms are the body's way of communicating an unmet need to the person; they are not a statement of defiance, mutiny, hostility, or weakness. Developing an awareness of inner body sensations and a precise sensation vocabulary are helpful steps toward restoring a sense of the body and its sensations as friend, not foe.
15. **Develop new outlets for discharging stress** and for creating a sense of well-being. The individual may have relied on re-enactment of stress and trauma, through endorphin release and dissociation, to achieve relief, numbing of the pain, and what has passed as a sense of well-being. In other words the pattern is to seek re-exposure to stress for the same effect as taking pain-killing opiates, providing a similar relief from stress. Expression rather than repression of feelings may be a new option for the person. It is important to engage in the process of

learning to discharge excess stress response in a healthy way. And that requires a clear understanding of one's own response patterns.

An individual who is aware of a tendency toward hyperarousal (fight/flight) response will discover that it is, in fact, the body's attempt to discharge excess parasympathetic load. This individual is only now becoming aware that there is a momentary DOWN reflex before the customary and conscious UP kicks in. The quick temper or incessant talking (dissociated sympathetic response) is *preceded by*, if only momentarily, a fear of rejection or sense of shame (parasympathetic response). Likewise, if a person tends toward hypoarousal (freeze) stress response, he/she will discover that it may be, in fact, the body's attempt to discharge excess sympathetic load. The sense of exhaustion or constant snacking (dissociated parasympathetic response) is *preceded by*, if only momentarily, a flash of rage or frustrated SEEKING (sympathetic response). With this knowledge, the therapist can encourage the client to look directly at the momentary initial response that needs discharging rather than at the dissociated defensive response, and to develop new outlets to accomplish that discharge.

16. **Increase self-regulation and thereby prevent the escalation of arousal** to the point of discharge through aggression or other undesirable behavior. Hyperactive defenses can take the form of uncontrollable rage or frenzy (directed at self or others), and learning to sense the physical precursors to full-blown aggressive outbursts will extinguish the conditioned response, and therefore increase the person's feeling of safety. When we help the client achieve biological completion, i.e., discharge following activation, and unlock the constriction of the nervous system, he/she can then integrate the experience for future use with triggering events, replacing the dysfunctional autonomic pattern of response.

The capacity to bring the automatic reaction into a state from which choice is possible, to internalize new cognitive information, depends on having portions of the cortex activated, which, in turn, requires a state of *attentive calm* (Castro-Alamancos & Connors, 1996). That attentive calm is often called *mindfulness* (Linehan, 1993). Some effective techniques to increase this state are increasing conscious awareness to current emotions; having the vocabulary and the self-sensitivity to identify and label emotions; creating,

acknowledging and anchoring positive emotional events; and using support and other resources to improve tolerance for stressful situations.

It must be remembered that a common symptom of childhood trauma and the resulting loss of self-regulation is attention deficit, or loss of ability to focus on relevant stimuli. Such attention deficit or neural hyperactivity is a huge obstacle to the individual's gaining the skill of *attentive calm*. Non-judgmental and unpressured encouragement from the therapist is vital to assist the client to overcome the obstacles.

**17. Increase the sense of personal safety** through increasing the sense of internal locus of control and greater reliability of external forces.

Clients find that they can gradually slow down and maintain an integrated awareness from start to finish throughout the incident, including the most invasive aspects. Then perceptually, the incident can move from seeming to be ever-present, or fixated in the future, back into the past where it belongs. Symptoms diminish. Triggers of fear, panic and anger are extinguished as continuity of self is re-established and it is experienced as truly over.

## References

- Abarbanel, A. (1995). Gates, states, rhythms, and resonances: The scientific basis of neurofeedback training. *Journal of Neurotherapy*, 1(2), 15-38.
- Aggleton, J. (1992). *The Amygdala: Neurobiological Aspects of Emotion, Memory, and Mental Dysfunction*. New York: Wiley-Liss.
- American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders, Fourth edition*. Washington, DC: American Psychiatric Association.
- Appelfeld, A. (1994). *Beyond Despair*. New York: Fromm International.
- Bauer, P. J., & Wewerka, S. S. (1995). One- to two-year-olds' recall of events: The more expressed, the more impressed. *Journal of Experimental Child Psychology*, 59, 475-496.
- Beck, A. T. (1976). *Cognitive Therapy and the Emotional Disorders*. Harmondsworth: Penguin.
- Binder, R. L., McNeil, D. E., & Goldstone, R. L. (1996). Is adaptive coping possible for adult survivors of childhood sexual abuse? *Psychiatric Services*, 47(2), 186-188.
- Blumenthal, S., Epps, R. P., Heavenrich, R., Lauer, R. M., Lieberman, E., Mirkin, B., Mitchell, S. C., Boyar-Naito, V., O'Hare, D., McFate-Smith, W., Tarazi, R. C., & Upson, D. (1977). Report of the task force on blood pressure control in children. *Pediatrics*, 59, 797.

