Environmental factors affecting the nervous system and health:
The role of trauma

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Somatic Psychology Theory as a Mode for Understanding the Origins of Chronic Illness: A Case Study of Type 1 Diabetes

Chapter 2: *Literature Review: Trauma*

Abstract

Trauma is described as an experience that is perceived by a vulnerable and helpless individual as inescapable and life-threatening. In addition to PTSD, the long term consequences of trauma can include chronic pain syndromes and chronic illness, such as those associated with autonomic dysregulation. Symptom severity is associated with the intensity and frequency of the original traumatic event, and symptoms may not begin for weeks, months, or years following the event. The experience of trauma is most intense when experienced in relationship, and the impact is often most severe for children who experience neglect or abuse by a parent. Individuals with a history of trauma often unconsciously experience further trauma in a process referred to as reenactment, which is increasingly understood to be fostered by biological consequences of prior trauma.

This article presents some of the physiological processes that lead to symptomatology, and describing the creation and kindling of conditioned responses as one example. This article draws primarily from work by Robert Scaer and Peter Levine, whose work contributes to the growing body of scientific evidence that supports these findings.

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Trauma

Introduction

This section on trauma is based on a book written by a Boulder, Colorado neurologist who has been studying the physiology of trauma for over a decade. Robert Scaer's (Scaer, 2000, 2001a, 2001b) theories have been instigated and strongly influenced by Peter Levine's (1997) work. Unless otherwise specified, the content of this section specifically refers to Scaer's book *The Body Bears the Burden: Trauma, Dissociation, and Disease* (2001a).

Trauma is described as the experience of a perceived inescapable life-threatening event and the result of such experiences often results in symptoms of avoidance, hyperarousal, and a decreased ability to cope with even small stressors. The symptoms of trauma vary, and because many of them make no apparent physiological sense to physicians trained in the conventional medical system, these symptoms are often disregarded or actively denied consideration or evaluation (Scaer, 2001a). This was also the case in the Victorian era with respect to symptoms of "hysteria".

A Brief History of Trauma Theory

The field of trauma has gone through waves of interest ranging from periods of considerable attention to times when it has been ridiculed or completely disregard. High interest was expressed in the late 1800s and early 1900s when the fields of psychiatry and neurology were still a single discipline and physical as well as emotional illnesses were both considered to be related to problems with the nervous system. Janet and Jean Martin Charcot were two important neuropsychiatrists of the time who believed that "hysteria" and other unexplainable symptoms were real and were due to effects of experiences from early childhood trauma.

Freud, the father of psychoanalysis as we know it today, was a mentee of Charcot's and a neurologist / neuropsychiatrist who was also particularly interested in understanding the origins of these types of unexplainable symptoms (Young Doctor Freud [Television broadcast], 2002, November 27). Although Freud originally thought these symptoms to be due to childhood trauma, he thought they occurred specifically as a result of sexual abuse (Young Doctor Freud, 2002). Freud attempted to apply this understanding to treating his own personal symptoms of headaches, depression and backpain. He spent agonizing years psychoanalyzing himself and working with his assumption that if he had these symptoms, he must have been sexually abused even though he had no recollection of such an event (Young Doctor Freud, 2002). Freud eventually rejected the concept of sexual abuse as a cause of the symptoms of trauma. This is said to be due to his fear of rejection by Viennese society (Boadella, 1990) but may also be due to his lack of success in treating his own symptoms and his eventual realization that he had not been sexually abused (Young Doctor Freud, 2002).

In the long run, Freud's conclusion had a significant impact on the pursuit of trauma theory. The almost universal acceptance of psychoanalytic theory by the field of psychiatry resulted in the almost total rejection of the effect of child abuse and trauma on personality development, character traits, and psychiatric illness for almost eighty years. Although two World

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Wars brought back to reality the fact that overwhelming life trauma can result in profound emotional dysfunction, sporadic attempts to relate this association in a clinical setting were overwhelmed by the prevailing concepts of psychoanalytic theory (Scaer, 2001a, p. 59).

Interest in trauma theory recurred after the Vietnam War, when the symptoms exhibited by many soldiers post-combat were acknowledged and designated as Post-Traumatic Stress Disorder (PTSD). Since then, research in the field of trauma has exploded and "there has been an increasing emphasis on the neurochemical, neurohumoral, and neurophysiological changes produced by the experience of trauma and the resulting breakdown of the separation of psychological and biological processes of mental illness" (Scaer, 2001a, p. 60).

This section of the literature review provides an overview of basic concepts of trauma as they relate to the development of physical disease. Scaer builds a case for understanding the manner in which the experience of trauma leads to the development of physical as well as psychological symptoms, describing a continuum between mind, brain, and body. Throughout his work, Scaer confronts the myth that PTSD is a purely psychological phenomenon.

