

The Neurophysiology of Dissociation and Chronic Disease¹

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Dissociation as a clinical psychiatric condition has been defined primarily in terms of the fragmentation and splitting of the mind, and perception of the self and the body. Its clinical manifestations include altered perceptions and behavior, including derealization, depersonalization, distortions of perception of time, space, and body, and conversion hysteria. Using examples of animal models, and the clinical features of the whiplash syndrome, we have developed a model of dissociation linked to the phenomenon of freeze/immobility. Also employing current concepts of the psychobiology of posttraumatic stress disorder (PTSD), we propose a model of PTSD linked to cyclical autonomic dysfunction, triggered and maintained by the laboratory model of kindling, and perpetuated by increasingly profound dorsal vagal tone and endorphinergic reward systems. These physiologic events in turn contribute to the clinical state of dissociation. The resulting autonomic dysregulation is presented as the substrate for a diverse group of chronic diseases of unknown origin.

KEY WORDS: autonomic nervous system; conversion; dissociation; kindling; RSD (reflex sympathetic dystrophy).

During the last two decades of the nineteenth century, psychiatrists in Europe began to explore and define the peculiar behavior manifested by patients of theirs who fell under the diagnostic category of hysteria. Pierre Janet at the Salpêtrière described dissociation as phobias of memories, in the form of expressions of excessive or inappropriate physical responses to thoughts or memories of old traumas (Janet, 1920). After visiting Janet, Freud adopted many of these concepts of dissociation as a splitting of consciousness, often associated with bizarre physical symptoms and manifestations, and ultimately attributed such symptoms in his hysterical patients to a history of childhood sexual abuse (Freud, 1896/1962). Evolution of the concept of dissociation led to the description of a constellation of varied clinical manifestations attributed to it, including altered perceptions of physical sensation, time, memory, sense of self, and reality. Complex expressions of these states came to include conversion disorder, fugue states, and multiple personalities (dissociative identity disorder; Bremner et al., 1992; Freud & Breur, 1893/1952; Mayer-Gross, 1935; Spiegel & Cardena, 1991). Thus the concept of dissociation evolved to include not

¹This paper is based on a Keynote Address presented at the 31st annual meeting of the Association for Applied Psychophysiology and Biofeedback, March 29–April 2, 2000, Denver, CO.

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only mental and emotional aberrations, but also stereotyped and unusual somatic perceptual and motor experiences and expressions.

All of these symptoms and behaviors were felt to be the sequellae of prior life trauma. The basic mechanism of dissociation was felt to involve the splitting off of parts of memory or perception in order to escape intolerable anxiety triggered by those areas of the mind that retained elements of the traumatic conflict. Relief from that conflict through hysterical dissociation resulted in relief from anxiety, resulting at times in the seemingly blasé acceptance of disabling physical conditions (*la belle indifférencé*).

Freud, however, soon began revising his concepts of hysteria, and by 1925 had recanted his theories of the relationship of hysteria and dissociation to prior childhood trauma (Freud, 1925/1959). He ultimately attributed the stories of childhood sexual abuse in his hysterical patients to fabrication, based on unacceptable sexual wishes and fantasies that they could not acknowledge. As a result, the role of childhood trauma in the etiology of dissociation was basically ignored for decades.

The introduction of the diagnosis of Post Traumatic Stress Disorder (PTSD) into the *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edition (*DSM-III*) in 1980 also resulted in the reclassification of many of the conditions formerly attributed to trauma and dissociation, and in some cases, ignored their association with prior life trauma (American Psychiatric Association [APA], 1980). Van der Kolk et al. (1996) note that in the *DSM-IV*, dissociative symptoms are included under the diagnostic categories of not only PTSD, but also of Acute Stress Disorder, Somatization Disorder, and Dissociative Disorders themselves (van der Kolk et al., 1998). In fact, in the *DSM-IV*, Dissociative Disorders do not include Conversion Disorder, which has now been placed under the Somatiform Disorders. Since the *DSM-III*, the diagnosis of hysteria is nowhere to be found. Van der Kolk et al. make a strong case for the consideration of dissociation, somatization, and affect dysregulation as late expressions of trauma even in the absence of continuing criteria for the diagnosis of PTSD. In doing so, they echo the concerns of Nemiah (1995), who notes that the diagnoses of PTSD, conversion disorder, and dissociation are connected by the common process of dissociation itself, whereas their disparate placement in different categories of the *DSM-IV* inhibits investigation of the psychodynamics of trauma.

This attempt to return to the concepts of a relatively broadly-based response of the organism to traumatic stress is critical to our consideration of the neurophysiology of trauma and its effects not only on systems of the brain and endocrine systems, but also on the body itself. When one accepts the tenet that the clinical expressions of a multitude of psychiatric syndromes derive not only *de novo* or through gene expression, but perhaps also through life experience and its lasting effects on brain physiology, one must return to the concept of a physiological continuum between many psychiatric diagnoses.

CLINICAL TYPES OF DISSOCIATION

One of the dilemmas of classification of symptoms of dissociation is that these symptoms assume many and varied forms and expressions. They may be emotional, perceptual, cognitive, or functional. They may involve altered perception of time, space, sense of self, and reality. Emotional expressions may vary from panic to numbing and catatonia. Altered sensory perceptions may vary from anesthesia to analgesia to intolerable pain. Motor

expressions frequently involve weakness, paralysis, and ataxia, but may also present as tremors, dysarthria, shaking, and convulsions (see discussion of conversion reaction later). Cognitive symptoms may involve confusion, dysphasia, dyscalculia, and severe deficits in attention. Perceptual symptoms include ignorance and neglect. Memory alteration may appear as hypermnesia in the form of flashbacks, or as amnesia in the form of fugue states or more selective traumatic amnesia. The varied symptoms of dissociation therefore mimic the intrinsic bipolar nature of the defining symptoms of PTSD (arousal, reexperiencing, avoidance).

