

WHIPLASH @ KINDLING

Robert C. Scaer, M.D.

- [INTRODUCTION](#)
- [THE BIODYNAMICS OF WHIPLASH](#)
- [POSTTRAUMATIC STRESS DISORDER](#)
- [DISSOCIATION AND THE FREEZE RESPONSE](#)
- [PTSD, MEMORY AND KINDLING](#)
- [MYOFASCIAL PAIN](#)
- [MINOR TRAUMATIC BRAIN INJURY](#)
- [POST CONCUSSION SYNDROME](#)
- [CONCLUSIONS](#)
- [BIBLIOGRAPHY](#)

INTRODUCTION

Recognized for over 100 years, criticized, analyzed, debated, defended, debunked and generally misunderstood, the complex group of symptoms that follow velocity-related injuries in vehicles of conveyance continues to defy medical scientific explanation. With the advent of the railroad in the 19th century, a group of symptoms of chronic neck and back pain associated with severe emotional disturbance was described in train accidents. The syndrome was generally called "Railroad Spine", and was placed in the category of psychoneuroses (Trimble, 1981). With the invention of the automobile, reports of persisting cervical pain after motor vehicle accidents [MVA's] began to appear as early as 1919 (Marshall, 1919). Davis popularized the term "whiplash" in an article on cervical injuries in 1945 (Davis, 1945). In 1953, Gay and Abbott described the condition of "whiplash" in detail. They clearly stated the dilemma that has continued to plague physicians, patients and insurance companies: "Characteristically, these patients were more disabled and remained handicapped for longer periods than was anticipated, considering the mild character of the accident" (Gay & Abbott, 1953). Severy further amplified this dilemma in his 1955 study, observing: "...unlike most injury-producing accidents, there is generally no visible sign of injury for the rear-end collision victim." (Severy, Mathewson & Bechtol, 1955). The apparent discrepancy between the severity of symptoms and the trauma causing them has remained the core of the controversy. Testing the forces of "perturbations of daily living", such as plopping into a chair, slapping on the back and jumping off a step, has demonstrated forces comparable to a low velocity MVA (Allen, Weir-Jones, Eng, Motiuk, Flewin, Goring, Kobetitch & Broadhurst, 1994). Compensation neurosis or other emotional causes have been blamed for whiplash related symptoms based on

their remarkable diversity (Bosworth, 1959). Indeed, patients suffering from even a minor to moderate velocity rear end MVA may suffer from a confusing variety of symptoms. Not only do they have the typical complaints of head pain and neck stiffness, they also often complain of anxiety, phobias, sleep disturbance and depression. Neurological complaints are common, ranging from dizziness and vertigo, blurred vision, fainting spells and balance difficulties, to remarkable problems with cognition, especially with concentration and memory. Rather than making a steady recovery as with a comparable sports-related accident, a select group of whiplash patients tend to pursue a slow, unpredictable course, often taking several years to improve, with episodic periods of worsening. Long term studies in whiplash patients in general show that a majority (70-80%) had returned to normal activities in 6 months (Radanov, DiStefano, Schindrig & Ballinari, 1991, Balla, 1988, Pearce, 1989). On the other hand, in other studies, persistent chronic pain has been noted in 18% at 3 years (Friedmann, Marin & Padula, 1992), and up to 40% at 8-10 years (Packard & Ham, 1994). It therefore is not surprising that certain segments of the insurance, legal and medical communities express skepticism concerning the validity of many whiplash symptoms.

THE BIODYNAMICS OF WHIPLASH

Numerous biomechanical laboratory studies of whiplash suggest that the backward/forward movement of the head on the neck is probably less than has been thought, and that horizontal translation of the skull on the neck is a prominent movement. The initial movement actually is chin flexion due to relative early backward movement of the torso (Severy et al, 1955, McNab, 1971, Luo, & Goldsmith, 1991, McConnell, Howard, Guzman, Bornar, Raddin, Benedict, Smith & Hatsell, 1993). These studies have also shown that the forces on the head itself are 3-4 times greater than on the body with a sudden change in velocity, due to the pendular effect produced by the weight of the head supported by the stalk-like neck. The diagnosis most commonly given to the neck pain after whiplash is "cervical strain". Stretching and tearing of the ligaments of the jaw joints and the facet joints of the cervical spine, damage and rupture of the intervertebral discs and compression of cervical nerve roots have all been attributed to these forces (Davis, Teresi, Bradly, Ziemba, & Bloze, 1991, Weinberg & LaPointe, 1987). Thoracic outlet syndrome has been attributed to myofascial postural dysfunction (Sucher, 1990). Acute cervical disc syndrome with radiculopathy, however, is a rare occurrence after whiplash (Wickstrom, Martinez, & Rodriguez, 1967). The cognitive disturbance seen in many whiplash cases has been attributed to axonal shearing by some authors. (Ommaya, Faas & Yarnell, 1968). Vestibular symptoms have been attributed to disruption of middle ear conduction mechanisms or otolith displacement (Ward, 1969). Visual blurring associated with binocular dysfunction and convergence insufficiency has been attributed to brain injury in some cases involving whiplash. (Roy, 1961).

The entire spectrum of injuries attributed to whiplash has been based on theories dependent on at least a minimum force applied to the body. At exposure to change of velocity as low as 5 MPH, however, a small cluster of patients present with the full-blown syndrome described above, making a justification for physical injury as a basis for the symptoms extremely challenging. On the other hand, the remarkably similar cluster of symptoms among these patients qualifies whiplash as a syndrome, and begs for the definition of a coherent and specific etiology.