- Bowman, E. S. (1996). Delayed memories of child abuse. Part I: An overview of research findings on forgetting, remembering, and corroborating trauma. *Dissociation*, 9, 221-231.
- Breiter, H. C., Etcoff, N. L., Whalen, P. J., Kennedy, W. A., Rauch, S. L., Buckner, R. L., et al. (1996a). Response and habituation of the human amygdala during visual processing of facial expression. *Neuron*, 17, 875-887.
- Breiter, H. C., Rauch, S. L., Kwong, K. K., Kennedy, D. N., Savage, C. R., Olivares, M. J., et al. (1996b). Functional magnetic resonance imaging of symptom provocation in obsessive-compulsive disorder. *Archives of General Psychiatry*, 53, 595-606.
- Brennan, K. A., Clark, C. L., & Shaver, P. R. (1998). Self-report measurement of adult attachment: An integrative overview. In J. A. Simpson & W. S. Rholes (Eds.), *Attachment Theory and Close Relationships*, 46-76. New York: Guilford.
- Bucci, W. (2003). Varieties of dissociative experiences: A multiple code account and a discussion of Bromberg's case of 'William.' *Psychoanalytic Psychology*, 20(3), 542-557.
- Buhl, H. S. (n.d.) Autonomic nervous system and energetic medicine: Bioenergetic and psychosomatic causes for health and illness. Available online at <http://www.erthworks.co.uk/>.
- Burgess, A. W., Hartmann, C. R., & Baker, T. (1995). Memory representations of childhood sexual abuse. *Journal of Psychosocial Nursing*, 33, 9-16.
- Burgstein, A. (1985). Posttraumatic flashbacks, dream disturbances and mental imagery. *Journal of Clinical Psychiatry*, 46, 374-378.
- Bustan, M. N., & Coker, A. L. (Mar 1994). Maternal attitude toward pregnancy and the risk of neonatal death. *American Journal of Public Health*, 84(3), 411-414.
- Campos, J., Mumme, D. L., Kermoian, R., & Campos, R. (1994). A functionalist perspective on the nature of emotion. *Monographs of the Society for Research in Child Development*, 59, 284-303.
- Carmen, E. H., Reiker, P. P., & Mills, T. (1984). Victims of violence and psychiatric illness. *American Journal of Psychiatry*, 141, 378-379.
- Carney, R., Saunders, R., Freedland, K., Stein, P., Rich, M. W., & Jaffe, A. S. (Sept 1995). Association of depression with reduced heart rate variability. *American Journal of Cardiology*, 76, 562-564.
- Carroll, R. (n.d.). PANIC, SEEKING and PLAY in Psychotherapy. Available online at <http://www.thinkbody.co.uk/papers/Panic%20Seeking%20%20play.htm>.
- Carroll, R. (2001). The autonomic nervous system: Barometer of emotional intensity and internal conflict. A lecture given for Confer, 27th March, 2001. Available online at <http://www.thinkbody.co.uk/papers/autonomic-nervous-system.htm>.
- Castro-Alamancos, M. A., & Connors, B. W. (1996). Short-term plasticity of a thalamocortical pathway dynamically modulated by behavioral state. *Science*, 272, 274-276.
- Coffey, P., Leitenberg, H., Henning, K., Turner, T., & Bennett, R. T. (1996). The relation between methods of coping during adulthood with a history of childhood sexual abuse and current psychological adjustment. *Journal of Consulting and Clinical Psychology*, 64, 1090-1093.
- Cohen, H., Kotler, M., Matar, M., Kaplan, Z., Loewenthal, U., Miodownik, H., & Cassuto, Y. (1998). Analysis of heart rate variability in posttraumatic stress disorder patients in response to a trauma-related reminder. *Biological Psychiatry*, 44(10), 1054-1059.
- Cole, P. M., & Putnam, F. W. (1992). Effect of incest on self and social functioning: Developmental psychopathology perspective. *Journal of Consulting and Clinical Psychology*, 60(2), 174-184.