Time perception is often greatly altered, most commonly characterized by a sense of slowing of time (Terr, 1983). Altered perception of self (depersonalization) may manifest as an out-of-body experience, or a sense of intense familiarity (*déjà vu*) (Pynoos et al., 1987). In its most extreme expression, depersonalization may encompass perception of several separate states of self in the form of distinct and separate personalities (dissociative identity disorder), each with distinct personality characteristics and even physical attributes (Mayer-Gross, 1935). Strange persons or events may appear familiar, whereas familiar faces and scenes may appear alien and strange.

Abnormal memories also constitute a significant dissociative phenomenon. Simple amnesia for the traumatic event is common, and may present as complete amnesia, or as distorted or inaccurate memory content (Terr, 1983; Torrie, 1944). Fugue states present an extreme state of amnesia, characterized by periods of time for which the dissociative patient has no memory, often triggered by exposure to cues reminiscent of prior trauma. During that time the person may appear distracted and may not remember personal facts. More often, they may appear confused, histrionic, socially inappropriate, or bizarre (Fisher, 1945).

Perhaps the most unique symptom of dissociation is that of flashbacks. These episodes are distinctive in that they involve intense arousal and reexperiencing, symptoms more related to acute PTSD than to dissociation (Mellman & Davis, 1985). During these episodes, which may last briefly or for several hours or even days, the person will also usually experience more typical dissociative experiences such as depersonalization. Sensory processing and perception may be greatly distorted. During flashbacks, the person may appear confused and detached, but later may report vivid sensory and memory experiences, often associated with intense emotions and states of arousal. The accuracy of the associated memories may be variably valid or distorted.

Conversion reaction and hysteria are no longer described in the *DSM-IV* under dissociation (APA, 1994). In fact the *DSM-IV* goes so far as to assert that if dissociative and conversion-based symptoms occur in the same patient, both diagnoses must be made. The neurophysiological and pathophysiological basis for dissociation proposed in this paper, however, demands that conversion be reintroduced as a specific form of dissociation, one that is closely linked to somatic perceptual alterations that are an acceptable and in fact intrinsic feature of the dissociative process. The model presented proposes that the atypical neurologic symptoms and signs that characterize conversion constitute perceptual alterations based on prior trauma, and represent the same splitting of consciousness that produces disorders of perception of time, space, reality, and self just presented. As such, conversion may be associated with the same spectrum of positive and negative phenomena as PTSD as well as other symptoms of dissociation (analgesia/pain, paralysis/seizures).

MEMORY, TRAUMA, AND DISSOCIATION

Disorders of memory constitute one of the diagnostic categories for PTSD in the form of reexperiencing. As noted in the previous section, this may be in the form of hypermnesia, amnesia, or distortion of memory. Trauma-based memory phenomena often involve declarative (explicit, semantic) memory in the form of variably accurate verbal and imaginal recall of the traumatic event. Declarative memory, the form of memory that relates to facts and events, initially involves hippocampal and prefrontal cortical pathways and plays an important role in conscious recall of trauma-related events. It also is notoriously inaccurate and subject to decay. Procedural memory relates to acquisition of motor skills and habits, to the development of emotional memories and associations, and to the storage of conditioned sensorimotor responses. Procedural memory is unconscious, implicit, and extremely resistant to decay, especially if it is linked to information of high emotional or threat-based content (van der Kolk, 1994). Although declarative memory may account for much of the arousal-based cognitive symptoms of PTSD, procedural memory provides the seemingly unbreakable conditioned link that perpetuates the neural cycle of trauma and dissociation.

Endogenous opiate reward systems very likely contribute to the establishment of conditioned procedural memory in trauma. Researchers have known for decades that exposure to overwhelming trauma in combat often results in a sustained period of analgesia. Soldiers wounded in battle frequently require much lower doses of morphine than in other types of incidental injury (Beecher, 1946). Stress-induced analgesia is a well-documented phenomenon in many forms of traumatic stress (van der Kolk, Greenberg, Orr, & Pitman, 1989). Release of endorphins at the time of acute stress has a distinct survival benefit. An animal ministering to his wounds due to pain at the time of aggressive, life-threatening injury would suffer significant compromise of his defensive capabilities. Endorphins also persist during freeze/immobility, rendering the animal analgesic in the face of the injury from the attack. This also has a potential survival value because the persistence of immobility in the face of painful injury might serve to end the predator's attack behavior. In the event of lack of completion of the freeze/immobility response, however, persistent recurrent dissociation with associated endorphinergic reward might well potentiate the kindled trauma reflex. Endorphinergic influences might also contribute to the phenomenon of compulsive trauma reenactment (van der Kolk, 1989).

THE ANIMAL MODEL

The behavior and physiology of the freeze response have been studied for decades. Freezing of course is routinely seen in the wild, initially as a state of alert immobility, as in the fawn that assumes an immobile state in the presence of a predator. This state may proceed to sudden flight or, if the fawn is attacked and captured by the predator, to a deeper state of freeze, one associated with apparent unresponsiveness and with marked changes in basal autonomic state. Early immobility has the advantage for the prey animal of remaining hidden, especially because movement cues often are necessary to elicit attack by the predator. All animals manifest alert immobility, a state termed "animal hypnosis" by Krystal (1988).

In the event of attack, when the creature is rendered helpless, a different state of freezing is elicited, as just noted. Laboratory studies of this phenomenon yield interesting

results. Hofer (1970) exposed rodents to a variety of predator-related stimuli in an open space with no means of escape. All rodents entered a deep phase of freeze, persisting for up to 30 min. This state was associated with marked bradycardia associated with cardiac arrhythmias, suggesting a pronounced state of vagal or parasympathetic tone.

Ginsberg (1974) immobilized chicks, and then allowed one group to recover spontaneously, and one to recover, but with prodding and stimuli to terminate the freeze. These groups, along with a third group of chicks that had not been immobilized, were then tested for resiliency to avoid death by drowning. The group that had not been allowed to complete recovery from immobility died first, the group not exposed to immobility next, and the group that had spontaneously recovered from the freeze survived the longest. Clearly the experience of and the spontaneous recovery from freezing carries survival benefits, whereas not being allowed to go through this recovery process seemed to reduce resiliency to life threat.