POSTTRAUMATIC STRESS DISORDER

The incidence and implications of posttraumatic stress disorder [PTSD] in MVA's has been relatively unappreciated until the past decade. An epidemiological study by Norris in 1992, however, suggested that MVA's were perhaps the single most significant event as a source of traumatic stress in our society (Norris, 1992). More recent studies of incidence of PTSD in populations suffering minor traumatic brain injury (MTBI) in MVA's estimate the frequency of PTSD to be between 17% and 35% (Layton & Wardi-Zonna, 1995, Middleboe, Anderson, Birket-Smith & Fiis, 1991, Ohry, Solomon & Rattock, 1996, Rattock & Ross, 1993). A large study by Blanchard and Hickling in 1996 has contributed greatly to the identification of the substantial role that PTSD plays in post-MVA morbidity (Blanchard & Hickling, 1996). Comparison of the epidemiological literature, they found, has been difficult, in part due to the changes in criteria for PTSD between the Diagnostic and Statistical Manual of Mental Disorders, editions 3, 3 revised and 4. Combining these criteria and defining a subsyndromal form of PTSD for MVA's, they estimated an incidence of PTSD of 44.3% in their 158 survivors. Of these, 64% remitted by 18 months, but 36% remained very symptomatic after that time, and were predicted to be potentially permanent. They conclude that PTSD in MVA's is far more pervasive and disabling than previously thought, that prolonged PTSD correlated with prolonged soft tissue pain, that prior life trauma, depression and PTSD sensitized MVA victims to the development of PTSD, and that the process of litigation itself may be traumatizing. These findings emphasize the importance of traumatic stress in the morbidity of whiplash, and form part of the foundation for the exploration of this phenomenon as the core event that may lead to the entire spectrum of symptoms in whiplash syndrome. Blanchard and Hickling have partially addressed the dilemma that the DSM-IV definition of PTSD presents (Blanchard & Hickling, 1996). Traumatic stress is a continuum, not a threshold event, and the pervasive effects of such an event on selected brain structures and neurohormonal and neurotransmitter systems also does not follow an all-or-none principle. The resulting symptom complex produced by trauma therefore may vary in the pattern and degree of its expression, and may include somatic symptoms not addressed in the DSM-IV. Strict criteria for a diagnosis are critical to studies of its epidemiology, and in fact to consistent interpretation and treatment of the condition. Standards for criteria based on human behavior, however, are clearly difficult to establish. The relatively

dramatic changes in sequential DSM's reflect this dilemma, especially in the evolving concept of PTSD and the criteria for its diagnosis. Cognitive, somatic and emotional symptoms related to traumatic stress may persist long after the traumatic event although DSM-IV criteria may no longer be sufficient to make the diagnosis of PTSD. These persistent complaints represent continued stress-induced alteration of neurophysiologic function that may be significant in contributing to persistent emotional and somatic dysfunction. The late somatic symptoms of trauma, which may be musculoskeletal, gastrointestinal, cardiac or hormonal, are especially important to the consideration that whiplash may be primarily a consequence of traumatic stress. Many of these symptoms are not associated with documented pathology, and are frequently attributed to psychosomatic causes or somatization. Van der Kolk, et al (van der Kolk, Pelcovitz, Roth, Mandel, McFarlane & Herman, 1996) consider somatization, as well as dissociation and affect dysregulation, to be specific late symptoms of trauma which may exist in the absence of residual traumatic symptoms addressed under the DSM-IV. They refer to these late syndromes as associated disorders of PTSD. They further state: "...people whose presenting problem consists of somatization are likely to be treated with medical interventions, which can be expected to be ineffective in alleviating their distress." (van der Kolk et al, 1996, p.14). Acknowledgement of the somatic manifestations of traumatization is very important to the hypothesis of the late symptoms of whiplash as representing a somatic expression of trauma.

DISSOCIATION AND THE FREEZE RESPONSE

Individuals who dissociate at the time of a traumatic event are much more likely to develop symptoms of PTSD in the future (Bremner, Southwick, Brett, Fontana, Rosenheck & Charney, 1992, Holen, 1993, Cardena & Spiegel, 1993,). Dissociation is defined as "An unconscious process by which a group of mental processes is separated from the rest of the thinking process, resulting in an independent functioning of these processes and a loss of the usual relationships" (Stedman, 1982). Clinical symptoms of dissociation include depersonalization, derealization, conversion hysteria, fugue states and flashback memories. Symptoms of dissociation at the time of a MVA include out-of-body experiences, shock, numbing, detachment and confusion. Although these symptoms are included in criteria for minor traumatic brain injury (Minor Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation, 1993), their occurrence in low velocity accidents insufficient to cause axonal injury suggests that they indeed are probably dissociative phenomena. Dissociation has been recognized as an equivalent of the freeze/immobility response as part of the fight/flight/freeze reflex seen in almost all species. Dissociation and freeze/immobility probably have neurochemical correlates in marked autonomic instability (Gellhorn, 1960), and possibly endorphinergic input (Sherman, Lewis, Terman & Liebeskind, 1983).

The effect of endorphins in dissociation may account for the sense of numbing and shock seen in the acutely traumatized individual (van der Kolk, 1989). Levine presents an intriguing theory of traumatization using the ethologic model of freeze/immobility resolution in animals in the wild (Levine, 1997).

When a prey animal can no longer escape a predator, it will enter a state of freezing, or immobility, during which it is unresponsive, atonic and analgesic, but also is undergoing cyclical high levels of sympathetic, parasympathetic and probably endorphinergic activity. If the animal survives, it will arouse, and go through a stereotyped sequence of behaviors manifested by variably intense, repetitive motor activity (trembling, twitching, convulsing) followed by deep respiration and apparent recovery. Levine hypothesizes that humans who freeze or dissociate when confronted with a life-threatening trauma in the face of helplessness, tend not to "discharge" the associated autonomic arousal through neuromuscular activity. Rather, they tend to retain it in a state of the sustained potential for cue-related arousal, as in the Vietnam veteran who experiences adrenergic arousal at the sound of a helicopter. According to Levine, absence of the freeze discharge may facilitate imprinting of the traumatic event in memory linked to arousal, and lead to retention of the "energy" of the fight/flight response in brain circuitry. This state of memory imprinting and sustained arousal-based energy, Levine believes, is the substrate for the development of the symptoms of PTSD, as well as for the development of an additional potential spectrum of psychological and somatic symptoms. He also emphasizes the concepts of resiliency and reserve capacity as limited resources, with each subsequent trauma diminishing the individual's capacity to tolerate further trauma.

