THE WHIPLASH SYNDROME

II: A MODEL FOR TRAUMATIZATION

MEMORY MECHANISMS IN TRAUMA

THE NEUROPHYSIOLOGY OF TRAUMATIC MEMORY

Memory mechanisms play a critical role in forming the complex neurophysiologic fabric if traumatization. The odds of a lasting memory being implanted in those centers of the brain related to memory storage is directly proportional to the arousal or emotional content of the experience that accompanied the event to be remembered (1). One usually tends to remember important life events with little difficulty. Not only are they associated with specific meanings to us, but they are also usually associated with strong emotions, both negative and positive. Those of you in my generation tend to remember with vivid clarity what they were doing at the exact moment that they learned about the fact that President Kennedy had been shot. We usually remember with remarkable detail, the events of our weddings, the funerals of loved ones, the greatest moments of success of our children, the news that we have a critical illness, or the vivid experience of a serious physical injury. All of these events are associated with heightened emotions, alertness or arousal. Animal experiments reveal that they learn certain tasks more quickly if the task is paired with an arousing stimulus as opposed to a neutral stimulus. The area of the brain most responsible for processing of memory associated with emotion is the amygdala, the small bulb-shaped nucleus at the anterior tip of the limbic system in the mesial temporal lobe. The sensory nuclei of the thalamus receive afferent input from sensory organs throughout the body that they then transmit to the amygdala. The amygdala also receives

information concerning the state of arousal associated with this sensory information in the form of norepinephrine-mediated messages from arousal centers of the brainstem, particularly the locus ceruleus. The locus ceruleus has rich connections with the primary sensory organs of the head and proprioceptive receptors in the neck. Positioning of the head both in the act of orienting to sources of food as well as sources of threat is critical to accessing environmental sensory information via the organs of smell, taste, vision, hearing and vestibular sensation. This sensory input is transmitted via the thalamus to the locus ceruleus as well as to the amygdala except for olfaction, which sends messages directly to the amygdala. With sensory input via these sources, the amygdala evaluates the emotional meaning of the incoming information, and integrates the memory image of the event with the emotional experience (2). This information is then transmitted to the hippocampus, another mesial temporal structure in each temporal lobe. Here the information is categorized, compared with past stored information and then passed on to right orbitofrontal lobe structures which begin the process of organizing behavior in response to the incoming sensory stimuli (3), and of routing messages to appropriate areas of the cerebral cortex for more complex memory organization.

DECLARATIVE VS NONDECLARATIVE MEMORY

Two clearly distinguishable and separate memory systems in the brain have been recognized. Declarative or explicit memory is concerned with verbal and semantic memory for facts, events and information- i.e. “knowing that” (4). It is conscious, intentional and is the part that we use in acquiring information and a formal education. It contains subsystems for episodic memory related to personal experience, and semantic memory transmitted from another’s experience, as in education. Damage to the hippocampus, the brain region probably most important in declarative memory, results in the inability to store conscious new information, as well as excessive responsiveness to environmental stimuli (5,6). Post-traumatic amnesia is characterized by the loss of a segment of declarative memory. It is often one of the earliest functions lost in Alzheimer’s disease and other organic dementias. Declarative memory is also affected and sometimes distorted by the emotional content of the associated experience, and may be notoriously inaccurate. This is clearly illustrated by the varying stories told by survivors of the same traumatic natural disaster or life event, and by the occasional change and distortion of old memories through new life experiences. Declarative memory probably represents only a small fraction of stored memory.

Nondeclarative, or implicit memory, is responsible for storing acquired skills, conditioned responses and emotional associations- i.e. “knowing how” (4,7). It is unconscious, and governs much of that part of our daily activity that is automatic and instinctual, based on past experiences and training. The part of nondeclarative memory that serves skill and habits as well as conditioned sensorimotor responses is called procedural memory. All of the motor skills that we learn and never forget, such as musical, artistic and athletic talents are stored

in procedural memory. Procedural memories are readily acquired without intention, and retained forever without awareness, especially if they are linked to a coincident emotional event (2). They are acquired and stored without the necessary involvement of conscious memory centers serving declarative memory, such as the hippocampus and prefrontal cortex. Motor skill memories are probably in part stored in the brainstem and extrapyramidal centers controlling basic primitive postural reflexes. Another type of nondeclarative memory is involved in the process of unconscious conditioned behavior. Conditioned responses or memories may involve the sensorimotor or autonomic nervous systems and are by definition unconscious. In Pavlov's classic experiment, the induction of consistent salivation in a dog by ringing a bell after pairing the bell with feeding is linked to nondeclarative memory mechanisms. Conditioned responses of this type require reinforcement by continued pairing of the external stimuli to maintain the conditioned behavior. If the bell rings enough times without presentation of the food, the salivation response will undergo extinction and will disappear. This type of conditioning, unlike most procedural memory, is not permanent. If, however, the paired stimuli include a component involving high arousal or emotion, it will take fewer trials of exposure to produce the conditioned behavior, and more trials of unpaired stimuli to extinguish it. In fact, if one of the stimuli represents a life-threatening event, the conditioned response may appear after one trial and never be extinguished. This of course is an example of the conditioned response to acute trauma, and is specifically related to the events and symptom complex accompanying the Whiplash Syndrome. With these concepts of procedural and conditioned memory linked to arousal we can begin to reexamine the Whiplash Syndrome in an entirely new light.