- Darwin, C. (1872). *The Expression of the Emotions in Man and Animals*. New York: D. Appleton.
- Davies, J. M., & Frawley, M. G. (1992). Dissociative processes and transference/ countertransference paradigms in the psychoanalytically oriented treatment of adult survivors of childhood sexual abuse. *Psychoanalytic Dialogues*, 2, 5-36.
- DiPietro, J. A., Novak, M. F. S. X., Costigan, K. A., Atella, L. D., & Reusing, S. P. (May 2006). Maternal psychological distress during pregnancy in relation to child development at age two. *Child Development*, 77(3), 573-587.
- Dixon, A. K. (1998). Ethological strategies for defence in animals and humans: Their role in some psychiatric disorders. *British Journal of Medical Psychology*, 71, 417-445.
- Drevets, W. C., Price, J. L., Bardgett, M. E., Reich, T., Todd, R. D., & Raichle, M. E. (2002). Glucose metabolism in the amygdala in depression: Relationship to diagnostic subtype and plasma cortisol levels. *Pharmacology Biochemistry and Behavior*, 71, 431-447.
- Ehlers, A., & Clark, D. M. (2000). A cognitive model of persistent PTSD. *Behaviour Research and Therapy*, 38, 319-345.
- Ehling, T., Nijenhuis, E. R. S., & Krikke, A. (2003). Volume of discrete brain structures in florid and recovered DID, DDNOS, and healthy controls. *Proceedings of the 20th International Fall Conference of the International Society for the Study of Dissociation*, Chicago, November 2-4.
- Ekman, P. (1992). An argument for basic emotions. *Cognition and Emotion*, 6, 169-200.
- Emerson, W. (2002). Somatotropic therapy. *Journal of Heart-Centered Therapies*, 5(2), 65-90.
- Erickson, K., Drevets, W., & Schulkin, J. (2003). Glucocorticoid regulation of diverse cognitive functions in normal and pathological emotional states. *Neuroscience & Biobehavioral Reviews*, 27, 233-246.
- Farkas, B. (2004). Etiology and pathogenesis of PTSD in children and adolescents. In R. R. Silva (Ed.), *Posttraumatic Stress Disorders in Children and Adolescents [Handbook]*, 123-140. New York: W. W. Norton.
- Feldmar, A. (1979). The embryology of consciousness: What is a normal pregnancy? In D. Mall & W. Watts (Eds.), *The Psychological Aspects of Abortion*, 15-24. University Publications of America.
- Field, T., Diego, M., & Hernandez-Reif, M. (Sept 2006). Prenatal depression effects on the fetus and newborn: A review. *Infant Behavior & Development*, 29(3), 445-455.
- Field, T., Diego, M., Hernandez-Reif, M., Dieter, J., Schanberg, S., Kühn, C., Yando, R., & Bendel, D. (2001). Depressed, withdrawn and intrusive mothers' effects on their fetuses and neonates. *Infant Behavior & Development*, 24, 27-39.
- Fish Murray, C. C., Koby, E. V., & van der Kolk, B. A. (1987). Evolving ideas: The effect of abuse on children's thought. In B. A. van der Kolk (Ed.), *Psychological Trauma*. Washington, DC: American Psychiatric Press.
- Fonagy, P., Target, M., & Gergely, G. (2000). Attachment and borderline personality disorder: A theory and some evidence. *Psychiatric Clinic of North America*, 23, 103-123.
- Fraiberg, S. (1982). Pathological defenses in infancy. *Psychoanalytic Quarterly*, 51, 612-635.
- Friedman, B. H., & Thayer, J. F. (Nov 1998a). Anxiety and autonomic flexibility: A cardiovascular approach: Erratum. *Biological Psychology*, 49(3), 303-323.
- Friedman, B. H., & Thayer, J. F. (Jan 1998b). Autonomic balance revisited: Panic anxiety and heart rate variability. *Journal of Psychosomatic Research*, 44(1), 133-151.

- Gelinas, D. J. (1983). The persistent negative effects of incest. *Psychiatry*, 46, 312-332.
- Giannakouloupoulos, X., Fisk, N., Glover, V., Kourtis, P., & Sepulveda, W. (1994). Fetal plasma cortisol and B-endorphins response to intrauterine needling. *Lancet*, 344, 77-81.
- Gilbert, P., & Allan, S. (1998). The role of defeat and entrapment (arrested flight) in depression: An exploration of an evolutionary view. *Psychological Medicine*, 28, 585-598.
- Gilbertson, M. W., Shenton, M. E., Ciszewski, A., Kasai, K., Lasko, N. B., Orr, S. P., & Pitman, R. K. (2002). Smaller hippocampal volume predicts pathologic vulnerability to psychological trauma. *Nature Neuroscience*, 5(11), 1242-1247.
- Goleman, D. (Feb 4, 2003). Behavior; Finding Happiness: Cajole Your Brain to Lean to the Left. *New York Times*.
- Greatrex, T. (2002). Projective Identification: How does it work? *Neuro-Psychoanalysis*, 4(2), 187-197.
- Green, A. H. (1980). *Child Maltreatment*. New York: Jason Aronson.
- Gunderson, J. G. (1996). The borderline patient's intolerance of aloneness: Insecure attachments and therapist availability. *American Journal of Psychiatry*, 153, 752-758.
- Gur, R. C., Mozley, L. H., Mozley, P. D., Resnick, S. M., Karp, J. S., Alavi, A., Arnold, S. E., & Gur, R. E. (1995). Sex differences in regional cerebral glucose metabolism during a resting state. *Science*, 267, 528-531.
- Gurrera, R. J. (1999). Sympathoadrenal hyperactivity and the etiology of neuroleptic malignant syndrome. *American Journal of Psychiatry*, 156, 169-180.
- Halgren, E., Walter, R. D., Cherlow, D. G., & Crandall, P. H. (1978). Mental phenomena evoked by electrical stimulation of the human hippocampal formation and amygdala. *Brain*, 101, 83-117.
- Hanoch, Y., & Vitouch, O. (2004). When Less is More: Information, Emotional Arousal and the Ecological Reframing of the Yerkes-Dodson Law. *Theory & Psychology*, 14(4), 427-452.
- Hartman, D., & Zimberoff, D. (2004). Existential resistance to life: Ambivalence, avoidance & control. *Journal of Heart-Centered Therapies*, 7(1), 3-63.
- Harvard Mental Health Letter. (June 2005). The biology of child maltreatment: How abuse and neglect of children leave their mark on the brain. Harvard Health Publications, Harvard Medical School.
- Harvey, A. G., & Bryant, R. A. (1999). Dissociative symptoms in acute stress disorder. *Journal of Traumatic Stress*, 12, 673-680.
- Herpertz, S. C., Dietrich, T. M., Wenning, B., Krings, T., Erberich, S. G., Willmes, K., Thron, A., & Sass, H. (August 15, 2001). Evidence of abnormal amygdala functioning in borderline personality disorder: A functional MRI study. *Biological Psychiatry*, 50(4), 292-298.
- Hoagwood, K. (1990). Blame and adjustment among women sexually abused as children. *Women & Therapy*, 9(4), 89-110.
- Hon, E., & Lee, S. (1963). Electronic evaluation of fetal heart rate. *American Journal of Obstetrics and Gynecology*, 87, 814-826.
- Horsten, M., Ericson, M., Perski, A., Wamala, S., Schenck-Gustafsson, K., & Orth-Gomer, K. (1999). Psychosocial factors and heart rate variability in healthy women. *Psychosomatic Medicine*, 61(1), 49-57.