PTSD, MEMORY AND KINDLING

Alterations of memory constitute one of the three subsections of the DSM-IV criteria for the diagnosis of PTSD, including reexperiencing in the form of intrusive recollections, dreams, flashbacks and cue-related arousal. They also participate in the subsection involving avoidance phenomena, including inability to recall important aspects of the trauma, and avoidance of places and events that stimulate recall of the traumatic event. These traumatic epiphenomena involve both declarative (explicit, semantic) and nondeclarative (implicit, procedural) forms of memory. Declarative memory, the form that relates to facts and events and initially involves primarily hippocampal and prefrontal cortical pathways, plays an important role in the conscious recollection of trauma-related events. Procedural memory relates to acquisition of skills and habits, to the development of emotional associations, and to the storing of conditioned sensorimotor responses (van der Kolk, 1994). This form of unconscious memory plays a critical role in the development of many of the linked arousal, sensorimotor and declarative memory symptoms in PTSD. Combining basic concepts of the linking of memory in trauma with retained cue-related arousal

leads to a logical hypothesis that attributes the origin of the complex symptoms of whiplash to an actual alteration of brain circuitry. This hypothesis involves the concept of retained neural patterns of activation in the form of kindling, involving centers of arousal (hypothalamus, locus ceruleus, amygdala), declarative memory circuitry (hippocampus), and that portion of brain centers and pathways linked to neuromuscular procedural skill and conditioning memory (basal ganglia, cerebellospinal, vestibulospinal pathways).

The pathophysiological model of kindling was developed in rats by applying a repetitive electrical stimulus to a part of the brain with specific frequency and intensity (Goddard, McIntyre & Leetch, 1969). Application of subthreshold stimuli at a critical frequency could eventually summate and lead to a seizure that ultimately could be self-perpetuating and result in a seizure disorder. The amygdala proved to be the most sensitive area for the development of kindling (Post, Weiss & Smith, 1995). Kindling has been postulated to play a significant role in a variety of psychophysiological syndromes, including affective disorders, PTSD, chronic pain, post concussion syndrome and multiple chemical sensitivities (Miller, 1997). The phenomenon of kindling has special appeal when considering the genesis of PTSD, a syndrome characterized by variable latency of onset, self-perpetuation, exquisite stimulus sensitivity, the potential to progress and worsen, and in some cases, permanence. These very features characterize many whiplash patients who demonstrate delayed recovery.

The concept that external behavioral stimuli might be the source of theoretically permanent changes in brain physiology and neurochemistry might seem speculative at best. PET scan imaging of regional brain metabolism, including glucose utilization and blood flow, however, shows consistent regional patterns of alteration associated with specific conditions and cognitive activities (Bremner, 1998). These findings lend credence to concepts of brain plasticity required to support such theories as experience-induced neuronal pathway alteration as is felt to occur in the process of kindling.

This hypothesis of a truncated somatically-based discharge of arousal-based activation of strategic brain centers is closely linked to the hypothesis of kindling as the generator for PTSD-related symptoms. In this hypothesis, procedural sensorimotor memory is likely to be established for protective muscular bracing patterns generated by the basic flexor reflexes precipitated by stretch receptor activation at the moment of trauma/impact. Acute arousal would simultaneously be elicited by visual and sensorimotor input signifying threat, accessed by the thalamus and evaluated for emotional content by the amygdala. Declarative memory of this sensory input would establish an explicit memory base for the traumatic event. Lack of dissipation of arousal-based neuronal input associated with the truncated freeze discharge would establish a stimulus generator for continued message transfer through involved linked neuronal circuitry. The amygdala would be exposed to repetitive neuronal input based on persistent cue-related arousal stimuli as part of this circuit, rapidly setting up a kindled response

between centers of arousal, declarative memory and neuromuscular centers for procedural memory. Soon sensorimotor or memory input into any limb of this circuit would activate that center and lead to potentiation of kindling, and progression of the symptom complex. As a result, any incidental declarative memory of the trauma would elicit arousal and patterned regional muscular bracing. Excessive movement of the regional muscle group involved in the original trauma-associated reflex bracing response would elicit arousal linked to procedural memory for that movement pattern, possible conscious memory of the trauma, and further enhancement of reflex muscular bracing. Eventually any nonspecific source of arousal might elicit the same stereotyped regional muscular bracing, as well as on occasion explicit memory for the trauma. Sleep patterns associated with dreams or nightmares of the trauma would cause cyclical nocturnal muscular bracing, bruxing or autonomic manifestations of arousal, including tachycardia and nocturnal diaphoresis. With time, stimuli specific to the trauma might generalize, leading to a broader range of kindled circuitry, and apparent worsening and spread of trauma-related symptoms in the face of nonspecific ambient life stress.

MYOFASCIAL PAIN

Myofascial pain is well recognized as a significant cause for persistent skeletal pain in whiplash (Evans, 1992, Miller, 1998). Earlier called fibrositis or myofibrositis, myofascial pain was described initially by clinicians in the 19th century and early 1900's (Helleday, 1876, Gowers, 1904, Steindler, 1920). It has often been attributed to sudden violent strains of muscles, ligaments or tendons, or to extended overuse of a musculotendinous unit. Clinical features involve the concepts of trigger points, which are tender areas found in taut fibrous bands in affected muscle groups. Compression of these trigger points often produces a flinching withdrawal termed the "jump sign", and may lead to referred pain and/or paresthesias to predictable distant referral patterns of the body (Travell & Simons, 1983). Dry needling, or injection of saline or local anesthetic into trigger points may relieve related muscular pain for variable periods of time. Biopsies of trigger points reveal subtle changes in mitochondria and ATP, with mucopolysaccharide deposition and sarcolemmal disruption (Travell & Simons, 1983). Although these findings might be suggestive of a dystrophic process related to increased metabolic demand in the face of reduced circulation, their implications are at best speculative. Theories of causation include a sarcolemmal energy crisis (Simons, 1996), a dysfunctional muscle spindle (Hubbard, 1996), a dysfunctional extrafusal motor endplate (Hong & Simons, 1998), dysfunction in the gamma motoneuron circuitry (Donaldson, Nelson & Schulz, 1998), and a sustained positive feedback loop from muscle spindle and joint capsule proprioceptors to cerebellum, basal ganglia and spinal cord (Elson, 1990). These theories all assume that myofascial pain is derived from peripheral neuromuscular dysfunction.