The key to this process appears to revolve around the state of helplessness, or lack of control. In drowning experiments, wild rats will swim for up to 60 h before dying from exhaustion. If these rats experience immobility in the hands of the investigator, and then are placed into the water, they will drown in minutes. Some rats experience sudden death *during* induced immobility (Richter, 1957). The freeze response clearly is associated with high risk to the creature if it is not allowed to dissipate spontaneously.

Studies in animals with inescapable shock (IS) further illustrate this dilemma. Animals exposed to significant shock stimuli in an escape-proof environment predictably freeze with subsequent shock exposure. Subsequent introduction of routes of escape in these animals do not elicit escape behavior—the animals remain frozen, and continue to exhibit helplessness (Seligman, 1975). They appear to be unable to learn from new experiences, even those experiences that promote escape or survival. Animals exposed to escapable shock (ES), however, soon learn to use the escape route and do not freeze (Fanselow & Lester, 1988). The critical factor in trauma, therefore, appears to be the controllability of the outcome of the threat versus a state of helplessness. Van der Kolk (1989) has noted the remarkable similarities between the human response to trauma and the animal response to IS, and have suggested that IS may be a biological model for PTSD (van der Kolk, Greenberg, Boyd, & Krystal, 1985). Nijenhuis, Vanderlinden, and Spinhoven (1998) have presented the novel model of dissociation in humans as an analogy to the alteration in defensive and recuperative behaviors in animals exposed to IS (Nijenhuis et al., 1998). Threat-associated conditioned stimuli (CS) in this model would automatically elicit a dissociative or freeze response, rather than a conditioned response more specific to the stimulus. Persistent dissociation would, therefore, prompt the animal, or human, to be sensitized to continue to freeze, or dissociate, to a wide range of stimuli that might be associated with threat.

Levine (1997) takes the phylogenetic model a step further, by equating the lack of recovery from the freeze, or immobility, response with retention of the stored and undissipated energy of the truncated fight/flight response. This sustained state of sympathetic arousal serves as the drive for the memory and arousal-based symptoms of trauma and PTSD. He attributes the tendency for traumatization in the human species to the inhibitory influence of selected neocortical centers that block the instinctual capability that other wild animal species possess, to “discharge” this retained energy. Noting that animals emerging from immobility often manifest repetitive, almost seizure-like motor activity, he postulates that these stereotyped motor responses are able to allow completion of the motor sequences of successful escape or defense, and therefore, to effect an energetic discharge.

Dissociation in the animal model, then, appears to have many similarities to behavior in animals in whom freezing has been elicited in a state of helplessness with subsequent prevention of spontaneous recovery from immobility. Furthermore, dissociation also may be associated with predominantly parasympathetic tone, impaired cognition and learning behavior, and a tendency for conditioned perpetuation.

TRAUMA AND DISSOCIATION: THE WHIPLASH MODEL

We have previously presented an hypothesis that the Whiplash Syndrome constitutes a model for traumatization rather than for physical injury, and that many of its symptoms and clinical manifestations are in fact a universal response to a life threat in the face of helplessness (Scaer, 1997, 2000, in press). This hypothesis is based on the occurrence of dissociation at the time of the motor vehicle accident (MVA) in the form of numbing and the altered state of awareness often attributed to concussion. Subsequent clinical symptoms are based on theories of limbic kindling in the development of the arousal-memory cycle in PTSD (Goddard, McIntyre, & Leetch, 1969; Miller, 1997; Post, Weiss, & Smith, 1995). Kindling is the name given to the phenomenon in rats of the progressive development of self-perpetuating neural circuits produced by repetitive time- and frequency-contingent regional electrical brain stimulation (Goddard et al., 1969). The behavioral expression of kindling may include epileptic seizures, but kindling is also widely felt to be a model for a number of clinical syndromes, including PTSD.

The neural pathways involved in the process of acquisition of this kindled physiologic response to threat probably involve a series of events involving primarily the locus ceruleus, amygdala, thalamus, hippocampus, and right orbitofrontal cortex (OFC; van der Kolk, 1994). Arousal-based input from a variety of sensory organs, especially those of the head and neck, is transmitted to the thalamus and locus ceruleus. The locus ceruleus then provides input to the thalamus and to the amygdala, which evaluates this input for its emotional content. The amygdala then transmits this information to the hippocampus, the center for declarative memory, which establishes a cognitive context to the information. This data is then transmitted to the right OFC, which organizes the appropriate cortical and autonomic response based on the implications of the sensory information for survival. The OFC therefore functions as a master regulator for organization of the brain's response to threat. Inadequate development of the OFC resulting from a maladaptive childhood experience or from prior brain injury may result in faulty modulation of this arousal response (Schoe, 1994).

Further regulatory control is provided by the anterior cingulate cortex, a center that may provide a gating function inhibiting fear conditioning by inhibitory input to the amygdala (Morgan & LeDoux, 1995). The locus ceruleus, through intense adrenergic input triggered by an acute arousal stimulus, inhibits both the anterior cingulate and the OFC, thereby inhibiting the gating and modulation functions of these two centers. This in turn would allow exposure of the amygdala to overwhelming internal and external arousal cues, thereby promoting the kindled development of pathways producing the clinical syndrome of PTSD (Hamner, 1999).

Dissociation at the time of trauma is the primary predictor for the later development of PTSD (van der Kolk & van der Hart, 1989). Individuals who actively dissociate at the time of a traumatic event are much more likely to develop subsequent symptoms of PTSD than those who do not (Bremner et al., 1992; Cardena & Spiegel, 1993; Holen, 1993). Children

are especially prone to dissociate at the time of a traumatic experience, and therefore, people with a history of past trauma, especially child abuse, are more susceptible to arousal, freezing, and retraumatization after exposure to even nonspecific arousal or traumatic stimuli (Kolb, 1987).

