

Healing Traumatic Reenactment: Psyche's Return from Soma's Underworld

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Each October a young woman—I'll call her Beth—descended into the underworld of tumultuous relationship loss. When her abusive father died during this same month in her early childhood, she feared her hatred had killed him. As an adult, year after year the defense against her abandonment terror was recapitulated as she provoked breakups with boyfriends on the anniversary of her father's death.

Freud (1914/1959) first conceptualized such behavioral reenactments as “repetition compulsion,” observing that they sometimes substitute for remembering. He believed they were caused by unconscious conflict paired with repression. According to Levine (1996), traumatic reenactment (TR) is also the psychesoma's strategy for completing the unfinished fight-flight/freeze survival cycle. Until this survival cycle is resolved, traumatized patients suffer TR due to potentially permanent impairment in their stress coping mechanisms, which are centrally involved in the capacity for self-regulation (Schoore, 2002).

For Beth this survival cycle had been triggered from earliest infancy by recurring attachment disruptions, which even in moderate degrees can have lasting traumatic impact. Unwanted and physically abused by Father, she was inconsistently soothed (regulated) and contained by Mother, who had been abandoned and sexually abused during her own childhood and later became psychotic. In Beth's adulthood, despite a positive work experience and social life, she experienced TR in the form of repetitions, flashbacks, intrusive terrors, and somatization (including multiple food, airborne, and environmental sensitivities).

This article examines psychodynamic and neurobiological origins of TR, interweaving somatic and analytic treatment approaches for therapists challenged by patients with chronically impaired self-regulation. Incorporating updated neuropsychanalytic models and advances in traumatology, this integrative approach acknowledges that early attachment trauma induces a neurobiological susceptibility to TR, while also postulating specific methods for helping dysregulated patients discover an emerging psychobiological sense of well being.

Neurobiology of Traumatic Reenactment

Body, mind, and brain are inextricably linked via the central and autonomic nervous systems (CNS, ANS). A complex chemical network mediates communication within and between these systems, including all the stress response systems and organ systems of the body. Below the threshold of conscious awareness, these regulatory functions are manifestations of the somatic unconscious.

During the earliest months of life when infants become upset over unmet needs, and caregivers are unable to soothe them sufficiently, the effect is traumatic. This is so because two extremes of ANS dysregulation are induced in these infants: 1) frantic distress during sympathetic hyperarousal, followed by 2) helplessness, then collapsed hopelessness during parasympathetic hypoarousal-dissociation. Such dysregulated states trigger a cascade of stress hormones, which during infancy severely reduces synaptogenesis in the developing right brain and adversely affects immune and endocrine function (Schoore, 2002).

Even when caregivers have good intentions, they may have limited capacity for sensitive attunement to their infants' needs for a variety of reasons, including illness (physical or psychological), marital discord, death of a loved one, substance abuse, the caregiver's own unresolved early trauma, as well as the unavoidable separation of infant and caregiver or their personalities being mismatched. Loss of secure attachment—through deprivation and/or dissonance resulting from failed interactive regulation—is the outcome. Depending on the severity of disturbance to a child's nervous system, the consequence may

constitute abuse and neglect. Ambient and cumulative, developmental trauma such as this contributes to a proclivity for TR by provoking potentially permanent structural and functional deficits in the stress-response systems of the body-mind-brain.

According to van der Kolk (1996), chronic stress such as early attachment trauma does not exist on a continuum with the normal stress response. Instead, it is a distinct phenomenon that leads to abnormalities in four stress-response systems: psychophysiological, neurohormonal, neuroanatomical, and neuroimmune. Beth manifested abnormalities in all four systems; space limitations permit discussion of only two. First, psychophysiological “sensitization”—heightened stress reactivity—results when chronic ANS stress impairs cortical capacity for discrimination. Beth’s sensitization was expressed via exaggerated startle responses, heightened emotionality, extreme sensitivity to sound, and sleep difficulties. Secondly, neuroimmune abnormalities surfaced with Beth’s sinus allergies at age 7. When pain from father’s whippings led to dissociation, Beth lost the capacity to cry. Later, via the somatic unconscious, hay fever triggered allergic “tears” in the form of a continually runny nose and drippy eyes.

In clinical work with adults the counterbalancing forces of the autonomic nervous system (ANS) are visible amid several familiar emotions. Excitement, terror, and rage are expressions of ANS sympathetic hyperarousal, which involves increased heart rate, agitation, muscular tension, and difficulty breathing. In TR, these emotional states often trigger fight–flight defensive reflexes, which may take the form of hyperresponsiveness and hypervigilance. Following Janet, Jung (1928/1960) recognized that such overwhelming affects alter the psychesoma’s biology, precipitating the protective mechanism of dissociation. Depression, deadness, disconnection, alexithymia, and exhaustion express ANS parasympathetic/dissociation. In TR, these states often trigger immobility/freeze responses, such as feeling dazed or out of the body. Dissociation may appear in its mildest form as “spaciness,” while its more extreme forms include Dissociative Identity Disorder and Borderline Personality Disorder.