SOMATIC RESPONSES TO STRESS

At or even before the moment of impact of two automobiles, the occupants undergo a predictable series of neurophysiological events. Whether or no the impact was anticipated based on the presence of preceding sensory input may have a bearing on these events and the resulting symptoms. Attempts to determine whether symptoms are worse in the presence or absence of a warning have had mixed results. The prevailing opinion is that anticipation allowing the ability of the person to brace for the impact should diminish injuries. At least one study suggests that the element of surprise resulted in more severe injuries (8). Symptoms indeed may differ in these two situations, but the outcomes are probably similar. Sensory input usually involves visual or auditory awareness of the impending crash and may have an effect on the content of subsequent memories related to the trauma. In either case, within milliseconds, the thalamus receives a barrage of messages from visual, auditory, proprioceptive and vestibular sensory organs, which are then sent to the amygdala for analysis of emotional content. Based on the potential life-threatening nature of the event, and with input from locus ceruleus, the systemic release of epinephrine from adrenal medulla along with brainstem norepinephrine, will trigger the

physiological sequence of the fight/flight response, both within the central nervous system and somatic end organs.

The systemic manifestations of the fight/flight response are of course predictable, but worth reviewing in the context of the accident itself. The eyes will widen, the pupils will dilate and the eyes diverge slightly in order to achieve maximal visual access to the threat. The pulse, systolic blood pressure and cardiac output will markedly increase. Skeletal muscles will exhibit increased tone in preparation for action, but at the same time will be subjected to powerful forces involving stretch and torque in a variety of directions. As the car is moved in one direction as a result of the impact, inertia will inhibit the body from immediately moving, resulting in an apparent movement in the direction of the impact. Stretch receptors in muscles on the side opposite the source of impact will be activated, resulting in selective intense contraction of these muscles on a reflex basis through the gamma motoneuron circuitry. As the rate of acceleration decreases, the body will then rebound, moving next in the direction of the impact, and activating stretch receptors on the side of the impact, but to a much less degree. Based on any rotational positions of the body at the moment of impact, torque forces will also activate varied muscle groups on both sides of the body, causing a complex and diffuse pattern of muscle bracing which is predictably asymmetrical and regional in distribution. The rapid acceleration/deceleration forces in varied directions will cause marked perturbation of vestibular receptors in the semicircular canals. Activation of vestibulospinal pathways will further contribute to postural muscle bracing patterns based on directions of movement of the head. Proprioceptive input from ligaments, tendons and muscles will result in further reflex postural holding patterns. Those parts of the body involved in control of the vehicle, especially the hands on the steering wheel and feet on brake or accelerator pedals, will react based on procedural memory patterns of motor driving behavior, and will brace, clench, or turn in reflex reaction to visual, vestibular and proprioceptive input. At the same time, the marked shift in sympathetic tone will inhibit the vegetative functions of the viscera and cardiovascular system. With the MVA victim in an acute state of fight/flight readiness, and subjected to powerful gravitational forces, the next physiological demand calls for completion of the high level physical activity required to dissipate the effect of adrenergic arousal on the body. At this point the meaning of the event and the relative empowerment of the victim to pursue the physical activity of the fight/flight response become critical. The basic concept of transport in a vehicle of conveyance actually becomes a profoundly important issue.

THE MEANING OF SPEED

VEHICULAR TRANSPORT AND SOCIETY

Although human beings have traveled in wheeled vehicles drawn by horses or other animals for centuries, the speed of travel has been generally slow, and the distances short. The advent of mechanized, relatively high-speed travel with the