Conversely, the hypothesis that myofascial pain may be a peripheral neuromuscular expression of kindled and arousal-triggered reflex central neuronal input to the gamma motor system has many compelling features. Regional predominance is typical of myofascial pain, and in whiplash one can often map the logical patterns of that pain based on analyzing which muscle groups were exposed to the predominant stretch reflex as a result of the directions of force. Thus, the muscle groups opposite the point of impact are the ones subjected to the most stretch. They often eventually become the major source of pain. Recurrence of local spasm is more dependent on trauma-related emotional stressors than on any other factor, although flare-ups of spasm related to nonspecific stress is also very common. Nocturnal exacerbation of myofascial pain, frequently linked to trauma-related dream content is a well-known event, especially in the example of nocturnal bruxing. All of these clinical characteristics of myofascial pain seen in whiplash are consistent with a central neuronal origin. They are also consistent with the concept of myofascial pain as a condition of trauma-linked patterns of muscular bracing, stored in procedural motor memory, and linked to declarative memory and arousal in circuitry perpetuated by kindling.

MINOR TRAUMATIC BRAIN INJURY

The concept of concussion, or minor traumatic brain injury [MTBI] in whiplash has also been extensively investigated. Cognitive impairment as a measure of brain injury usually involves impairment in speed of information processing, divided attention, and short-term memory (Gronwall & Wrightson, 1981). Specific alteration of consciousness without loss of consciousness or amnesia is felt to constitute sufficient criteria for the diagnosis of concussion, or MTBI (Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Group of the American Congress of Rehabilitation, 1993). The mechanism of MTBI is felt to be the tearing or stretching of axons (axonal shearing) as a result of forces related to excessive relative changes in velocity of the head associated with the acceleration/deceleration of the head during the movement of whiplash (Wickstrom, et al, 1967, Varney & Varney, 1995).

Studies have shown that actual head impact is not necessary to cause axonal shearing (Povlishock, Becker, Cheng & Vaughn, 1983). Attempts to correlate brain injury with velocity in the absence of head impact, however, have been fraught with uncertainty and controversy. At forces of 6G, a fighter pilot may begin to lose consciousness, and at 8G be unable to lift his extremities (U.S. Naval Flight Surgeon's Manual, 1978). A 15 miles-per-hour accident generates approximately 10G of force on the head (Severy, et al, 1955). These data suggest that low impacts may have substantial potential for soft tissue, and presumably brain injury. Other authors have emphasized the importance of stopping distance, or rate of change of velocity, in determining relative force

exposure to the head (Varney & Varney, 1995). As previously noted, however, analyses of acceleration perturbations of daily living reveal that such activities as a back slap and hopping off of a step generate significant forces without injury. Plopping backward into a chair, for instance, generates a force vector of 10.1G (Allen, et al, 1994). Cognitive and emotional deficits after MTBI may even be more likely in less severe injuries than those with more prolonged loss of consciousness (Alexander, 1992). In addition, PTSD (Bremner, Scott, Delaney, Southwick, Mason, Johnson, Innis, McCarthy & Charney, 1993, Alexander, 1992), depression (Goodwin, 1997), and chronic pain (Grigsby, Rosenberg & Busenbark, 1995) have been shown to be associated with cognitive deficits of a severity and similarity sufficient to make the diagnosis of a head injury on neuropsychological test batteries. Cognitive deficits attributable only to PTSD in the face of documented traumatic events have been demonstrated in such events as terrorist attacks, imprisonment, vehicular accidents and combat-related ship sinkings (Gill, Calev, Greenberg, Kugelmas & Lerer, 1990, Miller, 1993). Differentiating the cognitive deficits due to chronic pain and PTSD from those attributable to MTBI in MVA's is therefore clearly a major dilemma.

POST CONCUSSION SYNDROME

The varied symptom complex of the post concussion syndrome also continues to present a major challenge to the clinician treating MTBI. In addition to cognitive impairment, patients experiencing a concussion commonly complain of headaches, dizziness and vertigo, nausea, tinnitus, blurring of vision and emotional symptoms. The symptoms involving mood and emotional control include irritability, emotional lability, anxiety, stimulus sensitivity, depression and sleep disturbance. Most are consistent with the diagnosis of PTSD. The frequency of post concussion symptoms following minor traumatic brain injury approaches 50%, and minor head injuries constitute the vast majority of all head injuries presenting to emergency room (Alves, Colohan, O'Leary, Rimel & Jane, 1986). The natural history of post concussion syndrome is that of the presentation of relatively few symptoms at the time of hospital or emergency room discharge, but increase in frequency of symptoms at three months, especially dizziness, cognitive problems, fatigue, visual and auditory problems. Thereafter, many symptoms tend to improve, but headaches, dizziness, and cognitive problems tend to persist at one year in 20-25% (Alves, et al, 1986). Mood changes and fatigue may be uncommon early in the course of post concussion syndrome, but often appear in cases of delayed recovery (Binder, 1986). Alexander (Alexander, 1992) compared clinical features of post concussion syndrome in MTBI (loss of consciousness [LOC] < 15 minutes, posttraumatic amnesia [PTA] < 24 hours), and moderate-to-severe brain injury (LOC > 25 minutes, PTA < 24 hours). Although symptoms were similar between the groups, at six months post accident patients in the mild group were significantly more disabled than in the moderate-to-severe group, raising

substantial doubt about the contribution of structural brain injury to the severity and prolongation of post concussion symptoms. Other authors have questioned the evidence in available literature for a specific single cause for post concussion syndrome (Rutherford, Merrett & McDonald, 1977, Lidvall, Linderoth & Norlin, 1974).