In the whiplash hypothesis, spontaneous recovery from dissociation or freeze/immobility at the moment of traumatic impact often will not occur, based on the premise that involvement in an MVA is by its very nature a model of helplessness. The potential for dissociation to occur will predictably be greatly enhanced by a prior history of trauma and dissociation. This state of altered memory, perception, and autonomic function may potentiate kindling between centers for memory and arousal (amygdala, hippocampus, locus ceruleus) that we have already described. The resulting self-generated and maintained kindled loop will then serve as the substrate for development of clinical PTSD.

From a somatic standpoint, procedural or conditioned memory for sensory input and motor responses to the physical events associated with the actual accident will also be incorporated into this kindled trauma response. In an event of great arousal and threat, only one trial may be necessary for a conditioned response to be established. Thus vestibular, ocular, and sensorimotor experiences of the accident will be imprinted on procedural memory through traumatic operant condition. These perceptions will then subsequently be elicited in exact form by memories, flashbacks, nightmares as well as internal and external cues reminiscent of the MVA. All of the elements of the postconcussion syndrome—vertigo, blurring of vision, tinnitus, headache, myofascial pain—now constitute symptoms precipitated by cue- and memory-based stimuli, and eventually by a wider and wider range of nonspecific arousal-based events. Myofascial pain probably represents procedural memory for the specific defensive motor stretch reflex and its proprioceptive template precipitated by the movement of the body in the MVA, thereafter elicited by stress or any movement pattern reminiscent of the accident, in the form of bracing and muscle spasm. Cognitive impairment may appear and in fact worsen based on well-documented attention and memory deficits in dissociation and PTSD (Alexander, 1992; Bremner et al., 1993; Gill, Calev, Greenberg, Kugelmas, & Lerer, 1990; Grigsby, Rosenberg, & Busenbark, 1995; Miller, 1993). None of this diverse array of symptoms would therefore require tissue injury to produce them.

This hypothesis is dependent on the occurrence of dissociation contributing to an unresolved freeze response as a result of life threat in the face of helplessness. Resulting kindling would then incorporate not only the centers for memory and arousal noted earlier, but also the centers providing the sensory information of the MVA (visual, auditory, vestibular, proprioceptive sensory receptors), and the motor centers that organized the defensive response (cerebellum, brainstem, basal ganglia, motor cortex). Kindling and dissociation would explain the vexing tendency for whiplash symptoms to be resistant to most forms of physical therapy, to persist indefinitely in many cases, and to worsen dramatically in situations of ambient life stress. The proposal also incorporates somatic symptoms into the basic theories of PTSD and dissociation, leading to a somatic definition of dissociation that is the core of this paper.

THE AUTONOMIC NERVOUS SYSTEM IN DISSOCIATION

Patients with chronic PTSD cycle in and out of exaggerated levels of arousal and avoidance, of panic and numbing, and of terror and confusion. The panorama of autonomic

symptoms includes pallor and flushing; nausea, abdominal cramps and diarrhea; tachycardia and light-headedness; and diaphoresis and shivering. The *DSM-IV* criteria for PTSD (arousal, reexperiencing, avoidance) reflect the dramatic cycling of mood from panic, hypervigilance, and irritability, to numbing, withdrawal, and flattened affect. Physiologic markers of PTSD referenced in the *DSM-IV* include measurements of pulse rate and electromyographic and electrodermal responses, all primarily measures of sympathetic tone. The role of the cyclical increase in parasympathetic tone or function in trauma, however, has been largely neglected. PTSD is in fact a bipolar syndrome, one that reflects remarkable cyclical autonomic instability, with patterns of heightened sympathetic arousal alternating at times with clear and dramatic parasympathetic dominance.

Oscillatory phenomena in a variety of biological systems have been studied and documented in a number of settings. Many physiologic subsystems (endocrine, autonomic, neurohumoral) operate in a bimodal fashion based on a variety of rhythmic environmental and internal physiologic influences. Antelman et al. (1997) propose that exposure of such systems to chemical or behavioral stressors of sufficient intensity can induce cyclical patterns of increase and decrease in response to each subsequent exposure (Antelman & Caggiula, 1996; Antelman et al., 1996). This phenomenon seems to be applicable to such a variety of physiological systems that the authors conclude that oscillation in response to chemical or behavioral input may represent a general principle of biological functioning (Antelman et al., 1997). This may well be an innate biological reflex designed to reestablish homeostases, the rhythmic and balanced fluctuation of all biologic systems, be they endocrinologic, neurophysiologic, metabolic, or immunologic (Antelman et al., 1997).

In PTSD, through unresolved peritraumatic dissociation, internal and external stimuli impacting the central neural circuits mediating memory and arousal will contribute to kindling, leading to internally-based stressors of associated neural subsystems, especially the autonomic nervous system. By this model, cyclical autonomic dysfunction will result, leading to many of the divergent but dramatic autonomic symptoms of the traumatized victim. Thus periods of sympathetic arousal will include symptoms of muscle bracing, bruxism, ocular divergence, tachycardia, diaphoresis, pallor, tremor, startle, hypervigilance, panic, rage, and constipation. These will alternate with parasympathetic dominance, including symptoms of palpitations, nausea, dizziness, indigestion, abdominal cramps, diarrhea, and incontinence. Although many of these symptoms are often attributed to somatization disorder, they in fact represent the extremes of the cyclical autonomic dysfunction seen in trauma, are inherently self-perpetuating, and contribute to continued abnormal autonomic oscillation. The syndrome of trauma has now literally taken control of the body.

As the kindled cycle of PTSD continues and becomes chronic, avoidance and withdrawal become increasingly prominent, often with subsidence of symptoms of arousal, hypervigilance, and phobia. At this point, the *DSM-IV*-based criteria of PTSD no longer specifically justify the diagnosis, and patients are usually given diagnoses of somatization disorder, dissociative disorder, conversion, or depression. With time, the role of trauma in the patient's syndrome may be ignored. Although autonomic oscillation is still apparent, it is clear that the prevailing symptom complex reflects a state of parasympathetic dominance. Endocrinologic measures now tend to show a state of *low* serum cortisol (Mason, Giller, Kosten, Ostroff, & Podd, 1986; Yehuda et al., 1990), also commensurate with evolving parasympathetic tone. This trend is associated with behavioral responses including social isolation and withdrawal, substance abuse, constricted affect, denial, cognitive impairment, and dissociation, all relatively parasympathetic states.