invention of the steam engine in the 19th century has had a remarkable effect on human society. Distances have shrunk, the pace of life and the amount "accomplished" in a unit of time have increased, and humans have become accustomed to the unnerving sensations of traveling at a high rate of speed. Nevertheless, a person's first experience with the sensations of rapid acceleration and centrifugal force is often arousing and even frightening. Fear of flying in an airplane for the first time is common, and even repeated exposure with benign experiences does not always extinguish the fear. Amusement park rides are often terrifying before they become exhilarating. There is nothing in our physiologic evolution that prepares us for this particular experience of acute and violent vestibular stimulation. Although the peculiar syndrome involving pain, emotional and cognitive symptoms associated with vehicular accidents has probably been around since the Roman chariots, the first apparent recorded reference to this condition probably relates to "Railroad Spine", referred to in Chapter 3 (9). During that period of the 19th century, this condition was felt to fall into the nebulous category of the neuroses, and as with present day whiplash, was predominantly seen in women. As mechanized transport has transformed the 20th century world, vehicles have assumed roles and meanings far more varied and complex than simply a means of travel. This is especially true in the case of the automobile, which in its century of evolution has progressed from a curiosity to a sign of power and control, a symbol of wealth and accomplishment, and a source of thrill and exhilaration. In much of the civilized world, the automobile and its operation is considered a God-given right, and a necessity of life. On the other hand, those individuals subjected to the forces associated with rapid mechanized transport that suddenly operate outside of the boundaries of control seldom consider the experience exhilirating. They tend to report the experience of panic or arousal in situations where there is loss of control of a motor vehicle, and an accident is averted. If a crash does occur, however, they frequently undergo an experience of numbing or shock for a period of time. As first reported in cases of "Railroad Spine", the effects of sudden high speed and unexpected changes in velocity or direction may produce distressing and longstanding symptoms in certain individuals, especially if this loss of control culminates in a crash. The increasing frequency of soft tissue injuries in MVA's, the relative resistance to treatment of victims of whiplash, and the incredible costs of their medical care suggests that some unique process is taking place when the human body is subjected to these forces in the context of mechanized transport. Yet the paradox exists in cases of loss of control of a motor vehicle, even associated with a crash, when the operation of the vehicle is connected with an environment of aggression, competition or intentional risk- taking, or when the driver is inebriated at the time of the crash. Although meaningful statistics are not available, there seems to be no significant incidence of true Whiplash Syndrome in racecar or stunt drivers involved in impact crashes, despite the occurrence of significant physical injuries, cerebral concussions and life-threatening situations. In well over 5000 cases of whiplash that I have treated, I have never seen manifestations of the Whiplash Syndrome in a person substantially under the influence of alcohol at the time of the accident. The

common denominator in cases of MVA's where Whiplash Syndrome develops, therefore, appears to be the occurrence of the accident in a state of helplessness.

THE AUTOMOBILE AS A THREAT

Most automobile drivers fortunately respect the risks associated with the operation of their car, and realize that bodily injury and death occur with distressing frequency with their use. Admittedly this appropriate sense of vulnerability seems to be uniquely lacking during the teen years, but it seems to develop in most people with the acquisition of what we define as maturity. Although automobile manufacturers have made admirable progress in improving the handling of cars, there is always a fine line in their control which, if crossed, results in the immediate condition of complete helplessness. Most drivers have experienced this sensation of loss of control at some point in their driving careers, and having done so, will never again feel the complete sense of driver confidence that they did before that experience. Even a small collision will usually be enough to change forever the driver's sense of safety in a car. Obviously gender, personality and past life experiences have a major effect on the evolution of this sense of awareness of vulnerability in a car. Nevertheless, for most drivers, operation of a car takes place with a variable sense of risk and helplessness. In the context of the fight/flight/freeze response, a state of helplessness by definition eliminates the option of fighting or fleeing, and obviously neither of these two options exists when one automobile is impacted by another. The event itself, however, is intrinsically life threatening, and will inevitably trigger the appropriate physiological responses of the fight/flight response. In a state of helplessness, the initiation of the fight/flight physiological events will just as inevitably trigger the freeze response, which I feel occurs in a substantial number of MVA victims.

Detailed histories of the initial subjective experiences of MVA victims often reflect the frequency of symptoms of freezing. The immediate experiences of the whiplash victim during the period around the impact of the automobiles are often remembered in vague and even surreal terms. During the moments after the impact, many patients will describe a sense of shock, confusion and detachment, often with no describable emotional tone. "Numbness" is the word most often used to describe the quality of this experience. On rare occasions, the patient will describe the sensation of being "full of adrenaline", or "shaking all over", but an immediate feeling of arousal is uncommon. Often the memory for specific events is vague and unclear, and details of the impact are often remembered piecemeal or out of sequence. Occasionally a sense of detached calm is present, and the victim may appear remarkably rational and in control of themselves. More often, however, the victim will describe a feeling of helplessness and of being overwhelmed. Others will have a sense of unreality, saying to themselves, "How

could this happen to me?". On occasion, a number of my patients have described frank "out-of-body" experiences. Witnesses may describe them as dazed. Attempts by the accident victim to describe the events of the MVA at a later time will frequently reflect the fragmented nature of their recall. Many of these experiences fall into the category of derealization or depersonalization, symptoms typical of dissociation (See Chapter 6).