- Huizink, A. C., Mulder, E. J. H., & Buitelaar, J. K. (2004). Prenatal stress and risk for psychopathology: Specific effects or induction of general susceptibility? *Psychological Bulletin*, 130(1), 115–142.
- Huttunen, M. O., Machon, R. A., & Mednick, S. A. (1994). Prenatal factors in the pathogenesis of schizophrenia. *British Journal of Psychiatry*, 23(Suppl.), 15–19.
- Institute for Applied Meditation. Available online at <http://www.appliedmeditation.org/tests/HRV.shtml>.
- Jacobs, W. J., Laurance, H. E., Thomas, K. G. F., Luzcak, S. E., & Nadel, L. (1996). On the veracity and variability of traumatic memory. *Traumatology*, 2(2).
- Jaffe, P., Wolfe, D., Wilson, S. K., et al. (1986). Family violence and child adjustment: A comparative analysis of girls' and boys' behavioral symptoms. *American Journal of Psychiatry*, 143, 74–77.
- Jung, C. G. (1988). *Nietzsche's Zarathustra Vol I*, James L. Jarret (Ed.). Princeton: Princeton University Press.
- Kalsched, D. E. (April 2003). Daimonic elements in early trauma. *Journal of Analytical Psychology*, 48(2), 145–169.
- Kawachi, I., Sparrow, D., Vokonas, P. S., & Weiss, S. T. (1995). Decreased heart rate variability in men with phobic anxiety. *The American Journal of Cardiology*, 75, 882–885.
- Keleman, S. (1989). *Patterns of Distress: Emotional Insults and Human Form*. Berkeley, CA: Center Press.
- Keleman, S. (n.d.). Depression and Panic: An interview with Stanley Keleman by Terrence MacClure. Available at <http://home.wxs.nl/~form.psy/panic.htm>.
- Kosfeld, M., Heinrichs, M., Zak, P. J., Fischbacher, U., & Fehr, E. (June 2005). Oxytocin increases trust in humans. *Nature*, 435(7042), 673–676.
- Kosten, T. R., & Krystal, J. (1988). Biological mechanisms in posttraumatic stress disorder: Relevance for substance abuse. In M. Galanter (Ed.), *Recent Developments in Alcoholism*, 49–68. New York: Plenum.
- Krittayaphong, R., Cascio, W., Light, K., Sheffield, D., Golden, R., Finkel, J., Glekas, G., Koch, G., & Sheps, D. (1997). Heart rate variability in patients with coronary artery disease: Differences in patients with higher and lower depression scores. *Psychosomatic Medicine*, 59, 231–235.
- Krystal, J. H., Karper, L. P., Seibyl, J. P., Freeman, G. K., Delaney, R., Bremner, J. D., Heninger, G. R., Bowers, M. B., & Charney, D. S. (1994). Subanesthetic effects of the noncompetitive NMDA antagonist, ketamine, in humans: Psychotomimetic, perceptual, cognitive, and neuroendocrine responses. *Archives of General Psychiatry*, 51, 199–214.
- LeDoux, J. E. (1996). *The Emotional Brain*. New York: Simon & Schuster.
- LeDoux, J. E. (1999). Psychoanalytic theory: Clues from the brain. *Neuro-Psychoanalysis*, 1, 44–49.
- Levine, P. (1997). *Waking the Tiger*, Berkeley, CA: North Atlantic Books.
- Linehan, M. M. (1993). *Cognitive Behavioral Therapy of Borderline Personality Disorder*. New York: Guilford Press.
- Makino, S., Gold, P. W., & Schulkin, J. (1994). Corticosterone effects on corticotropin-releasing hormone mRNA in the central nucleus of the amygdala and the parvocellular region of the paraventricular nucleus of the hypothalamus. *Brain Research*, 640, 105–112.