Another compelling rationale for this process may be drawn from the phylogenetic role of the parasympathetic nervous system, specifically the vagal system, as presented in the Polyvagal Theory of Emotion by Porges (1995). Porges emphasizes the phylogenetic layering of arousal responses in mammals, based on the varied functions of the vagal nuclei. The dorsal vagal complex (DVC), composed of the dorsal motor nucleus of the vagus and nucleus tractus solitarius, is a vestigial and primitive center, primarily useful in reptiles for energy conservation. In the low oxygen-demand system of the reptile, the DVC shuts down the energy-use system by inducing marked bradycardia and apnea, as in the reptilian dive reflex. The ventral vagal complex (VVC), unique to mammals, is a recent adaptation to the high oxygen need of this class of animals, and finely tunes energy utilization by subtle and flexible influences on heart rate. The early alerting response seen in animals consists of raising the head from grazing, orienting with the head to the source of the new, potentially threatening stimulus, widening of palpebral fissures, and sniffing for scents. This energy-conserving reflex is mediated by the VVC, and employs the locus ceruleus, which has rich connections with sense organs of the head, as well as with the muscles of the head and neck. If sufficient information of threat is attained through this reflex, the VVC response is inhibited and the animal will progress to the neuromuscular and cardiovascular mechanisms of the epinephrine-based fight/flight response. If deterrence of the threat through defense or flight fails, the animal enters a state of helplessness, associated by a marked increase in DVC tone, initiating the freeze/immobility response. This state of deep parasympathetic tone is associated with marked bradycardia, apnea, sphincter relaxation, and gastrointestinal activation.

A persistent state of DVC activation, however, is common to reptiles, but in fact dangerous for mammals due to its association with marked bradycardia and life-threatening arrhythmias. The spontaneous death of wild animals during induced states of immobility in the laboratory setting attests to this danger, as does the remarkable mortality rate of wild mammals introduced to the zoo environment (Seligman, 1992). In humans, this state of immobility and “suspended animation” perhaps has its most extreme expression in the phenomenon of Voodoo death, as described by Cannon (1942). The study of death in the freeze/immobility response in animals reveals that death occurs by cardiac arrest during diastole, or relaxation of the heart, in a state of complete cardiac flaccidity and engorgement with blood (Hofer, 1970; Richter, 1957). The extremes of vagal parasympathetic tone as manifested in the state of DVC activation, therefore, contribute greatly to the generation of severe emotions, especially those of terror and helplessness. Although freeze/immobility states in mammals may be useful for short-term survival, prolongation or repeated activation of that state clearly has serious implications for health and long-term survival. The model of disease presented here suggests that the gradual descent into dissociation and parasympathetic dominance in chronic unresolved PTSD constitutes just such a state of peril.

SOMATIC DISSOCIATION

As suggested earlier, 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 clinical impairment experienced by the dissociated individual under those circumstances will almost always present as physical deficits that defy physiologic explanation by examination, laboratory tests, or imaging studies. Diagnoses entertained by physicians in these states

include hysteria, conversion, and psychosomatic disorders. The cause for these states is uniformly assumed to be psychological, and the common factor to be stress. Almost all of the deficits have a neurological nature, and may affect any system, including visual, auditory, vestibular, speech, balance, sensation, and motor function. Seizures and fainting are common expressions of this state.

Symptoms associated with conversion may appear to be exaggerated, and findings do not conform to those objectively seen in actual disease or injury of the nervous system. Thus, sensory loss usually presents in a “stocking/glove” distribution, rather than the layered dermatomal loss seen in lesions of the spinal cord. Weakness is diffuse and inconsistent, with a “give-away” quality. Symptoms are often one-sided, and findings may fluctuate in time, with ambient stress often enhancing the symptoms. Conversion symptoms occur more commonly in lower socioeconomic and less developed countries and cultures, and in women (APA, 1994). From patient to patient and culture to culture, however, the seemingly varied syndromes of conversion have a remarkably constant theme that demands consideration of a common and as yet undefined neurophysiologic mechanism.

The medical literature does not address this attempted crossover between psychological and physiological factors in conversion and related disorders. Concepts presented in this model are based on the evaluations of thousand of patients who have experienced physical trauma in motor vehicle and other types of accidents, and who, to a varying degree, have also manifested symptoms of having been traumatized as well. Many of these patients have presented with symptoms and signs of conversion, and with minute observation of their physical states and behavioral symptoms, several conclusions appear to be inescapable.

Patients with conversion in this setting seldom present with *la belle indifférence*, but rather exhibit early symptoms of arousal and distress consistent with PTSD. Their symptoms are remarkably common from patient to patient. Difficulties with speech elocution and mechanics are common, with word blocking, stutter, and unusual dysarthric patterns of speech. One-sided or one upper extremity sensory loss is almost universal, associated with severe problems with dexterity on the same side. This sensory loss is invariably “non-physiological,” often stocking and glove in distribution. Balance is impaired with variable swaying and staggering patterns not consistent with impairment of intrinsic brainstem balance centers. With careful observation, many such patients experience a physical sensation of arousal if the examiner presents a visual stimulus to them from a part of the room on the same side as their predominant nonphysiologic symptoms. This pattern of arousal is most commonly experienced as nausea and dizziness, and may be associated with flushing, suggesting the influence of VVC activation as part of the early response to threat.

