

OBSERVATIONS ON TRAUMATIC STRESS UTILIZING THE MODEL OF THE "WHIPLASH SYNDROME"

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In his book, Affect Regulation and the Origin of the Self Allen Schore has outlined in exquisite detail the psychobiology of early childhood development involving maturation of orbitofrontal and limbic structures based on reciprocal experiences with the care giver. (14). Dysfunctional associations in this dyadic relationship result in permanent physicochemical and anatomical changes which have implications for personality development as well as for a wide variety of clinical manifestations. An intimate relationship may exist, with negative child/care giver interaction leading to a state of persisting hypertonicity of the sympathetic and parasympathetic systems that may profoundly affect the arousal state of the developing child. Sustained hyperarousal in these children may markedly affect behavioral and characterological development.

This phenomenon has its correlation in the adult traumatic experience and its effect on the autonomic nervous system. Although this effect has been described extensively in Viet Nam veterans and other groups of traumatized individuals, one of the more fascinating models of the physiology of the traumatic experience had been developed by Peter Levine, PhD., (7,8) based on the ethological model of the fight/flight/freeze response seen in animals in response to life-threatening experiences. In the wild, the preyed-upon animal will flee or attempt to fight, but if trapped, will enter a freeze response where it assumes a state of immobility while physiologically still manifesting high levels of activity of both parasympathetic and sympathetic nervous systems. If the animal survives the attack, it will go through a period of discharge of this high level automatic arousal through the motor system involving trembling, running movements, shaking, diaphoresis and deep breathing. Following this, the animal will return to its prior state of calm alertness. Interestingly, game keepers in Africa interviewed by Levine who capture animals for examination or tagging routinely note that if the animal does not go through the shaking/breathing response after release, they will inevitably die in the wild, possibly due to the inability to initiate appropriate self-protective behavior.

Presumably due to acculturation or neocortical inhibition, the human species frequently will not discharge this high state of autonomic arousal after a freeze response in the face of perceived trauma, but will suppress the discharge phenomenon, resulting in storage of a high state of autonomic arousal, probably in limbic and procedural memory systems of the brain. Memory mechanisms in trauma probably involve both explicit (conscious, declarative), and implicit (unconscious, non-declarative) memory. Procedural memory is a form of implicit memory involving learned sequences of synchronized motor acts such as athletic, musical or artistic skills. Once learned, these motor sequences are stored with a high degree of recoverability, probably in orbitofrontal and limbic, as well as cerebellar, vestibular and basal ganglia connections of the brain. There is strong evidence that memories of the motor sequences of a traumatic experience may well be stored in this memory system. Levine has found that by accessing the "felt sense" through therapeutic guidance, the individual may then access complex patterns of movement representative of prior traumatic experiences, may activate the sympathetic nervous system, and lead to a discharge of retained autonomic energy through the somatic/motor system analogous to that which occurs with resolution of the freeze response in animals. This may be used as a therapeutic technique for desensitizing or deconditioning the autonomic nervous system and reducing the symptomatic load of autonomic arousal which causes many of the symptoms of post-traumatic stress disorder.

The clinical implications of the trauma syndrome with regard to both emotional and physical symptoms are substantial. There is strong evidence that traumatic experiences are cumulative, with sequential elevation of the arousal set of the autonomic nervous system with accumulated unresolved trauma. It is well known that a substantial number of chronic pain patients have a history of child abuse or multiple traumatic experiences. Childhood trauma has been linked to a variety of clinical syndromes including chronic pain, fibromyalgia, premenstrual syndrome, functional bowel syndrome, and chronic pelvic pain (2,5,16,17). The acute symptoms attributable to post-traumatic stress disorder (hyperarousal, hypervigilance, intrusive imagery and flashbacks, stimulus hypersensitivity, exaggerated startle, nightmares and sleep disturbance, panic attacks, anxiety and phobias) are only a part of the post-traumatic syndrome. Depression, emotional constriction, mood swings, substantial cognitive dysfunction,

chronic sleep disturbance, dissociative reactions and schizoid social behavior are all typical of the later manifestations of this syndrome.