- Malatesta, C. Z., Culver, C., Tesman, J. R., & Shepard, B. (1989). The development of emotion expression during the first two years of life. *Monographs of the Society for Research in Child Development*, 54(1-2), 1-104.
- Mann, S. C., Caroff, S. N., Keck, P. E., & Lazarus, A. (2003). *Neuroleptic Malignant Syndrome and Related Conditions* (2nd ed.). Arlington, VA: American Psychiatric Publishing.
- Markowitsch, H. J. (1999). Functional neuroimaging correlates of functional amnesia. *Memory*, 7, 561-583.
- Markowitsch, H. J., Calabrese, P., Fink, G. R., Durwen, H. F., Kessler, J., Harting, C., König, M., Mirzaian, E. B., Heiss, W. D., Heuser, L., & Gehlen, W. (1997). Impaired episodic memory retrieval in a case of probably psychogenic amnesia. *Psychiatry Research: Neuroimaging Section*, 74, 119-126.
- Markowitsch, H. J., Fink, G. R., Thone, A., Kessler, J., & Heiss, W. D. (1997). A PET study of persistent psychogenic amnesia covering the whole life span. *Cognitive Neuropsychiatry*, 2, 135-158.
- Markowitsch, H. J., Kessler, J., Van der Ven, C., Weber-Luxenburger, G., Albers, M., & Heiss, W. D. (1998). Psychic trauma causing grossly reduced brain metabolism and cognitive deterioration. *Neuropsychologica*, 36, 77-82.
- Markowitsch, H. J., Kessler, J., Weber-Luxenburger, G., Van der Ven, C., Albers, M., & Heiss, W. D. (2000). Neuroimaging and behavioral correlates of recovery from mnestic block syndrome and other cognitive deteriorations. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, 13, 60-66.
- Mason, J., Giller, E., Kosten, T., Ostroff, R., & Podd, L. (1986). Urinary free cortisol levels in posttraumatic stress disorder patients. *Journal of Nervous and Mental Disease*, 174(3), 145-149.
- McCarty, R., Atkinson, M., Tiller, W., Rein, G., & Watkins, A. (1995). The effects of emotions on short term power spectrum analysis of heart rate variability. *The American Journal of Cardiology*, 76(14), 1089-1092.
- McEwen, B. S., Gould, E. A., & Sakai, R. R. (1992). The vulnerability of the hippocampus to protective and destructive effects of glucocorticoids in relation to stress. *British Journal of Psychiatry*, 160, 18-24.
- Melges, F. T., & Swartz, M. S. (1989). Oscillations of attachment in borderline personality disorder. *American Journal of Psychiatry*, 146, 1115-1120.
- Middleton, H. (1990). Cardiovascular dystonia in recovered panic patients. *Journal of Affective Disorders*, 19(4), 229-236.
- Mikulincer, M., & Florian, V. (1998). The relationship between adult attachment styles and emotional and cognitive reactions to stressful events. In J. A. Simpson & W. S. Rholes (Eds.), *Attachment Theory and Close Relationships*, 143-165. New York: Guilford.
- Miller, R. (1991). *Cortico-Hippocampal Interplay and the Representation of Contexts in the Brain*. Springer-Verlag.
- Minzenberg, M. J., Poole, J. H., & Vinogradov, S. (May 2006). Adult Social Attachment Disturbance is related to childhood maltreatment and current symptoms in Borderline Personality Disorder. *The Journal of Nervous and Mental Disease*, 194(5), 341-348.
- Morgan, T., & Cummings, A. L. (1999). Change experienced during group therapy by female survivors of childhood sexual abuse. *Journal of Consulting and Clinical Psychology*, 67, 28-36.
- Moskowitz, A. K. (2004). 'Scared Stiff': Catatonia as an evolutionary-based fear response. *Psychological Review*, 111(4), 984-1002.

- Myers, C. S. (1940). *Shell Shock in France 1914-1918*. Cambridge: Cambridge University Press.
- Nadel, L., & Jacobs, W. J. (1996). The role of the hippocampus in PTSD, panic, and phobia. In N. Kato (Ed.), *Hippocampus: Functions and Clinical Relevance*. Amsterdam: Elsevier Science.
- Nakagawa, M., Saikawa, T., & Ito, M. (1994). Progressive reduction of heart rate variability with eventual sudden death in two patients. *British Heart Journal*, 71(1), 87-88.
- NIH publication. (1997). *The Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure*.
- Nijenhuis, E. R. S., & Van der Hart, O. (1999). Forgetting and re-experiencing trauma: From anesthesia to pain. In J. Goodwin & R. Attias (Eds.), *Splintered Reflections: Images of the Body in Trauma*, 39-66. New York: Basic Books.
- Nijenhuis, E. R. S., Quak, J., Reinders, S., Korf, J., Vos, H., & Marinkelle, A. B. (1999). *Identity dependent processing of traumatic memories in Dissociative Identity Disorder: Converging regional blood flow, physiological and psychological evidence*. Paper presented at the Proceedings of the 6th European Conference on Traumatic Stress: Psychotraumatology, clinical practice, and human rights. Istanbul, Turkey.
- Nijenhuis, E. R. S., Van der Hart, O., & Steele, K. (2004). Trauma-related structural dissociation of the personality. Trauma Information Pages website, January 2004. Web URL: <http://www.trauma-pages.com/nijenhuis-2004.htm>.
- Offerhaus, R. E. (1980). Heart rate variability in psychiatry. In R. J. Kitney & O. Rompelman (Eds.), *The Study of Heart Rate Variability*, 225-238. Oxford: Oxford University Press.
- Ogden, P., & Minton, K. (2000). Sensorimotor Psychotherapy: One method for processing traumatic memory. *Traumatology*, 6(3). Available online at <http://www.fsu.edu/~trauma/v6i3/v6i3a3.html>.
- Othmer, S. (n.d.). The Attachment Conference. EEG Institute Newsletter, 5(10). Available online at [http://www.eeginfo.com/newsletter/vol5\\_issue10\\_newsletter.htm](http://www.eeginfo.com/newsletter/vol5_issue10_newsletter.htm).
- Panksepp, J. (1982). Towards a general psychological theory of emotion. *Behavioral and Brain Sciences*, 5, 407-467.
- Panksepp, J. (1998). *Affective Neuroscience: The Foundations of Human and Animal Emotions*. Oxford: Oxford University Press.
- Pavin, D., Le Breton, H., & Daubert, C. (1997). Human stress cardiomyopathy mimicking acute myocardial syndrome. *Heart*, 78, 509-511.
- Pavlidis, C., & Winson J. (1989). Influences of hippocampal place cell firing in the awake state on the activity of these cells during subsequent sleep episodes. *Journal of Neuroscience*, 9, 2907-2918.
- Perry, B. D. (1999). The memory of states: How the brain stores and retrieves traumatic experience. In J. Goodwin & R. Attias (Eds.), *Splintered Reflections: Images of the Body in Treatment* 9-38. New York: Basic Books.
- Perry, B. D. (2001). The neurodevelopmental impact of violence in childhood. In D. Schetky and E. P. Benedek (Eds.), *Textbook of Child and Adolescent Forensic Psychiatry*, 221-238. Washington, DC: American Psychiatric Press.
- Perry, B. D., Pollard, R. A., Baker, W. L., Sturges, C., Vigilante, D., & Blakely, T. L. (1995). Continuous heartrate monitoring in maltreated children [Abstract]. Annual Meeting of the American Academy of Child and Adolescent Psychiatry, New Research.