Attempts to provide a rationale for post concussion syndrome have related symptoms to cranial, cranial adnexal and musculoskeletal trauma to soft tissues of the head and neck (Zasler, 1992). A variety of neurodiagnostic tests have been developed in an attempt to prove an organic etiology for such varied complaints as cognitive impairment, positional vertigo, visual dysfunction, disorders of taste and smell, and sleep dysfunction. Such tests include quantitative EEG (Thatcher, Walker, Gerson & Geisler, 1989), posturography (Shumway-Cook & Horak, 1990), electronystagmography (Zasler, 1992), somatosensory and brainstem auditory evoked potentials (Green, Leon-Barth, Dieter, Bengante & Walker, 1992), cognitive (P300) evoked potentials (Onofrij, Curatola, Malatesta, Bazzano, Colamartino & Fulgente, 1991), chemosensory evaluation (Doty, 1992) and polysomnography (Zasler, 1992). Although these tests provide a number of intriguing possibilities for organ-specific causes for several post concussion symptoms, results are in general too inconsistent to lead to predictable etiologic conclusions or treatments. In some cases, of course, demonstrable structural damage may occur to cranial adnexa. Vertigo and balance dysfunction may be due to otolith displacement, perilymphatic fistula or endolymphatic hydrops (Zasler, 1993). Visual disturbance may be associated with corneal or lens injuries or retinal detachment. Structural injury to the intraarticular disc of the temporomandibular joint may indeed occur and create specific symptoms (Talley, Murphy, Smith, Baylin & Haden, 1990). On the other hand, most whiplash and MTBI patients presenting with typical post concussion symptoms demonstrate no objective findings on examination or testing that provides objective evidence for a structural etiology for their symptoms.

Miller (Miller, 1997) has postulated that "diffuse axonal injury and excitatory neurotransmitter release" associated with MTBI may lead to "neurosensitization", a process that he relates to kindling, which may then lead to the varied symptoms of the post concussion syndrome. He also applies the theory of neurosensitization to other traumatic disability syndromes, including PTSD, affective disorder, toxic trauma and chronic pain. A comparison of post concussion syndrome and PTSD reveals that these two conditions share many symptoms, including stimulus sensitivity, sleep disturbance, headache, emotional symptoms and cognitive impairment. It has been noted that 17-35% of patients diagnosed as having MVA-related MTBI also demonstrate the presence of PTSD (Layton, et al, 1995, Middelboe, et al, 1991, Ohry, et al, 1996, Rattock, et al, 1993). A more recent study of 79 MVA related MTBI patients revealed an incidence of acute stress disorder in 14%, and a six-month follow-up incidence of 24% (Bryant & Harvey, 1998). Dissociative symptoms suggestive of acute stress disorder are also seen in MTBI. Depersonalization, derealization and amnesia

are commonly seen during posttraumatic amnesia in MTBI victims (Grigsby, 1986, Grigsby & Kaye, 1993). These associations illustrate the similarity and commonality of symptoms between acute MTBI, acute stress disorder, post concussion syndrome and PTSD.

Analysis of the specific symptoms of post concussion syndrome in this light reveals a clear association between them and the phenomena of dissociation, kindled neuromuscular bracing, cyclical autonomic instability and procedural memory. Dizziness may be a symptom of autonomic dysregulation of pulse, blood pressure and cranial vasomotor stability. When experienced as positional vertigo, it may also be a symptom of procedural memory for the vestibular sensory input experienced in association with directional velocity changes of the head at the time of the MVA, linked to arousal from the trauma. Posttraumatic headache has both myofascial and vascular features, and may be due to autonomic dysregulation, and its effects on cranial vasculature. Binocular visual dysfunction may well be due to reflexive ocular divergence and pupillary dilatation, a known oculomotor reflex triggered by arousal. Linked to subsequent experiences associated with arousal, this ocular bracing response is analogous to the hypotheses of arousal-induced regional muscular bracing, thereby leading to convergence insufficiency and lack of binocular congruency.

Late somatic symptoms seen frequently in patients with chronic PTSD are also stereotyped and predictable, but are frequently misunderstood by physicians since they do not conform to criteria-based symptoms of PTSD as outlined in the DSM-IV. They are frequently attributed to "psychological causes", or to "somatization", and are often blamed on premorbid characterological traits. These late symptoms include continuing stimulus hypersensitivity to bright lights and sound, often requiring the use of dark glasses and earplugs. Stress intolerance may present with cyclical exacerbation of most symptoms, especially cognitive dysfunction and fatigue, usually associated with nonspecific stress exposure. The syndrome may be associated with a pervasive feeling of having the victim's having lost their sense of self. Shyness, constriction and social withdrawal may develop. Somatic and visceral symptoms are also remarkably common long after exposure to traumatic stress. These symptoms may include irritable bowel syndrome, urinary frequency and urgency, premenstrual syndrome in women, myofascial pain, post-traumatic fibromyalgia and pervasive chronic fatigue. This late syndrome of emotional and somatic symptoms tends to assume a cyclical pattern directly associated with what otherwise would be considered normal life stresses. The symptoms described, when analyzed in the light of the freeze response and dissociation, are consistent with a pattern of alternating arousal and freezing (dissociation), with associated sympathetic and parasympathetic cyclical dominance and instability. With incidental life stress, these patients tend to dissociate, leading to the numbing of cognition, perception and emotions, as well as the visceral symptoms described above. In some patients, each cycle of arousal and dissociation potentates the process itself, and leads to a progressive decline in function.

The concept of vulnerability based on prior exposure to traumatic stress is also very important to the consideration of the meaning of the event in an MVA. Several studies have demonstrated that the outcome of a traumatic experience is better predicted by life experiences and factors occurring before the trauma than the severity of the trauma itself (Clayton & Darvish, 1979, Weisath, 1984). MacFarlane, in a study of PTSD in the wake of a natural disaster, found that premorbid vulnerability in the form of prior life traumatic events or psychiatric history accounted for a greater percentage of the variance of disorder than the impact of the disaster (MacFarlane, 1989). Prior life trauma must be considered when evaluating patients with delayed recovery from whiplash, especially those who have experienced relatively trivial accidents.