Symptoms related to Trauma

Trauma is defined as an event that is experienced by someone who a) *feels helpless and b) perceives the experience as inescapable and life threatening*. This experience, which cannot be resolved through fight or flight, requires the more extreme form of survival resources activated by the freeze response. The more significant and intense the arousal associated with the traumatic event, the less it may be possible to integrate, and the more significant the freeze response. In trauma, the more intense the state of parasympathetic helplessness, hopelessness, and despair, the greater the level of dissociation, and according to Scaer, the greater the eventual physical and emotional symptomatology.

The experience of dissociation occurs in the freeze state, and may be related to increased levels of endorphins (opioids), which numb the brain and body in an adaptive survival mechanism that decreases pain, such as in an animal that is about to be eaten. While the freeze state is adaptive in its capacity to foster survival in the short-term, it may be lethal in the long-term if maintained.

Levine (1997) describes freeze as a state of high, undischarged arousal in which the action of "discharging" may be the key to long-term survival. The role of discharge in long-term health is discussed below. According to Scaer, trauma is probably the source of chronic disease as well as chronic pain.

The symptoms of PTSD are those of exaggerated hyper- or hypo-arousal. Signs and symptoms of sympathetic arousal include jitteriness, tension, exaggerated startle reflexes, irritability, difficulty concentrating, nightmares, anxiety, and panic attacks, as well as diarrhea, muscular pain, headaches, blurred vision, elevated blood pressure and heart rate, and increased perspiration. Symptoms of hypo-arousal, which often alternate with the symptoms of hyper-arousal, include a sense of detachment (dissociation), numbness, depression and apathy, as well as constipation, hair loss, cold temperature, and tissue damage due to decreases in vascularization of localized areas, and syncope or dizziness due to low blood pressure and heart rate, among others. These symptoms are all related to the specific functions of the sympathetic

(SNS) and parasympathetic nervous systems (PNS) described in earlier sections.

Differences between Stress and Trauma

Trauma differs from stress in a number of ways, especially in the types of defensive nervous system responses that it evokes. Whereas stress is associated with varying degrees of sympathetic arousal, including activation of the sympathoadrenal medullary response (SAM) and hypothalamic-pituitary-adrenal axis (HPA) responses, trauma is associated with high arousal of both sympathetic and parasympathetic activity.

The physiological response to trauma is associated with the more extreme and evolutionarily primitive dorsal vagal mediated freeze response (Porges, 2001; Scaer, 2001a) and is evoked due to a heightened perception of threat.

Autonomic Nervous System

When PTSD develops after an undischarged freeze response, symptoms may consist of simultaneous parasympathetic and sympathetic dominance (Levine, 1997; Scaer, 2001a). or oscillation between extremes of these two states, which occurs as a result of exaggerated cycling of states of SNS and PNS dominance (Levine, 1997; Scaer, 2001a). In addition, PTSD can develop following one or more "sentinel" events, whereas the symptoms of stress are generally associated with *chronic* exposure to stimuli that activate sympathetic arousal to varying degrees (Scaer, 2001a).

Immune System

Immune responses differ between states of arousal. Activity of the immune system is increased during acute stress such as during fight/flight, but is decreased in chronic stress. The decrease in immune system activity during chronic stress is associated with high cortisol levels. Scaer (2001a) raises the possibility that chronic PTSD, which is associated with *low* levels of cortisol, may be associated with an increase in immune system activity and therefore may "contribute to vulnerability to autoimmune diseases" (p. 74). Increased immune system function in chronic PTSD may be the result of parasympathetic dominance (R. Scaer, personal communication, March 28, 2002). Studies evaluating PTSD and the possibility of increased risk for autoimmunity have yet to be conducted (Scaer, 2001a).

Vulnerability to Trauma

Trauma can occur at any time in life, including during the prenatal and perinatal time frames, childhood, and in adulthood. The state of helplessness in the response to threat as found in trauma is associated with a significant degree of vulnerability. By this definition, the greater a child's dependency on his or her caregivers for survival, the more at risk for the effects of trauma. Because of their inherent vulnerability, prenates, infants, and young children are therefore at greatest risk of suffering the consequences of trauma.

Trauma and Attachment Relationships

Experiences of misattunement associated with lack of psychobiological regulation through affective reunions (in relationships with caregivers) are associated with the inability to self-regulate (Schore, 1994), and are a form of relational trauma. This is in contrast to experiences of consistently welcoming reunions, in which high or low levels of arousal are discharged, metabolized, and integrated to enhance a child's ability to easily, and eventually autonomously, navigate between autonomic states of high,

low, and moderate arousal (Schore, 1994).