- Perry, B. D., Pollard, R., Blakely, T., Baker, W., Vigilante, D. (1995). Childhood trauma, the neurobiology of adaptation and 'use-dependent' development of the brain: How 'states' become 'traits'. *Infant Mental Health Journal*, 16(4), 271-291.
- Porges, S. W. (1995). Orienting in a defensive world: Mammalian modifications of our evolutionary heritage. A Polyvagal Theory. *Psychophysiology*, 32, 301-318.
- Porges, S. W. (2003). The Polyvagal Theory: Phylogenetic contributions to social behavior. *Physiology & Behavior*, 79, 503-513.
- Porges, S. W. (May 2004). Neuroception: A subconscious system for detecting threats and safety. *Zero to Three*.
- Putnam, F. W. (Fall 2004). Stuck in the past. *Psychiatry: Interpersonal and Biological Processes*, 67(3), 235-238.
- Pynoos, R. S. (1993). Traumatic stress and developmental psychopathology in children and adolescents. *American Psychiatric Press Review of Psychiatry*, 12.
- Rauch, S. L., van der Kolk, B. A., Fisler, R. E., Alpert, N. M., Orr, S. P., Savage, C. R., Fischman, A. J., Jenike, M. A., & Pitman, R. K. (1996). A symptom provocation study of posttraumatic stress disorder using positron emission tomography and script-driven imagery. *Archives of General Psychiatry*, 53, 380-387.
- Reich, W. (1967). *The Discovery of the Orgone, Vol. 1: The Function of the Orgasm*. New York: Farrar, Straus & Giroux.
- Reinders, A. A. T. S., Nijenhuis, E. R. S., Paans, A. M. J., Korf, J., Willemsen, A. T. M., & Den Boer, J. A. (2003). One brain, two selves. *NeuroImage*, 20, 2119-2125.
- Rosebush, P. I., & Mazurek, M. F. (1999). Catatonia: Re-awakening to a forgotten disorder. *Movement Disorders*, 14, 395-397.
- Ross, E. D., Homan, R. W. & Buck, R. (1994) Differential hemispheric lateralization of primary and social emotions. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, 7, 1-19.
- Rothschild, B. (1996). *Applying the Brakes: Theory and Tools for Understanding, Slowing Down and Reducing Autonomic Nervous System Activation in Traumatized Clients*. Paper presented at the Tenth Scandinavian Conference for Psychotherapists working with Traumatized Refugees, May 24-26, 1996, Finland.
- Rothschild, B. (1997). Slowing down and controlling traumatic hyperarousal. In L. Vanderberger (Ed.), *The Many Faces of Trauma, International Perspectives* (in press).
- Rothschild, B. (Feb 1998). Post-Traumatic Stress Disorder: Identification and Diagnosis. *The Swiss Journal of Social Work*.
- Russell, D. (1986). *The Secret Trauma*. New York: Basic Books.
- Saper, C. B., Chou, T. C., & Elmquist, J. K. (2002). The need to feed: Homeostatic and hedonic control of eating. *Neuron*, 36, 199-211.
- Scaer, R. C. (2001). The neurophysiology of dissociation & chronic disease. *Applied Psychophysiology and Biofeedback*, 26(1), 73-91. Available online at <http://www.trauma-pages.com/a/scaer-2001.php>.
- Scaer, R. C. (2005). *The Trauma Spectrum: Hidden Wounds and Human Resiliency*. New York: W. W. Norton.
- Schulkin, J. (1999). *The Neuroendocrine Regulation of Behavior*. New York: Cambridge University Press.