Undischarged fight-flight/freeze responses are analogous to depressing a car’s accelerator and brake pedals simultaneously. These contradictory responses remain in the brain as unconscious (implicit) procedural memory and in the body as constriction. Fight-flight/freeze responses trigger behavioral reenactment when held in striated muscles and somatic reenactment when held in smooth muscles (van der Kolk, 1996). These survival cycle responses must be completed and released from the body-mind-brain if TR is to be healed.

Though current research will expand the information offered here, the exquisite complexity of human neurobiology and its regulation remains undeniable. It is our animal instincts gone awry that serve as unconscious somatic precipitants of TR.

Dissociation and Memory in Traumatic Reenactment

Dissociation refers here to a fragmentation of experience whereby traumatic elements—thoughts, emotions, memories, and body states—remain outside a unitary sense of self. Predominantly a right brain subcortical regulatory process (Schore, 1994), dissociation is during infancy a primary protective mechanism against unbearable stress, since more complex self-regulatory systems have not fully developed. According to van der Kolk (1996), dissociation at the time of trauma is probably the single most important predictor of PTSD. Dissociation contributes to TR by disturbing a person’s normal sense of reality and time.

Scaer (2001) proposes a model of chronic trauma and disease that is linked to altered brain function (cyclical autonomic dysregulation) and dissociation, often originating with early traumatic attachment (childhood maltreatment-related PTSD). In this model contributing dissociation/freeze mechanisms include vegetative parasympathetic/vagal tone (reduced blood pressure, heart rate) and secretion of endogenous opiates (endorphins). Triggered by fear, these endorphins numb pain, reduce panic, and perpetuate increased dissociative tendencies.

Scaer postulates that dissociation/freeze responses fuel an ongoing arousal/memory circuit in the brain that precipitates TR. Conditioned fear contributes to this circuit. Essentially indelible, it is triggered by stress hormones and mediated by the amygdala (primitive right brain fear response center). For example, Beth's fear that she had "murdered" Father (aversive stimulus) was paired with seasonal changes in temperature and light (neutral stimulus) associated with October. Years later, these seasonal changes triggered unconscious recreations of the original repressed "murder."

Psychophysiological "sensitization" is also fear conditioned, precipitating overgeneralized responses to traumatic or even neutral stimuli. Beth's fear of Father's angry voice and frowning face was held undischarged in her somatic unconscious. During stressful encounters, unexpected subtle shifts in anyone's voice or facial expression left her disoriented and uncomprehending. Furthermore, Beth's allergies resulted from her immune system becoming oversensitized to certain pollens and foods, and conditioning is implicated in this response as well (Kiecolt-Glaser & Glaser, 1993).

Traumatic memory, largely unconscious and frequently distorted, is imprinted differently than ordinary experience. In traumatic dissociation unbearable aspects of experience are dispersed into the body in the form of sensory fragments (van der Kolk, 1996) that are stored in the non-verbal domain of the somatic unconscious. Stress hormones link these memories to one's psychophysiological state of arousal at the time, making them "state-dependent." Although dissociated trauma remains outside conscious memory, at certain moments it recurs via TR in the form of somatosensory flashbacks mediated by stress hormones. For example, at night in bed, after terrifying arguments with an abusive boyfriend, Beth experienced a recurring proprioceptive flashback from childhood: in bed after Father's angry whippings, she would feel herself closely contained inside a soft, quiet, white tunnel-like place in her imagination, her body overcome with numbness.

Treatment

Treatment of TR must involve two primary mechanisms: (1) completion of thwarted fight-flight/freeze responses in the body-mind-brain, and (2) reunion of repressed and dissociated material with consciousness. Effective treatment requires intrapsychic *and* intrasomatic change. Within the transference-countertransference relationship, safe enough containment (regulation)—*not* unregulated, potentially retraumatizing catharsis—is the goal. While patients tell their stories, therapists track moment-to-moment shifts in their posture, facial expressions, sounds, gestures, bodily movements, as well as changes in skin color, musculature, and breath. Levine's (1996) Somatic Experiencing method provides a model for integrating the "felt-sense" with Schore's regulation theory.