Because of the absolute need for caregiver protection for survival, trauma experienced in relationship has the most profound effect on psychophysiological functioning. Some refer to this early form of wounding as "developmental trauma" (Minton & Ogden, 2000). Because of the role of experience in shaping the developing nervous system in early life, traumatic experiences during this period shape nervous system responses to future exposures to stress and trauma. "Accumulation of traumatic life experiences then leads to a condition of increasing vulnerability and decreased resiliency to further trauma" (Scaer, 2001a, p. xxi).

Adaptive Capacity

The capacity of the ANS to transition between states of arousal is directly associated with survival and adaptability. Adaptability involves the "capacity [of the organism] to make a successful response to perturbations in its … environment such that the next time it encounters that same perturbation, or one sufficiently similar, it can respond with less cost" (Chisolm, 1990, p. 240, as cited in Schore, 1994, p. 366). This capacity to transition between ANS states using various modes of regulation is set as a result of interactions in early life (Schore, 1994).

Individuals initially respond to the presence of a novel stimulus through an orientation response in which there is nonreciprocal coactivation of both sympathetic and parasympathetic functions (Schore, 1994). The interpretation of the meaning of the stimulus determines the evolution of the response and, consequently, whether arousal states return to baseline or are engaged in defenses such as fight/flight, or freeze. The possibility is therefore entertained by this author (V. Mead) that the freeze state, which involves elevated levels of arousal of both systems, may represent a state of uncoupled activation, since it differs from the orienting state. The fact that autonomic balance is determined by interactions in early life may be relevant to understanding why individuals with a prior history of trauma are at increased risk for further trauma.

Procedural Memory and Conditioning

Nondeclarative memory is associated with the formation of habits, skills, conditioned responses, and emotional associations and is stored in the right hemisphere. This form of memory is

> automatic and instinctual, [and] is based on past experiences and training. The part of nondeclarative memory that serves skills and habits as well as conditioned sensorimotor responses is called procedural memory. Procedural memories are readily acquired without intention, and retained forever without awareness, especially if linked to a coincident event [associated with intense states of arousal] (Scaer, 2001a, p. 37).

Conditioned responses form another component of nondeclarative memory and may influence sensorimotor or autonomic nervous system responses to particular stimuli. By definition, these types of responses are unconscious.

Conditioned responses are familiar to many of us due to Russian researcher Pavlov's experiments in which he induced salivation in dogs by first pairing the sound of a bell to exposure to food, and then ringing the bell in the absence of food. These types of conditioned responses are not related to high arousal levels and extinguish after a while if the subject is

only exposed to the conditioned stimulus, in this case, the sound of the bell.

This type of conditioning, unlike most procedural memory, is not permanent. If, however, the paired stimuli include a component involving high arousal or emotion, it will take fewer trials of exposure to produce the conditioned behavior, and more trials of unpaired stimuli to extinguish it. In fact, if one of the stimuli represents a life-threatening event, the conditioned response may appear after one trial and never be extinguished (Scaer, 2001a, p. 38).

LeDoux (1996) presents another facet of conditioned responses and describes how some such responses appear to extinguish even though they originated in the presence of high states of arousal such as intense fear. Exposure to the original unconditioned stimulus (represented by salivation in Pavlov's dog) "or to some other stressful event", however, can *reinstate* the apparently extinguished conditioned response (Ledoux, 1996, p. 145). "Spontaneous recovery, renewal, and reinstatement [of conditioned responses] suggest that extinction does not eliminate the memory that the CS [conditioned stimulus] was once associated with danger but *instead reduces the likelihood that the CS will elicit the fear response* [italics added]" (LeDoux, 1996, p. 145). In other words, it may be possible to exert cortical control over arousal states but this does not mean that the memory has been erased or that it cannot be evoked in future stressful situations. This author (V. Mead) also speculates whether this also suggests that conditioned responses may be evoked to include physiological processes such as changes in heart rate and blood pressure without the associated emotional response.

LeDoux explains how this process of apparent but incomplete extinguishing might occur as a result of conditioning.

> Conditioning increased the functional interactions between neurons so that the likelihood that two cells would fire at the same time dramatically increased. These interactions were seen both in the response to the stimulus and in the spontaneous firing of the cells *when nothing in particular was going on* [italics added]. What was most interesting was that in some of the cells, these functional interactions were not reversed by extinction. Conditioning appears to have created what Donald Hebb called "cell assemblies", and some of these seemed to be resistant to extinction. [These] observations suggest clues as to how memories can live in the brain at a time when they are not accessible by external stimuli. All that it would take to reactivate those memories would be a change in the strength of the input to the cell assembly. This may be something that stress can accomplish" (LeDoux, 1996, p. 251-2).