- Schulkin, J., Gold, P. W., & McEwen, B. S. (1998). Induction of corticotropin-releasing hormone gene expression by glucocorticoids: Implication for understanding the states of fear and anxiety and allostatic load. *Psychoneuroendocrinology*, 23, 219–243.
- Schultz, W. (2002). Getting formal with dopamine and reward. *Neuron*, 36, 241–263.
- Seligman, M. (1975). *Helplessness: On Depression, Development and Death*. San Francisco: W.H. Freeman and Co.
- Sidoli, M., & Blakemore, P. (2000). *When the Body Speaks: The Archetypes in the Body*. London: Routledge.
- Siegel, D. (1999). *The Developing Mind: Toward a Neurobiology of Interpersonal Experience*. New York: Guilford.
- Silbert, M. D., & Pines, A. M. (1981). Sexual child abuse as an antecedent to prostitution. *Child Abuse and Neglect*, 5, 407–411.
- Sloan, R. P., Shapiro, P. A., Bigger, T. Jr., Bagiella, E., Steinman, R. C., & Gorman, J. M. (1994). Cardiac autonomic control and hostility in healthy subjects. *The American Journal of Cardiology*, 74, 298–300.
- Solursh, L. (1987). *Combat addiction: Implications in symptom maintenance and treatment planning*. Paper Presented at the Third Annual Meeting of the Society for Traumatic Stress Studies, Baltimore, Maryland.
- Spitz, R. (1961). Some early proto-types of ego defense. *Journal of American Psychoanalytic Association*, 9, 626–651.
- Sprick, U. (Jan 1995). Functional aspects of the involvement of the hippocampus in behavior and memory functions. *Behavioral Brain Research*, 66(1-2), 61–64.
- Squire, L.R. (1992). Memory and the hippocampus: A synthesis from findings with rats, monkeys, and humans. *Psychological Review*, 99(2), 195–231.
- Steriade, M., Gloor, P., Llinas, R. R., Lopes de Silva, F. H., & Mesulam, M. M. (1990). Basic mechanisms of cerebral rhythmic activities. *Electroencephalography and Clinical Neurophysiology*, 76, 481–508.
- Sterling, P. (2004). Principles of allostasis: Optimal design, predictive regulation, pathophysiology and rational therapeutics. In J. Schulkin (Ed.), *Allostasis, Homeostasis, and the Costs of Adaptation*. Cambridge University Press.
- Taylor, M. A., & Fink, M. (2003). Catatonia in psychiatric classification: A home of its own. *American Journal of Psychiatry*, 160, 1233–1241.
- Taylor, S. E. (2002). *The Tending Instinct: Women, Men, and the Biology of Our Relationships*. New York: Henry Holt and Company.
- Thomas, K. G. F., Lurance, H. E., Jacobs, W. J., & Nadel, L. (1995). Memory for traumatic events: Formulating hypotheses and critical experiments. *Traumatology*, 1(2).
- Torgersen, S. (1986). Childhood and family characteristics in panic and generalized anxiety disorders. *American Journal of Psychiatry*, 143, 630–632.
- Tsai, M., Feldman-Summers, S., & Edgar, M. (1979). Childhood molestation: Variables related to differential impacts on psychosexual functioning in adult women. *Journal of Abnormal Psychology*, 88, 407–417.

- Uhde, T. W., & Tancer, M. (1988). Chemical models of panic: A review and critique. In P. Tyrer (Ed.), *Psychopharmacology of Anxiety*, 110-131. New York: Oxford University Press.
- Van den Bergh, B. R. H., & Marcoen, A. (July 2004). High antenatal maternal anxiety is related to ADHD symptoms, externalizing problems, and anxiety in 8- and 9-year-olds. *Child Development*, 75(4), 1085.
- Van der Hart, O. (2000). *Psychic Trauma: The Disintegrating Effects of Overwhelming Experience on Mind and Body*. Melbourne: University of Melbourne.
- Van der Hart, O., & Nijenhuis, E. R. S. (2001a). Loss and recovery of different memory types in generalized dissociative amnesia. *Australian and New Zealand Journal of Psychiatry*, 35(5), 589-600.
- van der Hart, O., & Nijenhuis, E. R. S. (2001b). Generalized dissociative amnesia: Episodic, semantic and procedural memories lost and found. *Australian and New Zealand Journal of Psychiatry*, 35(5), 589-600.
- Van der Hart, O., & Op den Velde, W. (1995). Posttraumatische stoornissen [Posttraumatic disorders]. In O. Van der Hart (Ed.), *Trauma, dissociatie en hypnose [Trauma, dissociation and hypnosis]*, 103-145. Lisse: Swets & Zeitlinger.
- van der Kolk, B. A. (1987). *Psychological Trauma*. Washington, DC: American Psychiatric Press.
- van der Kolk, B. A. (1988). The trauma spectrum: The interaction of biological and social events in the genesis of the trauma response. *Journal of Traumatic Stress*, 1, 273-290.
- van der Kolk, B. A. (June 1989). The compulsion to repeat the trauma: Re-enactment, revictimization, and masochism. *Psychiatric Clinics of North America*, 12(2), 389-411.
- van der Kolk, B. A. (1994). The body keeps the score: Memory and the evolving psychobiology of post-traumatic stress. *Harvard Psychiatric Review*, Vol. 1.
- van der Kolk, B. A. (1996). The complexity of adaptation to trauma: Self-regulation, stimulus discrimination, and characterological development. In B. A. van der Kolk, A. C. McFarlane, & L. Weisaeth (Eds.), *Traumatic stress. The Effects of Overwhelming Experience on Mind, Body, and Society*, 182-213. New York: Guilford Press.
- van der Kolk, B. A. (2001). The assessment and treatment of complex PTSD. In R. Yehuda (Ed.), *Traumatic Stress*. American Psychiatric Press.
- Van der Kolk, B. A., & Fisler, R. (1995). Dissociation and the fragmentary nature of traumatic memories: Overview and exploratory study. *Journal of Traumatic Stress*, 8, 505-525.
- van der Kolk, B. A., Herman, J., & Perry, J. (1988). Childhood Trauma and Self Destructive Behavior in Adulthood. Unpublished data.
- Vanderwolf, C. H., & Robinson, T. E. (1981). Reticulo-cortical activity and behavior: A critique of the arousal theory and a new synthesis. *The Behavioral and Brain Sciences*, 4, 459-514.
- Vermetten, E., Vythilingam, M., Southwick, S. M., Charney, D. S., & Bremner, J. D. (2003). Long-term treatment with paroxetine increases verbal declarative memory and hippocampal volume in posttraumatic stress disorder. *Biological Psychiatry*, 54, 693-702.
- Vertes, R. P. (1982). Brain stem generation of the hippocampal EEG. *Progress in Neurobiology*, 19, 159-186.
- Vickers, B. (2005). Cognitive model of the maintenance and treatment of Post-traumatic Stress Disorder applied to children and adolescents. *Clinical Child Psychology and Psychiatry*, 10(2), 217-234.