Even within a safely regulated container, traumatized patients can easily become overactivated. Jung (1928/1960) realized that intense affects trigger unconscious traumatic "complexes" (dissociated self-states) that can behave independently, sometimes like "wild animals." Initially, complexes arise to preserve an infant's sense of "going on being" (Winnicott, 1974). Ferenczi (1955) proposed a model illustrating how these complexes take shape: before the ego solidifies, one part of the personality *regresses* to an infantile state, while another part *progresses*, becoming precociously mature. Split off from the body, this progressed part is described by Winnicott as a "false self," a mind that has become "a thing in itself."

Though originally intended to serve as "protector" for the weaker part (Kalsched, 1996), the actions of this false self are modeled after parental attachment failures. Consequently, the false self-protector "identifies with the aggressor" (Ferenczi, 1955) and manifests a persecutory style. Repeating the original parental abuse/neglect intrapsychically, the protector/persecutor uses criticism to intimidate the vulnerable self, banishing the emotions that would cause this weaker self to become overwhelmed. The protector/persecutor generates TR by functioning like a circuit breaker (Kalsched, 1996). Acting in true autoimmune fashion by losing "self-tolerance" for the vulnerable self, the protector/persecutor eradicates connections between body and mind, affect and image. In Beth's case, for example, when discord in a relationship led to increased vulnerability, her protector/persecutor simply precipitated another breakup,

and once again TR assumed the form of an October “murder.”

Given the proclivity for such splitting in traumatized patients, if embodied reintegration is to remain a therapeutic goal, sensitively attuned clinicians must help patients relate to the somatic qualities linked to these dissociated self-states. For example, when the critical protector/persecutor dominates the personality, the patient may present with hypervigilance and constriction in the body, and the vulnerable self may remain disconnected. At these times, if the vulnerable self emerges at all, it may present in a depressed, collapsed or mute state. Only when there is sufficient safety and support in the transference-countertransference relationship can the self give over to softer feelings.

As if exploring waking dreams, therapists can foster their patients’ developing interest in their sensory-affective experience by asking specific questions: Where in your body do you notice this sensation (of constriction or collapse)? How big is it? Is it moving or still? What is its quality? Does it have a temperature, texture, color, or pulse? At the same time therapists also track their own intrasomatic-intrapsychic countertransference. As attention is applied to these states, they begin to shift. By first interactively regulating these more primitive levels of sensory processing (subcortical, right-brain-dominant, “bottom-up” processing systems), patients manifest improved reflective capacity for “top-down” (memory influenced brain processing) assessment of TR dynamics.

Imagine the process of interactive regulation tracing a figure-8 image: therapists help patients cycle gently back and forth between sympathetic arousal states (in tolerable doses) and parasympathetic restful states, carrying the tension of these opposites until homeostasis is achieved. Eventually, their fears of intense sensations and affects give way to curiosity about them. Exploratory questions can deepen: What is the associated feeling? What does it remind you of? Is there an associated image?

Beth’s terrors about recurring October catastrophe--TR in the form of intrusion—are reflected in Winnicott’s (1974) notion of “fear of breakdown.” When therapists interactively regulate their patients’ hyperarousal/dissociation responses associated with this fear, they help them realize that trauma retires to the past only after being experienced directly in the present. An important mechanism for the recapitulation and dyadic repair of early trauma within the therapeutic alliance is the patient’s reactions to the therapist’s inadvertent failures and mistakes.

In Beth’s case, for example, there were times when her therapist’s empathic failures reduced her vulnerable self to a collapsed, mute state. Other times, despite Beth’s fear of her own aggression, her protector/persecutor became hyperaroused and exploded with angry complaints. Eventually, as Beth’s relationship with these dissociated self-states grew stronger, unbearable affects could once again be linked with associated images. As defenses gave way and tears that had remained frozen in an encapsulated enclave finally began to melt, Beth grieved the empathic failures of her therapist in the present moment. At the same time she also began to mourn the sorrows of the past--the emptiness of having been unwanted and abused by Father, the loss of sufficient protection from Mother--*experiencing directly* in both body and mind their painful meanings. Relieved that her “murderous” protector/persecutor had not destroyed the therapist’s non-retaliatory stance, Beth learned to differentiate defense from ego. Setting limits with its persecutorial tone, she began autoregulating the self-hatred endemic in early trauma.

When early abuse has become a self-fulfilling prophecy, the traumatized patient’s desperate need for healing can motivate a heroic journey. In the dance of interactive regulation that emerges between therapist and patient, movement pendulates between “then” and “now,” rupture and repair, hyperaroused fear and quiet courage. An alliance of self-states consolidates as the traumatized patient moves toward the wholeness of being one self while, at the same time, many. Carrying the tension of the opposites, this self comes to face and embrace its true suffering, a courageous act that paradoxically transforms hopelessness into healing. The traumatized patient attains the homeostasis of “psychosomatic indwelling” (Winnicott, 1974) as a well-regulated psyche finally returns from soma’s underworld.

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