I have studied the phenomenon of trauma extensively in motor vehicle accidents (MVA) as part of the "Whiplash Syndrome". This syndrome has been exhaustively documented and studied, but its severity in relationship to often trivial MVA trauma has never been adequately understood (4). The full blown syndrome consists of intractable but variable regional myofascial pain, usually involving the head, neck and shoulder girdle, associated with dental bruxing and often temporomandibular joint syndrome. Associated postural dysfunction may lead to what has been called myofascial or pseudothoracic outlet syndrome characterized by fluctuating distal paresthesias of the arm and hand, often with edema and coolness of the hand. Neurologic manifestations include binocular visual disturbance, particularly convergence insufficiency and impairment of binocular vision, unremitting tinnitus, atypical positional vertigo and balance disturbance, facial paresthesias, and autonomic dysfunction including orthostatic hypotension. Atypical vascular headaches with both vasodilatory and vasoconstrictive features similar to migraine are also common, consistent with a state of autonomic instability.

Cognitive dysfunction usually is characterized by deficits in sustained attention span and multitask thinking, deficits in short-term memory, and marked cognitive fatigability (6). Additional deficits often include word-finding difficulties, word blocking and reversals, and occasional neologisms. Higher level executive functioning may also be impaired in some cases.

Emotional symptoms typically involve the spectrum of symptoms of post-traumatic stress disorder (PTSD) described above, most specifically phobias of driving, anxiety and panic attacks, intrusive memories of the accident trauma, and sleep disturbance with repetitive awakening, sometimes with nightmares, often of helplessness.

In general, chronic upper quadrant pain has been attributed to "cervical ligamentous strain or segmental vertebral dysfunction". Neurologic symptoms and cognitive dysfunction have been routinely attributed to minor traumatic brain injury whereas symptoms of hyperarousal, anxiety and sleep disturbance have been diagnosed as PTSD.

A dilemma in the "Whiplash Syndrome" is that the symptoms as described above commonly far exceed what could reasonably be explained by the velocity of the accident or the severity of predictable trauma to the head, neck and brain tissues. Indeed, full blown syndromes have been frequently seen in low velocity MVA trauma below 10 to 15 mph. This fact has led many clinical observers to attribute much of the "Whiplash Syndrome" to psychological factors - or issues of secondary gain.

If carefully interviewed, most victims of even relatively minor MVA's will vividly describe a sense of detachment and shock as the first sensations experienced immediately after the impact. Frequently this will subside, and may be associated with subsequent development of increasing neck stiffness and pain. More often, however, the major symptoms of the "Whiplash Syndrome" do not appear for twenty- four to forty-eight hours, and may then progressively worsen for many days, weeks and even months despite extensive care. Clearly this is not in keeping with the natural course of events of either cervical ligamentous or brain injury. On the other hand, the truly remarkable consistency of symptoms among such MVA victims demands explanation and qualifies this injury as a valid syndrome. Based on the work of Schore and Levine, and these observations, I would like to propose that the entire spectrum of symptoms of the "Whiplash Syndrome", especially in low velocity MVA's is explainable by the effect of the traumatic experience on the autonomic and procedural memory portions of the central nervous system. The physiological basis for the permanent imprinting of this traumatic event on the central nervous system exists in the persistence of the unresolved and undissipated freeze response, itself initiated at the moment of impact, but thereafter locked in the autonomic nervous system because of the absence of discharge through the neuromuscular system. Each of the components of the syndrome may be analyzed and explained by this mechanism.

The neuromuscular manifestations of the traumatic event are characterized by activation of a sequence of coordinated muscular contractions designed to protect the body from harm. In the case of a MVA, this will involve a bracing pattern to protect portions of the body from impact. As a result of the freeze response, this bracing pattern is maintained indefinitely in procedural memory and is manifested by

sustained muscular contraction of muscle groups, usually in the neck and shoulder girdle, leading to the syndrome of regional myofascial pain. The typical postural dysfunction seen in these individuals consists of a head forward position associated with protraction and elevation of the shoulder girdle. This represents in many animal species the position of protection against threat. Bruxing and clenching of the teeth is a typical neuromuscular pattern associated with threat in most animal species and eventually leads to what is currently described as the temporomandibular joint syndrome. Constriction of the thoracic outlet by the posture described above often leads to changes in hand sensation and temperature producing a syndrome of variable venous obstruction described as thoracic outlet syndrome.

The alerting response to threat is also associated with pupillary dilatation and divergent positioning of the eyes for the purpose of optimally scanning the field of vision. Locking in this neuromuscular response as part of the freeze response leads to a chronic visual syndrome characterized by binocular visual dysfunction and especially convergence insufficiency. The dramatic diminution in cervical range of motion seen in whiplash patients resulting from myofascial pain leads to impairment of head movement in the face of gravitational input from the vestibular system, contributing to the syndrome of cervical vertigo and balance disturbance.