CONCLUSIONS

We have developed a hypothesis of the whiplash syndrome as an essentially brain-based reflex phenomenon, involving linked and kindled circuitry between centers of arousal, declarative and procedural memory, and subcortical motor centers of the basal ganglia and brainstem. Implicit in this theory is the concept of fueling of the development of this circuit by a truncated autonomic and somatically-based freeze/immobility response discharge at the time of the traumatic event, in this case an MVA (Levine, 1997). Once activated, this circuitry is self-perpetuating and relatively permanent as a result of kindling. Although it may be generalized and relatively suppressed by the passage of time and nonspecific therapy, it remains a substrate for increased susceptibility to emotional and somatic symptoms of trauma elicited by increasingly trivial life stresses. Thus a nonspecific life stress in such an individual may precipitate a recurrence of cognitive, emotional and myofascial symptoms from which they had supposedly recovered. Acceptance of the thesis that a relatively trivial stress or emotional trauma may trigger a marked dysfunctional somatic response requires suspension of the concept of Cartesian mind/body dualism, and embracing the burgeoning scientific data supporting the neurophysiologic basis for emotional symptoms and mental illness, as well as their associated somatic conditions. This hypothesis has its base in existing theories of PTSD, and is closely linked to these concepts. The PTSD literature, however, still does not adequately address the somatic elements of traumatization, which I believe to be critical to understanding the entire spectrum of whiplash symptoms, and for that matter, all late symptoms of post-traumatic somatization. This concept of whiplash resolves the dilemmas of delay of onset, persistence and occasional permanence of symptoms, the discrepancy in force exposure and apparent injury, and the occasionally pervasive cognitive and emotional complaints seen in some patients involved in low velocity accidents. It also emphasizes the role of prior life trauma in sensitizing the MVA victim to a disproportionate adrenergic response to MVA-induced trauma, and the development of delayed whiplash

recovery. This concept of reduced reserve capacity for stress tolerance, and therefore reduced resiliency and increased vulnerability to sequential episodes of traumatic stress, is a basic feature of patients with a history of trauma.

Treatment implications for whiplash are clear. Early therapy for exposure to traumatic stress is essential, and should include both counseling, education and preferably addition of somatically based trauma therapy designed to desensitize the autonomic nervous system (Levine, 1997, Wilson, Becker & Tinker, 1995). Some of the most effective medications for the arousal symptoms of PTSD fall into the category of anticonvulsants, including carbamazepine, valproic acid, gabapentin and clonazepam. These medications might predictably be expected to inhibit the process of kindling. Physical measures for pain treatment can be expected to provide only short-term relief, but inclusion of relaxation techniques and stretching exercises that emphasize inhibition of the stretch reflex should be useful. Acceptance of a unified and logical physiologic basis for the seemingly disparate and disconnected symptoms of whiplash is important to the effective management of the care of these patients. Use of the terms "psychosomatic", "psychophysiological" and "supratentorial" in a perjorative fashion should be avoided. Many whiplash patients feel dismissed and invalidated by their physicians and auto insurance company representatives. Understanding and validation by the physician, therapist, insurance payor and legal system is less likely to retraumatize the patient and perpetuate the syndrome.

BIBLIOGRAPHY

- Allen, M., Weir-Jones, I., Eng, P., Motiuk, D., Flewin, K., Goring, R., Kobetitch, R., Broadhurst, A. (1994): Acceleration perturbations of daily living: A comparison to 'whiplash', *Spine*, 19(11):1285-1290
- Alexander, M. (1992): Neuropsychiatric correlates of persistent postconcussive syndrome, *Journal of Head Trauma Rehabilitation*, 2:60-69
- Alves, W., Colohan, A., O'Leary, T., Rimel, R., Jane, J. (1986): Understanding posttraumatic symptoms after minor head injury, *Journal of Head Trauma Rehabilitation*, 1(2):1-12
- American Psychiatric Association, Diagnostic and Statistical Manual of Mental Diseases, 3rd edition, 1980
- American Psychiatric Association, Diagnostic and Statistical Manual of Mental Diseases, 3rd edition, revised, 1987
- American Psychiatric Association, Diagnostic and Statistical Manual of Mental Diseases, 4th edition, 1994
- Balla, J. (1984): Report to the Motor Accident Board of Victoria on Whiplash Injuries, In: Hopkins, A., ed., Headache, problems in diagnosis and management, Chapter 10, *Headache and Cervical Disorders*, London: Saunders, 1988:256-269

- Binder, L. (1986): Persisting symptoms after head injury: a review of the postconcussive syndrome, *Journal of Clinical and Experimental Neuropsychology*, 8:323-346
- Blanchard, E., Hickling, E. (1996): *After the Crash: Assessment and Treatment of Motor Vehicle Accident Survivors*, American Psychological Association: Washington, D.C.
- Bosworth, D. (1959): (Editorial) *Journal of Bone and Joint Surgery*, 1959;41-A:16
- Bremner, J. (1998): Neuroimaging of posttraumatic stress disorder, *Psychiatric Annals*, 28:8:445-449
- Bremner, J., Scott, T., Delaney, R., Southwick, S., Mason, J., Johnson, D., Innis, R., McCarthy, G., Charney, D. (1993): Deficits in short-term memory in posttraumatic stress disorder, *American Journal of Psychiatry*, 150:7:1015-1019
- Bremner, J., Southwick, L., Brett, E., Fontana, A., Rosenheck, R., Charney, D. (1992): Dissociation and posttraumatic stress disorder in Vietnam combat veterans, *American Journal of Psychiatry*, 149:328-332
- Bryant, R., Harvey, A. (1998): Relationship between acute stress disorder and posttraumatic stress disorder following mild traumatic brain injury, *American Journal of Psychiatry*, 155(5): 625-629
- Cardena, E., Spiegel, D. (1993): Dissociative reactions to the Bay Area earthquake, *American Journal of Psychiatry*, 150:474-478
- Clayton, P., Darvish, H. (1979): Course of depressive symptoms following the stress of bereavement. In *Stress and Mental Disorder*, Barrett, J., ed., New York:Raven Press
- Davis, A. (1945): Injuries of the cervical spine, *JAMA*, 127:149-156
- Donaldson, C., Nelson, D., Schulz, R. (1998): Disinhibition in the gamma motoneuron circuitry: a neglected mechanism for understanding myofascial pain syndrome? *Applied Psychophysiology and Biofeedback*, 23(1):43-57
- Doty, I. (1992): Diagnostic tests and assessment, in Zasler, N., Costanzo, R., eds: Smell and taste dysfunction, *Journal of Head Trauma Rehabilitation*, 7(1)47-65
- Elson, L. (1990): The Jolt Syndrome: Muscle dysfunction following low-velocity impact, *Pain Management*, Nov/Dec, 317-326
- Evans, W. (1992): Some observations on whiplash injuries, *Neurologic Clinics*, 10:975-997
- Friedmann, L., Marin, E., Padula, P. (1992): Biomechanics of cervical trauma, in *Painful Cervical Trauma: Diagnosis and Rehabilitative Treatment of Neuromuscular Injuries*, Tollison, C., Satterthwaite, J., eds, pp. 10-19
- Gay, J., Abbott, K. (1953). Common whiplash injuries of the neck, *JAMA*, 152:1698-1704
- Gellhorn, E. (1960): The tuning of the autonomic nervous system through the alteration of the internal environment (asphyxia), *Acta Neurologica*, 20:515-540