LeDoux's final point suggests that stress may be a trigger that evokes conditioned responses or further promotes them. The development of connections between cells is consistent with the concept of "kindling" sometimes referred to in the trauma literature, and which will be discussed in more detail shortly.

The memory of a perceived inescapable life-threatening event that occurs in a state of helplessness is stored in unconscious procedural (nondeclarative) memory. Unconscious memories are stored in the right hemisphere and, similar to the actions of internal representations of images formed during attachment experiences (Schore, 1994), provide an

internal reference from which to evaluate and respond to future experiences. These stored traumatic memories impair orbitofrontal cortical capacity to regulate arousal (Scaer, 2001a, p. 45) and lead to the development of pathways that organize together to quickly and efficiently respond to perceived threat.

High arousal and catecholamine levels, particular in the amygdala, enhance the storage of traumatic procedural memories, and simultaneously inhibit the creation of conscious "declarative" memories. The right amygdala is the primary facilitator for arousal-related memory (Scaer, 2001a; Schore, 1994). High cortisol levels, which are toxic to the hippocampus where conscious memories are stored, further inhibit the storage of these conscious memories. Damage to the hippocampus results in excessive responsiveness to environmental stimuli (Scaer, 2001a, p. 37) probably due to inhibition of conscious regulatory mechanisms of the left hemisphere. By definition, traumatic events are associated with extremely high levels of arousal and therefore leave permanent memories that are extremely difficult to eliminate or extinguish.

One of the findings in trauma theory is that once a traumatic event is stored in procedural memory, any internal or external environmental stimulus that was associated with the event, even if unrelated to the specifically traumatic part of the experience, can trigger arousal levels and physiological responses originally evoked in the first experience. Survival-based responses that occur in the face of significant threat are permanently stored such that the same response can be quickly and efficiently retrieved and implemented in the event of a future exposure to a similar threat. This saves the time it might take to have to figure out the response anew and is an evolutionarily adaptive function that increases an animal's chance of survival.

Discharge

The freeze response is associated with high levels of both parasympathetic and sympathetic arousal described as a state of "suspended animation" (Scaer, 2001a). For example, when a prey animal that has been running at maximum speed gets overtaken, it may suddenly drop as if dead in a last effort to fool its predator in an attempt to survive. When an organism transitions from such a dominant sympathetic flight response to a parasympathetically mediated and parasympathetically dominant freeze state[1], high levels of catecholamines are not instantaneously reabsorbed. This perspective is consistent with Porges' (2001) description of slow recovery from states of high arousal due to slow reabsorption of circulating catecholamines (in comparison to more rapid recovery from lower arousal states mediated by direct innervation). Levine describes this freeze as a state of high, undischarged arousal.

Wild animals discharge the freeze state through physical movements ranging from subtle trembling to grand mal seizures (Scaer, 2001a, p. 18). Discharge involves autonomic processes ranging from invisible or subtle, to visible activity such as shaking or perspiring, usually ending in a series of deep sighing breaths. Caldwell's (2001) somatic psychology perspectives are similar and in fact emphasize that *all* forms of discharge

[1] Caldwell disagrees with this statement and feels that the freeze state is consistent with high sympathetic arousal, not parasympathetic arousal (personal communication, April 15, 2003).

occur through movement at some level, ranging from invisible physiological movement to overt muscular activity.

Following the few minutes it may take to discharge, animals appear to be in a state of moderate, rather than high or low arousal and long-term studies show no evidence of disability or poor health. Although wild animals experience life-threatening circumstances on a frequent basis, they do not appear to suffer the negative consequences associated with high levels of arousal, even following intense states of death feigning.

According to traumatologist Peter Levine (1997), the difference between animals and humans is that animals tend to discharge states of high arousal following freeze responses whereas traumatized humans often do not. Rather than being an evolutionarily adaptive strategy, this "may represent a dangerous suppression of instinctual behavior, resulting in the imprinting of the traumatic experience in unconscious memory and arousal systems of the brain" (Scaer, 2001a, p. 20).

Levine (1997) and Scaer believe that humans who develop PTSD are the ones who do not discharge the high arousal at the time of the traumatic event. This lack of discharge is thought to be the result of socialization. This concept complements Schore's' (1994) description of infants who learn to discharge when in bonded relationships to psychobiologically regulating attuned mothers, and who fail to effectively learn this skill in misattuned dyads. The long-term consequences of undischarged high arousal, whether learned in relationship or in unrelated traumatic events, are a decreased capacity for physiological self-regulation. Levine (1997) further supports this theory by noting that wild animals that do not discharge after being captured usually die after release in the wild. He further states that inhibited discharge may also occur in animals maintained in chronic captivity, such as those kept in zoos, circuses, or laboratories.