Although cognitive impairment noted in the "Whiplash Syndrome" may at times be attributable to minor traumatic brain injury, in many cases it may exist in accidents of a velocity insufficient to explain axonal injury or neurotransmitter dysfunction. In addition, cognitive dysfunction may not appear for weeks or even months after the initial injury, a phenomenon inconsistent with reasonable models of minor traumatic brain injury. In many cases, measured cognitive impairment correlates with severity of symptoms of post-traumatic stress disorder and/or depression. The distracting effect of intrusion on the declarative portion of the memory system by unconscious traumatic memories from non-declarative memory systems may well explain what amounts to an attention deficit disorder in traumatized whiplash patients. The sustained state of abnormal arousal in PTSD includes intrusive thoughts related to the trauma triggered by life events related to the traumatic experience. Generalization of the arousal response also develops, leading to intrusion of traumatic memories by any stressful stimulus. Resulting low level interruption of attention leads to impaired acquisition of incidental information and short term memory encoding, resulting in the typical short term memory deficits seen in PTSD. The phenomenon of dissociation as part of the traumatic experience also is a potentially potent source of interference with sustained attention in such patients. Such interference phenomena as part of the traumatic experience may well explain cognitive dysfunction seen in many cases of "Whiplash Syndrome".

The clinical syndrome of PTSD constitutes a psychiatric syndrome described in the DSM-W. The entire constellation of symptoms attributable to this syndrome, however, I believe is explainable by both the sustained arousal and over-reactivity of the autonomic nervous system and by the procedural memory mechanisms activated in the traumatic experience. Stimulus hypersensitivity and symptoms of anxiety and panic are directly due to the over-responsiveness of the sympathetic nervous system to even minor stressful stimuli (3,11,15). This has been well documented in Vietnam veterans, both in exaggerated blood pressure and pulse rate responses to mild startle stimuli as well as to documented over-production of norepinephrine in response to stressful visual imagery or auditory stimulation (1,3,15). Recurrent stereotyped nightmares, intrusive accident-related thoughts, and phobic responses constitute reactivation of traumatic memory. Interestingly, these tend to fatigue and disappear earlier than the sympathetic arousal symptoms noted above. With time, many of the acute symptoms of PTSD may resolve, but usually then are associated with the onset of significant depression, commonly with a variable state of relative dissociation. These constrictive and dissociative emotional phenomena may constitute a sustained state of autonomic instability, combined with significant endorphinergic input. Endocrinologic sequelae of trauma maybe initially associated with increased cortisol secretion. Studies of urinary free cortisol in the later phases of PTSD demonstrate lowered cortisol levels, and may represent the late constrictive stages in PTSD. (12).

Perhaps the strongest support for this hypothesis is found in the fact that many of the components of the "Whiplash Syndrome" - myofascial pain, cognitive and emotional dysfunction - disappear at least in part with resolution of the freeze response through the process of Somatic Experiencing. The end point of this therapy involves activation of neuromuscular and autonomic portions of the brain, eliciting a behavioral experience quite similar to the dissipation of the freeze response seen in animals. With successful treatment, regional myofascial pain diminishes or disappears, the defensive posture of flexion relaxes

into a more appropriate upright stance, sympathetic arousal diminishes along with symptoms of PTSD, and cognitive function improves.

Finally, one facet of the epidemiology of MVA trauma also provides strong support for the origin of the "Whiplash Syndrome" in sustained autonomic arousal. In an ongoing review of my patients with delayed recovery (over 6 months) from whiplash, the first 50 patients manifested a 70% incidence of prior major life traumatic events and a 26% incidence of severe child abuse. Preliminary results in MVA's below 15mph suggests that an even higher percentage have experienced major child abuse (molestation, incestation, physical or psychological abuse by an alcoholic parent). Although these numbers appear startling, they are probably in keeping with prior estimates of the prevalence of abuse in our society. Studies in PTSD also would suggest that any form of traumatic stress (natural disasters, medical/surgical trauma, criminal victimization, impaired maternal-child bonding (14)) may be a source of sustained autonomic alteration. The cumulative effect of traumatic experiences on the autonomic nervous system appears to be a powerful source of potentiation of the post-traumatic response (9,10).

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