- Gill, T., Calev, A., Greenberg, D., Kugelmas, S., Lerer, B. (1990): Cognitive functioning in posttraumatic stress disorder, *Journal of Traumatic Stress*, 3:29-45
- Goddard, G., McIntyre, D., Leetch, C. (1969): A permanent change in brain functioning resulting from daily electrical stimulation, *Experimental Neurology*, 25:295-330
- Goodwin, G. (1997): Neuropsychological and neuroimaging evidence for the involvement of the frontal lobes in depression, *Journal of Psychopharmacology*, 11:115-122
- Gowers, W. (1904): A lecture on lumbago: Its lessons and analogues, *British Medical Journal* 1:117-121
- Green, J., Leon-Barth, C., Dieter, J., Bengante, P., Walker, D. (1992): Somatosensory evoked responses via electronic brain imaging (EBI), *Clinical Electroencephalography*, 23:79-88
- Grigsby, J. (1986): Depersonalization following minor closed head injury, *International Journal of Clinical Neuropsychology*, 8:65-69
- Grigsby, J., Kaye, K. (1993) Incidence and correlates of depersonalization following head trauma, *Brain Injury*, 7:507-513
- Grigsby, J., Rosenberg, N., Busenbark, D. (1995): Chronic pain associated with deficits in information processing, *Perceptual and Motor Skills*, 81:403-410
- Gronwall, D., Wrightson, P. (1981): Memory and information processing capacity after closed head injury, *Journal of Neurology, Neurosurgery and Psychiatry*, 44:889-895
- Helleday, U. (1876): Om mysitis chronica (rheumatica). Ett bidrag till dess diagnostik och behandling, *Mord Med Ark*, art 8
- Holen, A. (1993): The North Sea oil rig disaster, In Wilson, J., Raphael, B., eds, *International handbook of traumatic stress syndromes*, New York: Plenum Press, pp 471-479
- Hong, C., Simons, D. (1998): Pathophysiologic and electrophysiologic mechanisms of myofascial trigger points, *Archives of Physical Medicine and Rehabilitation*, 79:863-871
- Hubbard, D (1996): Chronic and recurrent muscle pain: Pathophysiology and treatment and review of pharmacological studies, *Journal of Musculoskeletal Pain*, 4(1,2):123-143
- Layton, B., Wardi-Zonna, K. (1995): Posttraumatic stress disorder with neurogenic amnesia for the traumatic event, *Clinical Neuropsychologist*, 1995;9:2-10
- Levine, P. (1997): *Waking the Tiger: Healing Trauma*, North Atlantic Books:Berkeley
- Lidvall, H., Linderoth, B., Norlin, B. (1974): Causes of the post-concussion syndrome, *Acta Neurologica Scandinavia*, suppl 56:7-144
- Luo, Z., Goldsmith, W. (1991): Reaction of a human head/neck/torso system to shock, *Journal of Biomechanics*, 24(7):499-510

- MacFarlane, A. (1989): The aetiology of post-traumatic morbidity: Predisposing, precipitating and perpetuating factors, *British Journal of Psychiatry*, 154:221-228
- Marshall, H.(1919): Neck injuries, *Boston Medical Surgical Journal*, 180:93-98
- McConnell, W., Howard, R., Guzman, H., Bornar, J., Raddin, J., Benedict, J., Smith, H., Hatsell, C. (1993): Analysis of human test subject kinematic responses to low velocity rear end impacts, *Society of Automotive Engineers*, 930899
- McNab, I. (1971): The whiplash syndrome, *Orthopedic Clinics of North America*, 2:389-403
- Middelboe, T., Anderson, H., Birket-Smith, M., Fiis, M. (1991): Minor head injury: impact on general health after 1 year: a prospective follow-up study, *Acta Neurologica Scandinavia*, 85:5-9
- Miller, L. (1997): Neurosensitization: A pathophysiological model for traumatic disability syndromes, *The Journal of Cognitive Rehabilitation*, Nov/Dec, pp12-23
- Miller, L. (1998): Motor vehicle accidents: Clinical, neuropsychological, and forensic considerations, *The Journal of Cognitive Rehabilitation*, Jul/Aug, pp.10-23
- Miller, L. (1993): The 'trauma' of head trauma: Clinical, neuropsychological and forensic aspects of chemical and electrical injuries, *Journal of Cognitive Rehabilitation*, (i)6-18
- Minor Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation, Definition of mild traumatic brain injury, (1993), in, *The Journal of Head Trauma Rehabilitation*, 18(3):86-87
- Norris, F. (1992): Epidemiology of trauma: Frequency and impact of different potentially traumatic events on different demographic groups, *Journal of Consulting and Clinical Psychology*, 60:409-418
- Ohry, A., Solomon, Z., Rattock, J. (1996): Post traumatic stress disorder in traumatic brain injury, *Brain Injury*, 10:687-695
- Ommaya, A., Faas, F., Yarnell, P. (1968): Whiplash injury and brain damage-an experimental study, *JAMA*, 204:285-289
- Onofrij, M., Curatola, L., Malatesta, G., Bazzano, P., Colamartino, P., Fulgente, T. (1991): Reduction of P3 latency during outcome from posttraumatic amnesia, *Acta Neurologica Scandinavia*, 83:273-279
- Packard, R., Ham, L. (1994): Posttraumatic headache, *Journal of Neuropsychiatry*, 6:119-136
- Pearce, J. (1989): Whiplash injury: a reappraisal, *Journal of Neurology and Neurosurgery*, 52:1329-31
- Post, R., Weiss, S., Smith, M. (1995): Sensitization and kindling: Implications for the evolving neural substrate of post-traumatic stress disorder, in Friedman, M., Charney, D., Deutch, A., eds, *Neurobiological and Clinical Consequences of Stress: From Normal Adaptation to PTSD*, Lippencott-Raven Publishers, Philadelphia