Symptoms arise from lack of discharge

The degree to which symptoms are experienced depends on the level of undischarged arousal and the associated degree of dissociation that occur at the time of the traumatic event. The process of discharge frees the nervous system of arousal and allows it to return to baseline. Discharge thus fosters the nervous system's capacity to know that the traumatic event is over.

> The self-perpetuated circuitry involved in kindling is remarkably compatible with the absence of discharge. Retention in procedural memory of this experience may serve as a recurrent internal cue for recurrent arousal patterns … Until the act of flight or self defense has been completed, therefore, the 'survival brain' may continue to perceive that the threat continues to exist, and is unable to relegate it to memory as a past experience (Scaer, 2001a, p. 21).

In somatic psychology (2001), discharge is referred to as a process of completion, and usually refers to the completion of a sequence or cycle.

Symptoms and unconscious perceptions

According to Scaer, the symptoms of PTSD are experience-based rather than injury-based. In other words, the muscle pain, blurred vision, and headaches associated with PTSD following a car accident are the result of ongoing arousal perpetuated by environmental cues, rather than due to actual physical injury to the muscles, head, or brain at the time of the accident. From this perspective, trauma is

perpetuated by a continuous state of triggered arousal and is expressed as an ongoing freeze state with simultaneous or alternating sympathetic and parasympathetic arousal.

Kindling

In trauma theory, the term kindling refers to a "relatively permanent change in the excitability of neuronal networks" (Scaer, 2001a, p. 44) within a sensitized part of the brain. This term comes from studies in rats in which the repeated use of low intensity electrical stimulus produces seizures that are self-perpetuating and *require no further stimulus for seizure activity to occur*. Doses of electrical shock that do not produce seizures in one application but that are repeatedly reapplied within a certain time frame "summate" and trigger a seizure. The seizures are the result of the creation of new arousal circuits that are activated by increasing numbers of internal and external cues to become self-perpetuating. This description is similar to Hebb's "cell assembly" described by LeDoux (1996). Kindling is thought to underlie the pathophysiology of trauma and is associated with a lack of discharge.

During the period following trauma, kindled connections form between centers of arousal, such as the amygdala and locus coeruleus, and the memory centers in the hippocampus and orbitofrontal cortex. Connections also form between the various systems involved at the time of the traumatic event. This results in an increase in arousal-generating pathways potentiated by internal and external cues that increasingly trigger arousal and evoke the freeze state that occurred during the accident. In time, progressively unrelated cues participate in kindling this ever-increasing circuit. An example is provided below to further elucidate this concept.

Example of experience-based self-perpetuation of trauma

Following a motor vehicle accident (MVA), a small percentage of individuals develop a whiplash syndrome associated with head and neck pain, blurred vision, dizziness, and difficulty concentrating, along with a number of other symptoms of PTSD described earlier. These symptoms, which often may not occur for a period of weeks, months or years following the accident, are remarkably similar in individuals who have been in different types of accidents, including small accidents occurring at 10 to 15 mph that result in no symptoms in the other driver. Contrary to the current medical model that frequently views these symptoms as a form of malingering or a ploy for financial gain, Scaer explains that these symptoms are the direct result of the psychophysiological process of trauma.

During an MVA, individuals are caught in what is frequently experienced as an inescapable life-threatening event in which they are helpless to defend themselves or to escape. In the moments prior to impact, a large amount of sensory input comes in from visual, auditory, and vestibular systems at the same time that a sudden state of freeze is evoked. While this type of input is usually unconsciously processed, prioritized, and selectively stored, a different course occurs as the result of trauma. The following is an imaginary description of what might occur and summarizes an example provided by Scaer (2001a) with a few elaborations such as the naming of different types of auditory or visual input (screeching brakes, color of the trees, etc).

Example of a car accident

During an MVA, input comes into the brain from forward head movement, from muscles tensing in preparation for action,

and from the simultaneous stretching and torquing of muscles due to the powerful forces of the nonlinear car movement as it spins out of control. Visual input includes the color and direction of the oncoming car, the street corner or location of the event, and the color of the trees at the curb. Auditory input includes the sound of screeching brakes, and the impact of metal. Olfactory input comes from the smell of burning rubber and the seasonal weather, such as wet fall leaves. While this type of information is usually prioritized and filtered (Caldwell, 1999), all of this sensory information is stored in procedural memory due to the high state of arousal.

Autonomic responses are also registered during the MVA. Fight/flight responses that occur, including increases in heart rate and blood pressure that maximize available blood volume, pupil dilatation and eye divergence that permit a maximized view of the oncoming threat, and glycogen breakdown and insulin inhibition that increase fuel availability in preparation for mobilization, are stored in procedural memory in association with the event.