The concept of peripheral perceptual boundaries in psychological terms relates primarily to subtle areas of our sense of self that we perceive in relationship to others, the regions of appropriate limitations in personal and social interaction. In the model of somatic dissociation presented here, this concept of boundaries relates to an actual physical perceptual whole, or continuity of self, that represents the limits of the unconscious but perceived area defining the safe extent of our physical expression. The area comprising this space is directly proportional to the experience of previously unresolved life threats, and the continuity of the perceptual boundaries surrounding this space is dependant on the perceptual experience of severe threat within a specific boundary sector. Findings in testing the boundaries of a traumatized patient reveals that the area of a person’s perception where they first experienced the warning of the impending threat (e.g., the approaching automobile)

will thereafter be an area where accessing *any* stimulus is intrinsically threatening. As a result, passing a hand around the periphery of that person's visual field at the distance of 3–4 feet will often produce an arousal response in the region of perception of prior threat. Such patients have developed a conditioned arousal reflex within areas of their perceptual surround, or boundary. Predictably, persistent ambient subliminal sensory perceptual experiences within that region, whether visual, tactile, or proprioceptive in nature, will result in conditioned arousal and will perpetuate the kindled trauma reflex. Just as a chronic victim of PTSD will freeze or dissociate in the face of familiar threat, the part or region of the body representing the proprioceptive and somatic procedural memory for the threat experience will be selectively dissociated, leading to the nonphysiological signs of conversion. It will come as no surprise, therefore, that many patients with localized signs of conversion will experience symptoms of discomfort and arousal with presentation of visual or other seemingly benign stimuli within those regions of their boundary perception that now possess the sensory perception of threat.

In addition, with further close observation of such patients, one may detect unusual but reproducible physical changes in the dissociated portions of their body. My awareness of these physical phenomena began when one patient with "hysterical" right sided hemianesthesia, weakness, and clumsiness related that her hairdresser had noted that her hair grew much more slowly on the right side, and was of a different texture. Close observation revealed that, in addition, her hair was more sparse on the right side of her head. Examination of the patient's right hand and arm then revealed that her fingernails were broken and ridged, the hand was cooler than that on the left, and finger hair growth was diminished. Close observation of other similar patients subsequently documented signs of dystrophic skin, hair, and nail changes in many patients in parts of the body manifesting signs of conversion. Finally, several of these patients proceeded to develop clear-cut signs of sympathetically maintained pain, or reflex sympathetic dystrophy (RSD).

REFLEX SYMPATHETIC DYSTROPHY

Sympathetically maintained pain, complex regional pain syndrome, and RSD comprise fairly common, well recognized but controversial and poorly understood pain syndromes, by definition associated with vasomotor autonomic symptoms and signs in the affected body parts. The extremities, especially their distal portions, are predominantly affected. Described by S. Wier Mitchell in the Civil War, the syndrome perhaps is most common in traumatic injuries of the extremities, but also may follow seemingly trivial injuries such as minor bruises or overuse injuries (Mitchell, Morehouse, & Deen, 1864; Schwartzman & McLellan, 1987). The syndrome is characterized by severe, often burning pain in the affected area, associated with variable signs of vasomotor dysfunction, both parasympathetic and sympathetic. These signs may include abnormal hair growth or loss, erythema and warmth, or pallor and coolness. With unsuccessful treatment and progression of the syndrome, signs of vasoconstriction and dystrophy predominate, hence to term sympathetic. Attribution of the syndrome to abnormal sympathetic autonomic tone is supported at least in part by the fact that the injection of related ganglia of the sympathetic nervous system may provide variable relief of pain. Many investigators feel that the central nervous system may be involved. Dystonic postures of the affected limbs are common. Electromyographic and

nerve conduction studies of RSD reveal that the character of this dystonia is more typical of *voluntary* holding of the posture than of comparable dystonias in patients with brain lesions (Koelman, Hilgevoord, Bour, Speelman, & Ongerboer, 1999). The authors go so far as to say, "In causalgia-dystonia, central motor control may be altered by a trauma in such a way that the affected limb is dissociated from normal regulatory mechanisms" (Koelman et al., 1999, p. 2198).

The model presented here proposes that regional somatic dissociation exposes the dissociated member or region of the body to selective vulnerability to the effects of existing cyclical and oscillatory autonomic dysfunction associated with the neurophysiologic changes of unresolved trauma. In this state, that region or part may then be vulnerable to vasomotor oscillation, with vasoconstriction and functional reduction in blood flow ultimately creating the ischemic tissue pathology characteristic of RSD. This syndrome, as often is the case, comprises a continuum, or spectrum of clinical expression, from the subtle signs seen in most patients, to the full-blown pain, dystrophic changes, and dystonia of RSD. Dissociation, by this model, is a neurophysiologic 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 of 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. This procedural memory acquisition initially is elicited by internal and external cue-specific stimuli, but because the threat itself has not been resolved, internal cues persist without inhibition from external messages of safety, and kindling is triggered in the cortical, limbic, and brainstem centers previously discussed. Recurrent dissociation in response to arousal accompanies this cycle and facilitates the development of pathologic autonomic oscillation. Physiologic inhibition of perception of those parts or regions of the body for which the brain holds procedural memory of their sensory input at the time of the threat results in the syndrome of conversion and regional somatic dissociation. Divorced from the normal trophic benefits of cerebral perception, these regions are subject to the extremes of vasomotor instability of late trauma and develop syndromes of pathologic vasoconstriction and ischemia.

THE DISEASES OF TRAUMA

Selye (1936) has generally been credited with the concept that prolonged or excessive exposure to stress could contribute to the development of a group of specific diseases. These diseases predominantly reflected exposure to elevated levels of adrenal cortical hormones as part of the modulating role of cortisol on the hypothalamic/pituitary/adrenal (HPA) axis in stress. Thus rats exposed to prolonged and excessive stress developed erosion of the gastric mucosa, atherosclerosis, and adrenal cortical atrophy. Other specific pathologic effects of excessive cortisol exposure include immune suppression, elevated serum lipids and atherosclerosis, diabetes, osteoporosis, hypertension, peptic ulcer disease, obesity, and cognitive/emotional impairment. Many of these effects are now well described in the medical and lay literature as "diseases of stress."