- Radanov, P., DiStefano, G., Schindrig, A., Ballinari, P. (1991): The role of psychosocial stress in recovery from common whiplash, *Lancet*, 338:712-715
- Rattock, J., Ross, B. (1993): Post traumatic stress disorder in the traumatically head injured (abstract), *Journal of Clinical and Experimental Neuropsychology*, 15:403
- Roy, R. (1961): The role of binocular stress in the post-whiplash syndrome, *American Journal of Optometry and Archives of American Academy of Optometry*, Nov., 1-11
- Rutherford, W., Merrett, J., McDonald, J. (1977): Sequellae of concussion caused by minor head injuries, *Lancet*, i:1-4
- Severy, D., Mathewson, J., Bechtol, C. (1955): Controlled automobile rear-end collisions: an investigation of related engineering and medical phenomena, *Canadian Services Medical Journal*, 11:727-759
- Sherman, J., Lewis, J., Terman, G., Liebeskind, J. (1983): The opioid/nonopioid nature of stress-induced analgesia and learned helplessness, *Journal of Experimental Psychology: Animal Behavior Processes*, 9(1)80-90
- Shumway-Cook, A., Horak, F. (1990): Rehabilitation strategies for patients with vestibular deficits, *Diagnostic Neurology, Neurologic Clinics*, 8:441-457
- Simons, D. (1996): Clinical features of myofascial trigger points, *Journal of Musculoskeletal Pain*, 1(1,2):95-121
- Stedman's Medical Dictionary, (1982) 24th Edition, Williams & Wilkins: Baltimore
- Steindler, A. (1920): The interpretation of sciatic radiation and the syndrome of low back pain, *Journal of Bone and Joint Surgery*, 22:28-34
- Sucher, B. (1990): Thoracic outlet syndrome: A myofascial variant: Part 1. Pathology and diagnosis, *Journal of the American Osteopathic Association*, 8:686-704
- Talley, R., Murphy, G., Smith, S., Baylin, M., Haden, J. (1990): Standards for the history, examination, diagnosis and treatment of temporomandibular disorders (TMD): a position paper, *Journal of Craniomandibular Practice*, 8:60-72
- Thatcher, R., Walker, R., Gerson, I., Geisler, F. (1989): EEG discriminant analyses of mild head trauma, *Electroencephalography and Clinical Neurophysiology*, 73:94-106
- Travell, J., Simons, D. (1983): *Myofascial Pain and Dysfunction: The Trigger Point Manual*, Baltimore: Williams & Wilkins
- Trimble, M. (1981): *Post-traumatic Neuroses*, Chichester: John Wiley & Sons
- U.S. Naval Flight Surgeon's Manual, 1978
- van der Kolk, B. (1989): The compulsion to repeat the trauma: Re-enactment, revictimization and masochism, *Psychiatric Clinics of North America*, 12(2):389-411

- van der Kolk, B. (1994): The body keeps the score: Memory and the evolving psychobiology of posttraumatic stress, *Harvard Review of Psychiatry*, Jan/Feb, 253-265
- van der Kolk, B., Pelcovitz, D., Roth, S., Mandel, F., McFarlane, A., Herman, J. (1996): Dissociation, affect dysregulation and somatization: the complex nature of adaptation to trauma, *American Journal of Psychiatry*, 153(Suppl), 83-93
- Varney, N., Varney, R. (1995): Brain injury without head injury: Some physics of automobile collisions with particular reference to brain injuries occurring without physical head trauma, *Applied Neuropsychology*, 2:47-62
- Ward, D. (1969): The histopathology of auditory and vestibular disorders in head trauma, *Annals of Otology, Rhinology and Laryngology*, 78:227-238
- Weinberg, S., LaPointe, H. (1987): Cervical extension-flexion injury (whiplash) and internal derangement of the temporomandibular joint, *Journal of Oral and Maxillofacial Surgery*, 45:653-659
- Weisath, L.: *Stress Reactions to an Industrial Accident*, MD thesis, Oslo University
- Wickstrom, J., Martinez, J., Rodriguez, T. (1967): Cervical sprain syndrome and experimental acceleration injuries of the head and neck, In Selzer, M. Gikas, P., Huelke, P., eds, vProceedings of Prevention of Highway Accidents Symposium, Ann Arbor:University of Michigan, 182-187
- Wilson, S., Becker, L., Tinker, R. (1995): Eye movement desensitization and reprocessing (EMDR) treatment of psychologically traumatized individuals, *Journal of Consulting and Clinical Psychology*, 63(6):928-937
- Zasler, N.: Neuromedical diagnosis and treatment of postconcussive disorders (1992), in: Horn, I., Zasler, N., eds., *Rehabilitation of Post-Concussive Disorders*, Philadelphia:Hanley & Belfus
- Zasler, N. (1993): Minor traumatic Brain Injury: Medical assessment and intervention, in Barth, J., Macciocche, S., eds, *The Journal of Head Trauma Rehabilitation*, 8(3): 13-29
- Zasler, N., Ochs, A. (1992): Oculovestibular dysfunction in symptomatic mild traumatic brain injury, *Archives of Physical Medicine and Rehabilitation*, 73:963