The time until the actual impact occurs seems to take forever as everything begins to happen in the slow motion of parasympathetic numbing and dissociation. The impact sends in additional input, as the change in direction is temporarily associated with inertia that inhibits the body from moving. Rebound sends further input from muscles related to changing head, neck, and body position. These fragments of data are also stored in procedural memory. Scaer describes the effects of the rapidly accelerating and decelerating car as causing

> marked perturbation of vestibular receptors in the semicircular canals, which affect balance. Those parts of the body involved in the control of the vehicle, especially the hands on the steering wheel and feet on brake or accelerator pedals, will react based on procedural memory patterns of motor driving behavior. The resulting motor response will involve bracing, clenching, or turning of the body in reflex reaction to visual, vestibular, and proprioceptive input from sense organs in the head, neck, and muscles of the body…. The next physiological demand calls for completion of the high level physical activity required to dissipate the effect of adrenergic [norepinephrine induced] arousal on the body. At this point the meaning of the event and the relative empowerment of the victim to pursue the physical activity of the fight/flight response become critical (Scaer, 2001a, p. 39-40).

In a state of helplessness, these responses of high sympathetic arousal will just as inevitably trigger the freeze response (Scaer, 2001a) as they will elicit fight/flight.

The moments following impact are associated with a sense of the surreal as numbness sets in along with the detached calm of dissociation as the event is stored in procedural memory. Hours, days, or perhaps years later, symptoms will begin to develop. Many of these symptoms are similar in different people regardless of the original trauma, as they are based on the perpetuation of the effects of arousal as much as they are based on the activities that occurred in the event. The following paragraphs describe some of the specific symptoms that develop and why.

Changes in vision following a traumatic event.

The pupil dilatation and eye

divergence that occurred involuntarily as a normal part of the sympathetic arousal response in the MVA are "frozen in the event of the freeze response, and dissipated by its physiological discharge in the event of survival after freezing" (Scaer, 2001a, p. 49). As a result of lack of discharge at the time of the accident, the eye changes

> triggered by traumatic arousal will continue to be linked in the evolving feedback circuit between centers of arousal and those for procedural memory, the storehouse for motor skills and habits. Under these conditions, any arousal, whether linked to the other memories of the accident or even occurring in the stresses of everyday life, will nonspecifically trigger ocular divergence and pupillary dilatation. In fact, these are basically the clinical findings that have been documented in examinations of patients with postconcussion vision abnormalities …. In this model … these changes are not the result of brain injury per se, but rather the incorporation of the eye muscles in the neuromuscular/arousal/memory conditioned and kindled circuit of unresolved trauma (Scaer, 2001a, p. 49).

Blurred vision can result from pupilary dilation and ocular divergence and can be aggravated when states of arousal occur. Arousal will be aggravated as objects cross the field of vision in the area associated with the memory of the visual input of the incoming car. This visual input, which occurs outside of conscious awareness, will trigger pupillary dilatation and ocular divergence. As a result of one visual cue, the kindled circuit will grow and will associate with other parts of the body systems involved in the traumatic event. In time, increasingly unrelated cues will simultaneously trigger all of the systems involved in the accident. As a result, muscles will tense, heart rate will increase, glycogen will be broken down into glucose, and insulin will be inhibited.

Similarly, movements of the head that access the same groups of muscle patterns involved in the accident will trigger sensations of vestibular overwhelm evoked by the procedural memory of neck movement that occurred during the MVA. Dizziness, vertigo, and loss of balance may ensue. Activation of bracing and movement patterns involving these muscles may arouse procedural memories of all the vestibular stimulation experienced in the accident and linked at that moment to perceived life threat. Vertigo in the trauma model therefore represents survival-based procedural memory (Scaer, 2001a, p. 50).

As kindling circuits develop, these head movements will trigger the symptoms of arousal associated with the accident, even if the neck or head movements are associated with benign activities such as brushing one's hair or looking before crossing a street. The smell of burning rubber, wet leaves, or the sound of screeching brakes may also trigger the full arousal response.

Autonomic activity as a trigger.

A similar circular feedback loop associated with kindling is the triggering of symptoms by autonomic arousal. An elevation in heart rate that occurs from conducting one's morning jog may trigger an arousal response of fight/flight, and consequently initiate vertigo and muscle symptoms. Scaer states that scores of patients have presented to his office with elevated blood pressure (over 170 systolic) and heart rates of 120 to 140 at rest, in

addition to their diagnostic symptoms of PTSD (American Psychiatric Association [APA], 2000). Patients have also presented in his office with predominantly parasympathetic responses including bradycardia and low blood pressure associated with dizziness. These ANS symptoms represent exaggerated SNS and PNS responses which appear to be due to ongoing kindling promoted by arousal states. These symptoms may be most dramatic in the early stages following an MVA.