The relationship of the long-term effects of trauma (as opposed to stress) and disease are less well documented. Whereas ongoing stress is easily identified, the past experience

of traumatization is masked by the evolution of the resulting syndrome into experiences, symptoms, and behaviors that ultimately are attributed to characterological and psychological causes that is, that are because of internal rather than external events. This perception is basically correct in that the internal events in trauma are self-driven and capable of changing somatic physiology in the absence of external influences. This concept is also in keeping with the physiologic effects of somatic dissociation, which are driven by internal brain-based mechanisms that are self-perpetuating. Therefore, one would not expect the diseases of trauma to reflect the generally cortisol-based syndromes of acute and even chronic exogenous stress. Rather, one would predict that diseases of trauma would reflect autonomic regulatory impairment, both sympathetic and parasympathetic, with a predominance of vagal and parasympathetic syndromes in the later stages.

The model of diseases in trauma would predict that vasomotor symptoms and signs would be likely, with both trophic and dystrophic components, the latter reflecting vasoconstriction and ischemia. Cardiac, pulmonary, bowel, and exocrine gland dysfunction should be predictable. Abnormalities of strength, muscle tone, and endurance should be common. Lowering of serum cortisol in late stages of trauma might lead to a relative lack of immune inhibition, and therefore to hyperimmune syndromes. One would also expect these syndromes in some cases to manifest remarkable periods of exacerbation and remission based on autonomic oscillation, and to be specifically sensitive to exacerbation by external stress. Fluctuating symptoms of cognitive impairment especially related to attention and memory would be common in many of these conditions. One would expect an unusual association of the emotional symptoms of late trauma, including affect dysregulation, dissociation, somatization, depression, hypervigilance, and denial/avoidance. A psychosocial trauma history in many cases might reflect a history of substantial life trauma, especially in childhood.

Among other manifestations, these diseases would at least in part show evidence of abnormal parasympathetic tone, perhaps along with sympathetic vasoconstrictive dystrophic and ulcerative phenomena. Diseases and syndromes of the gastrointestinal system that fall into this general concept of diseases of trauma include peptic ulcer and gastroesophageal reflux disease, irritable bowel syndrome, Crohn's disease (regional ileitis), and ulcerative colitis. All reflect organ hypermotility, excessive glandular secretion, and in some, ulcerative features. Cardiac syndromes would likely reflect the cardiac abnormalities associated with DVC dominance, and be associated with a variety of tachy- and bradyarrhythmias, including those seen in mitral valve prolapse. Bronchial asthma, a syndrome primarily manifested by stress and hyperimmune-induced abnormal organ-specific parasympathetic events (bronchospasm and hypersecretion), has many of the criteria predictable in diseases of trauma. Interstitial cystitis is a condition characterized by pain, spasm, and ulceration of the bladder wall, combining the parasympathetic/dystrophic elements of many of these syndromes.

One of the most perplexing and controversial chronic syndromes that may fall into this category is that of fibromyalgia/chronic fatigue. Protean symptoms include diffuse and severe musculoskeletal pain, impaired and nonrestorative sleep with chronic fatigue, stiffness, headaches, anxiety, hypervigilance, cognitive impairment, ocular and vestibular symptoms, and paresthesias. Associated syndromes include irritable bowel syndrome, interstitial cystitis, mitral prolapse, and esophageal dysfunction (Clauw, 1995). Low serum cortisol and HPA axis dysfunction similar to that in late PTSD has been documented (Crofford, 1996). Fibromyalgia syndrome primarily affects women, and controversial but suggestive evidence for an increased incidence of childhood sexual and physical trauma in fibromyalgia patients

has been documented (Boisset-Pioro, Esdaile, & Fitzcharles, 1995). Fibromyalgia arising *de novo* from a traumatic experience has been well documented (Waylonis & Perkins, 1994). While recognizing that overwhelming circumstantial evidence does not constitute medical scientific proof, fibromyalgia/chronic fatigue syndrome appears to present a prototypic syndrome for the model of the diseases of autonomic dysfunction seen in late trauma.

The rationale for RSD as a dissociative/autonomic posttraumatic disease has been presented. Chronic pain in instances where documented structural pathology is not apparent very likely represents another syndrome of late trauma. Phantom limb pain appears to represent the prototype for this model. This syndrome occurs much more commonly when the amputation was associated with a traumatic injury. Persisting representation of pain in an absent organ or body part suggests procedural memory for that pain as a conditioned response. The critical element for that memory to be conditioned, of course, is the unresolved threat associated with the injury producing the pain itself. One must remember that severe pain itself may be traumatizing, and that the medical system in which that pain was managed has many potential sources of traumatic stress (Scaer, in press, Chap. 9). Conditioned imprinting of pain in procedural memory of course implies that trauma will have occurred in a state of helplessness without opportunity for spontaneous resolution of a freeze response. Under those circumstances, the specific pain will continue to represent the threat and be retained for late survival purposes in conditioned procedural memory.

An underlying state of vulnerability to traumatization would also be a predictable substrate for the development of chronic pain in the injured individual. Victims of child abuse or multiple prior traumatic events clearly possess this vulnerability, and would be predicted to be susceptible to the incorporation of a newly painful experience into procedural memory in the model being described. The trauma literature amply documents the high incidence of chronic pain of many types seen in victims of child abuse relative to the general population. Types of pain represented in these studies include pelvic, back, abdominal, head, orofacial, and chronic pain in general. Also documented is the extremely high incidence of childhood abuse in patients referred to centers for chronic pain treatment (Rapkin, Kames, Darke, Stampler, & Nabiloff, 1990; Wurtele, Kaplan, & Keairnes, 1990; Toomey, Hernandez, Gittelman, & Hulka, 1993; Walling et al., 1994a, 1994b).