- Washburn, M. (2003). *Embodied Spirituality in a Sacred World*. Albany, NY: SUNY Press.
- Watkins, L., Grossman, P., Krishnan, R., & Blumenthal, J. (1999). Anxiety reduces baroreflex cardiac control in older adults with major depression. *Psychosomatic Medicine*, 61, 334-340.
- Wheeler, M. A., Stuss, D. T., & Tulving, E. (May 1997). Toward a theory of episodic memory: The frontal lobes and autoegetic consciousness. *Psychological Bulletin*, 121(3), 331-354.
- Wilbarger, P., & Wilbarger, J. (1997). *Sensory Defensiveness and Related Social/ Emotional and Neurological Problems*. Van Nuys, CA: Wilbarger.
- Wilson, M. A., & McNaughton, B. L. (1994). Reactivation of hippocampal ensemble memories during sleep. *Science*, 265, 676-679.
- Winnicott, D. W. (1945). Primitive emotional development. *International Journal of Psychoanalysis*, 26, 137-143.
- Winnicott, D. W. (1965). The theory of the parent-infant relationship. In *The Maturation Processes and the Facilitating Environment*, 37-55. New York: International Universities Press.
- Winson, J. (1972). Interspecies differences in the occurrence of theta. *Behavioral Biology*, 7, 479-487.
- Winson, J. (Nov 1990). The meaning of dreams. *Scientific American*, 86-96.
- Winson, J. (1993). The biology and function of rapid eye movement sleep. *Current Opinions in Neurobiology*, 3, 243-248.
- Wise, R. A. (2003). Brain reward circuitry: Insight from unsensed incentives. *Neuron*, 36, 229-240.
- Wittstein, I. S., Thiemann, D. R., Lima, J. A. C., Baughman, K. L., Schulman, S. P., Gerstenblith, G., Wu, K. C., Rade, J. J., Bivalacqua, T. J., & Champion, H. C. (Feb 10 2005). Neurohumoral Features of Myocardial Stunning Due to Sudden Emotional Stress. *New England Journal of Medicine*, 352(6), 539-548.
- Woodman, M. (Winter 1993). Stepping over the threshold: Into the black hole at the center of Self. *Noetic Sciences Review*, 28, 10-15.
- Wyatt, G. E., & Newcomb, M. (1990). Internal and external mediators of women's sexual abuse in childhood. *Journal of Counseling and Clinical Psychology*, 58, 758-767.
- Yehuda, R., Southwick, S., Nussbaum, G., et al. (1990). Low urinary cortisol excretion in patients with posttraumatic stress disorder. *Journal of Nervous and Mental Diseases*, 178, 366-369.
- Yeragani, V. K., Balon, R., Pohl, R., Ramesh, C., Glitz, D., Weinberg, P., & Merlos, B. (1990). Decreased R-R variance in panic disorder patients. *Acta Psychiatrica Scandinavica*, 81, 554-559.
- Yeragani, V. K., Pohl, R., Berger, R., Balon, R., Ramesh, C., Glitz, D., Srinivasan, K., & Weinberg, P. (1993). Decreased heart rate variability in panic disorder patients: A study of power-spectral analysis of heart rate. *Psychiatry Research*, 46, 89-103.
- Zajonc, R. B. (1980). Feeling and thinking: Preferences need no inferences. *American Psychologist*, 2, 151-175.
- Zeana, C. H. (2000). Disturbances of attachment in young children adopted from institutions. *Journal of Developmental and Behavioral Pediatrics*, 21(3), 230-236.