A labile and unstable autonomic nervous system is known to accompany PTSD. The primary measures in chronic cases involve documentation of unstable pulse and blood pressure responses to nonspecific arousal stimuli, sounds, pictures, smells reminiscent of the trauma, or guided imagery of the trauma. Electrodermal skin response has also been used with some success in identifying PTSD autonomic overresponders. Victims of PTSD are known to cycle in and out of arousal and dissociation, the former associated with adrenergic dominance, the latter with endorphinergic and probably selective vagal influences" (Scaer, 2001a, p. 50).

Chronic pain and physical symptoms.

Movement patterns associated with attempts at self-protection during an MVA are also affected by and facilitate kindling circuits. A visual cue or autonomic process such as pupil dilatation will trigger the circuit and the resulting nonspecific arousal

> will tend to facilitate procedural neuromuscular memory of regional protective muscular bracing patterns from the MVA. Reflex activation of muscle groups will then be produced in a pattern mimicking that associated with the body movements caused by the accident. The MVA victim will experience involuntary tightening of selective groups of … muscles in a repetitive pattern, triggered by arousal, dreams, driving activities, or memories of the accident …. Since muscles are designed to contract briefly on a reciprocal basis with their opponents, involuntary sustained contraction … leads to accumulation of metabolic waste (Scaer, 2001, p. 52).

The accumulating metabolic waste and associated arousal-related decreases in blood flow result in the accumulation of increased chemicals that together generate pain. The decreased blood flow can also affect a specific area over the long term and lead to local changes such as a decrease in hair growth and muscle atrophy from lack of blood supply.

Reenactment

Victims of trauma are at increased risk for future trauma, and are likely to have been victims of previous trauma (Levine, 1997; Scaer, 2001a; van der Kolk, McFarlane, & Weisaeth, 1996). This common occurrence is well documented in trauma research and is referred to as "reenactment". Reenactment can manifest as 1) repeated exposure to trauma of the same nature, such as multiple MVAs or domestic violence in adult relationships between individuals with a history of childhood abuse, 2) the experience of traumatic events on anniversaries (the same season, day of the month or time of year as an earlier event), and 3) thrill-seeking or risk-taking behavior (Scaer, 2001). This phenomenon, which is considered by some to be an active form of self-destruction or masochism, is instead the result of an *unconscious* biochemical

process that is "probably a neurophysiologically deep-seated conditioned response" (Scaer, 2001a, p. 87).

Reenactment is thought to result from unconscious attempts to seek the experience of reward and pleasure, which is psychophysiologically linked with the arousal that occurred during trauma. This reward system is associated with the endorphin (opioid) activity that facilitates the numbing, dissociative features of the freeze state (Caldwell, 2001; Scaer, 2001a) as well as the dopaminergic pleasure states (Schore, 1994) associated with bonding (Scaer, 2001a).

In relational trauma such as childhood abuse, the pleasure state of bonding occurs at a neurophysiological level even in the face of abuse, and is based on the fact that the child is simultaneously completely dependent on the adult caregiver for survival, while also intermittently or consistently in life-threatening danger. This desire to seek protection from the abuser is fostered by the unconscious preference for the familiar over the unfamiliar (Sheldon, A., 1969, as cited in Scaer, 2001a).

The dual association between arousal and pleasure is responsible for reenactment, and represents the mechanism underlying Freud's "repetition compulsion"(Caldwell, 1999), as well as the displacement behaviors referred to by Caldwell as "movement tags" (2001). In this context, thrill-seeking behaviors represent a reenactment of the pleasure and excitement that may have occurred in prior states of traumatic arousal. Trauma anniversaries are perceived to occur as the result of unconscious associations with temporal factors such as patterns of daylight, temperature, and circulating hormonal levels, etc. that mark a traumatic event in procedural memory.

Scaer describes the experience of a patient who had an MVA on January 26, followed by another MVA on March 26. She then developed a black eye after being hit by her roommate's leaping dog on May 26, and on June 26 severely sprained her ankle (Scaer, 2001a, p. 85). Scaer explains the actions of the dog as relating to the acute sensitivity of animals to the mood and arousal level of humans around them.

Another example of reenactment is described by psychiatrist Bessel van der Kolk (van der Kolk, B., 1989, as cited in Scaer, 2001a) whose patient attempted to rob a store every year from 1969 to 1986. The attempts to rob the store were committed unconsciously on the same date and at the exact hour and minute of the anniversary of a traumatic experience the patient had had while serving in the Vietnam War.