A number of studies and proposed models of disease suggest that trauma may in part contribute to autoimmune diseases. As noted, the lower serum cortisol documented in late PTSD might be related to increased immune activities *in vivo* (Yehuda et al., 1993). Indeed, Watson et al. (1993) have documented increased reactivity of skin to antigens in combat-related PTSD (Watson et al., 1993). Other authors have specifically proposed that the low cortisol state of late PTSD might well present the substrate for a hyperimmune state (Friedman & Schnurr, 1995, p. 518). More recently, the ratio of lymphocytic phenotypes documented in victims of childhood sexual abuse with PTSD showed a pattern indicative of lymphocytic activation. This finding supports the likelihood of increased immune activity in these patients, suggesting the potential for a hyperimmune state in late PTSD (Watson, van der Kolk, Burbridge, Fislser, & Kradin, 1999). Supported by an exhaustive literature review, Rothschild and Masi present a strong argument for a vascular hypothesis for rheumatoid arthritis (RA) incorporating as a cardinal feature vasoconstriction and tissue hypoxia, both of which have been well documented in RA. Hypoxia of the arteriolar wall leads to vascular permeability with release of antigens into surrounding tissues. The resulting immune response, therefore, represents a relatively secondary feature of RA

(Rothschild & Masi, 1982). This theory, supported by an extensive array of studies, is in keeping with the model of autonomic and vasomotor dysfunction presented previously as a template for trauma-related disease processes. Such findings by no means provide a specific link between prior trauma and autoimmune diseases, but suggest an avenue for further investigation of a possible relationship between trauma and autoimmune processes.

Perhaps a more compelling and immediate avenue for investigation relates to the studies of morbidity and mortality in trauma. Victims of trauma have long been known to experience increased morbidity and mortality rates (Friedman & Schnurr, 1995). Many of these studies focus on the late health problems of former prisoners of war (POW's; Beebe, 1975; Page, 1992). Cardiovascular and gastrointestinal diseases predominate in this group of late trauma victims, although emotional sequelae related to depression and cirrhosis of the liver associated with alcoholism contributed significantly to mortality. When the diagnosis of PTSD is added to the equation, however, the health effects of trauma are noted to increase substantially, with the cardiovascular diseases predominating (Friedman & Schnurr, 1995; Wolff, Schnurr, Brown, & Furey, 1994).

From the early morbidity intrinsic to Selye's model of stress, to the late effects of autonomic dysregulation, and pathologic vagal dominance, the potential and real health effects of trauma are clear. Unfortunately, the trauma necessary to place the individual at risk may be as subtle as adverse childhood experiences. Felitti et al. (1998) found a strong graded relationship between the breadth of exposure to abuse or family dysfunction during childhood and multiple risk factors for several of the leading causes of death in adults (Felitti et al., 1998). Adult diseases that were endemic in those who had experienced childhood abuse or family dysfunction included ischemic heart disease, cancer, chronic lung disease, skeletal fractures, obesity, and liver disease. Additional diseases attributable to risk exposure and behavior included sexually transmitted disease, alcoholism, drug abuse, depression, and suicide. This study is particularly disturbing in that it shows that the "trauma" of childhood in these high-morbidity cases was often as indirect as living with family members who were mentally ill or substance abusers. The sensitivity and vulnerability of the developing child to a loss of nurturing and safe boundary structure, and the adverse effects of this loss throughout life on emotional and physical health appear to be frighteningly clear.

CONCLUSIONS

We have presented a model of altered brain function precipitated by a traumatic event whose completion or resolution was truncated or aborted by lack of spontaneous resolution of a freeze/immobility response, a phenomenon closely allied to the clinical psychological state of dissociation. In addition to the arbitrary psychiatric diagnosis of PTSD, this state is associated with a complex set of somatic pathologic events characterized by cyclical autonomic dysregulation and an evolving state of vagal dominance involving primarily the dorsal vagal nucleus. The sympathetic portion of this cyclical physiologic complex primarily involves vasoconstriction, with dystrophic and ischemic regional changes, especially in regions of the body that have been subject to dissociation due to their residual representation of sensory messages of threat stored in procedural memory. The experimental model of kindling is intrinsic to the self-perpetuation of this pathologic process, driven by internal cues derived from unresolved procedural memory of threat, and enhanced by

endorphinergic mechanisms inherent to both the initial response to threat and to subsequent freeze/dissociation.

In this context, a variety of chronic diseases are postulated to represent late somatic expressions of traumatic stress. These diseases are of remarkably varied expression, but with a common thread of autonomic cyclical instability, frequently subtle vasoconstrictive/ischemic features, and usually pain. They are generally distinct from those diseases frequently attributed to stress, although these “stress-related” diseases often occur simultaneously and are definitely also more frequent in the adult population of those persons who have experienced trauma.

This model rejects the concept that the terms “somatization,” “conversion,” “hysterical,” “psychological,” or “psychosomatic” have any viable meaning in the definition of a symptom complex or disease state. It places all of these terms in a pathologic somatic context associated with subtle, but definable and objective clinical findings and manifestations of disease. It moves beyond the concept of mind/body medicine to the concept of a mind/brain/body continuum.

By attempting to isolate psychosomatic disease processes into a distinct category, we are perhaps ignoring the major cause for the group of diseases that members of the healing professions probably understand the least, and treat the most ineffectively—chronic diseases of unknown cause. Many of these diseases are because of impairment of regulation, rather than because of the invasion of microbes, toxins, or other extrinsic agents. As such, they present a unique opportunity for those practitioners, researchers, and teachers in the area of applied psychophysiology and biofeedback who have dealt with concepts of self-regulation and healing for the past 40 years. If one accepts the concepts of myofascial pain, visceral dysfunction, chronic pain, and systemic diseases such as fibromyalgia presented above, it quickly becomes apparent that biofeedback practitioners have been treating symptoms and conditions primarily driven by past trauma in most of their patients. Not surprisingly, their techniques have often been more effective than polypharmacy and many medical/surgical techniques. Application of advanced techniques such as cerebral regulation through neurofeedback and autonomic regulation through control of heart rate variability (HRV) may have profound implications for healing trauma by providing a unique means of access to the conditioned autonomic responses that drive the trauma reflex.

Finally, as clinicians, we must look beyond the dysfunctional behavior apparent in many of these patients, to the neurophysiologic and autonomic dysregulation that is the source of their symptoms and eventually their disease. Medical science must shed the concept that a symptom not measurable by current technology is “psychological,” and therefore invalid. And physicians must reject the pejorative implications of the term somatization, and stop further traumatization of patients by subtly implied rejection.

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