In a fourth example, Levine (1997) describes a form of reenactment associated with a transgenerational pattern, which he has seen in a number of individuals. In this example, Levine (1997) refers to the experience of a client who has survived a plane crash. In exploring the details of her history, he discovers that "both her father and her grandfather had survived separate plane crashes" (p. 190). Levine's client was not the pilot of a private plane that she might have unconsciously crashed, but was a passenger on a public airline. This is an example of forms of reenactment that are not clearly understandable given current knowledge, but that clearly exist (Levine, 2001a).

Gender

The individual response to trauma relates to the manner in which the freeze state was experienced during early trauma and these responses frequently differ between the genders. Males tend to dominate the aggressive end of the

sympathetic arousal spectrum of the freeze response in the face of trauma. Females, on the other hand, more commonly respond to trauma with immobility and consequently, demonstrate more symptoms of the parasympathetic immobility response in expressed patterns of PTSD. This difference has been hypothesized to be the result of 1) socialized differences in the genders, 2) defense strategies that evolved in tribal or clan life, or 3) frequency of exposure to trauma (Perry, B., Pollard, R., Blakley, T., Baker, W., and Vigilante, D. 1995, as cited in Scaer, 2001a), among other theories.

> In natural disasters and accident-related trauma, gender differences may cause markedly different responses in males and females. Under these conditions, women seem much more likely to experience a profound freeze, or immobility response, and to sustain that response for a prolonged period …. The roots of gender differences in response to trauma, although probably related in part to theories mentioned, are still not proven. One might speculate that the contribution of genetic differences in interaction between the maternal caregiver based on gender expectations from the moment of birth could contribute to gender-specific vulnerability. Another disturbing possibility, of course, is the fact that females, both in childhood and as adults, are selectively more exposed to trauma and abuse. Unfortunately, there is a great deal of evidence that this indeed is true as a cross-cultural phenomenon" (Scaer, 2001a, p. 91).

Many types of autoimmune diseases are more common in women (Braunwald, et. al., 2001), and this may be consistent with the fact that women more frequently respond to trauma with the immobility response. As a consequence of this common response pattern, women usually experience PTSD with signs and symptoms of parasympathetic dominance, such as depression and fatigue. Since autoimmunity may be associated with low cortisol states of chronic PTSD, a link may exist between diseases of autoimmunity and the experience of trauma (Scaer, 2001a).

Although the etiology of autoimmune diseases is unknown, but some diseases such as type 1 diabetes, systemic lupus erythematosis (SLE), and multiple sclerosis (MS) are known to be influenced by an environmental component and many are more common in women (Braunwald, et. al., 2001). Furthermore, individuals with autoimmune diseases are at increased risk for developing other types of autoimmune related disorders (Braunwald, et. al., 2001).

Concluding Remarks

Trauma differs from stress in a number ways including the fact that it is associated with a combination of high sympathetic and parasympathetic arousal. Symptoms of PTSD occur in the event that the freeze state does not get discharged. The lack of discharge is associated with kindling, which perpetuates states of high arousal.

An event experienced as traumatic is generally followed by "a remarkably prolonged freeze response" in which symptoms of parasympathetic dissociation may mask symptoms of sympathetic arousal. In addition, the symptomatology of PTSD may cycle and alternate between these states of high arousal. It is unclear to this author (V. Mead) whether the freeze state represents a condition of

nonreciprocal coactivation of the two branches of the ANS, or whether it may consist of a process in which they are uncoupled. Perhaps "discharge" enables the branches of the ANS to reorganize and reassociate into nonreciprocally coupled patterns.

The storage of traumatic events in memory is facilitated by arousal and is predominantly unconscious. These procedural memories are essentially permanent and foster a self-perpetuating loop of ongoing arousal as a result of conditioned responses to internal and external cues. Cues that stimulate these patterns of arousal are increasingly unrelated and removed from events surrounding the trauma, and the change in nervous system regulation following trauma is thus experience-dependent. Trauma is associated with unconscious patterns of reenactment behavior believed to be due to endorphin release (which mediates pleasure) associated with the freeze response.

The degree of dissociation and freeze at the time of the traumatic event is directly related to the intensity and severity of later symptomatology, and PTSD occurs in the event that the state of high arousal remains undischarged. Gender differences are seen in trauma and PTSD is generally more common in women (van der Kolk et al., 1996). While men tend to react to trauma with more aggressive behaviors and emotions, women more commonly react with a prolonged immobility response. The chronicity of the parasympathetic component of this freeze response is relevant to the more frequent diagnosis of PTSD seen in women, and may be helpful in understanding the origins of autoimmune disease.

The role of trauma as an environmental factor is an important one. It affects risk for and expression of a large number of symptoms, many of which cannot be well understood from our current paradigms. These perspectives, which are supported by growing body of scientific evidence, appear capable of enabling us to better understand origins of chronic illness and to consequently begin to develop more effective treatment options.

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