THE PHYSIOLOGY OF TRAUMATIZATION

How then does traumatization occurring in the context of a MVA explain the varied and multisystemic symptom complex of the Whiplash Syndrome? The experience of trauma primarily involves arousal and memory mechanisms as outlined earlier. Aberrations of memory in trauma involve both declarative and nondeclarative memory, and are characterized by both exaggerated and impaired memory functions (2). In many instances, victims of trauma are amnestic for various events associated with the traumatic event (2). Memory of the event itself is frequently distorted and inaccurate. Many adults abused as children have no specific memory of the trauma itself, and only a vague sense of having been traumatized. When memories themselves are "recovered", they often are remarkably inaccurate. Distortion or suppression of traumatic memories may be proportional to the severity of the freeze response, or dissociation at the time of the trauma. Enhancement of memories of the event may also be prominent, leading to involuntary resurfacing of these memories in a variety of settings. Arousal linked to the conscious memories of sensory experiences of the trauma leads to the laying down of a powerful feedback circuit within associated brain centers, in part probably involving locus ceruleus, amygdala and hypothalamus (1). This may result in the triggering of recurrent and intrusive memories of the trauma with even nonspecific arousal, and the triggering of arousal by even nonspecific events or perceptions reminiscent of the trauma.

This arousal/memory link in trauma may also be enhanced by nondeclarative and declarative memories of past trauma with links to the immediate event, such as prior experience with MVA's. Specific procedural memories of sensorimotor experiences from both current and past related trauma may also be incorporated into this newly activated circuitry. Subsequent arousal may then trigger recurrence of these experiential memories, such as pain, dizziness and the protective neuromuscular bracing response associated with the MVA. These experiences and sensations may then be incorporated in the arousal/memory circuitry.

KINDLING AND TRAUMA

The mechanism by which this self-sustaining feedback circuit is established may well be related to the physiological phenomenon of kindling. The term kindling was developed from the description of spontaneous combustion of materials reaching a certain critical temperature. The physiologic model was developed in rats by applying a repetitive electrical stimulus to an area of the brain with specific frequency and intensity (10). Although each stimulus was insufficient to trigger a convulsion, if the stimuli were applied with a critical frequency, they would summate and trigger a seizure. In addition, if kindled seizures were induced in newborn rats with many repetitions, the rats would exhibit the tendency for spontaneous seizures that thereafter would be self-perpetuating, and would occur without any stimulus. In other words, these rats developed a relatively permanent change in the excitability of neuronal networks within the kindled part of their brain. The brain region most susceptible to kindling is the amygdala. In the case of PTSD, the repetitive neural input to the feedback loop associated with recurrent memory events may well derive from the sustained high-level adrenergic arousal persisting as a result of the undischarged freeze response. This recurrent arousal would routinely activate the amygdala which in turn would interpret the resulting emotion-based memories as threatening, resulting in the triggering of arousal once again. The result would be the spectrum of memory events seen in PTSD: flashbacks, intrusive memories, arousal-triggered memories and nightmares. Another result would be the arousal symptoms of PTSD: anxiety, panic attacks, phobias of events and places reminiscent of the trauma, memory and situation-induced arousal, mood changes, irritability, stimulus sensitivity, exaggerated startle, and insomnia. The frequent delay in onset of symptoms of PTSD after a traumatic event is quite consistent with the concept of kindling as an evolving neurophysiologic process. Similarly, the tendency for PTSD to change in both the nature of the predominant symptoms and the occasional worsening of the condition is consistent with kindling, a process which by definition changes neural excitability and eventually becomes self-sustaining without further input. Delay in onset, change in basic characteristics and spontaneous worsening of memory and arousal-related symptoms are all typical of the progress of PTSD symptoms in the Whiplash Syndrome. The same characteristics are also typical of the cognitive and somatic symptoms of whiplash, which often do not appear for a variable period after the MVA, and may evolve and change character over many months. Other authors have also addressed the concept of kindling in relationship to the physiologic basis for the development of PTSD (11-13).

COGNITIVE DEFICITS IN TRAUMA

The DSM-IV criteria for the diagnosis of PTSD do not adequately take into account the complex interaction of emotional and somatic experiences in an MVA. Some investigators have advocated use of the designation of a subsyndromal form of PTSD in MVA victims (14). Their criteria for this form specify inclusion of criteria for Section B (reexperiencing), and either C (avoidance) or D (hyperarousal) to establish the diagnosis. Even acknowledgement of a subsyndromal form of PTSD may also not fully take into the account the fact that a remarkably prolonged freeze response, or period of posttraumatic dissociation, may mask many of the symptoms of arousal and reexperiencing for an indeterminate period of time, thereby discouraging the diagnosis of PTSD. Under these circumstances, the predominant symptoms may be those of numbing, distraction and cognitive symptoms usually attributed to a concussion, or minor traumatic brain injury (See Chapter 3). Even in the absence of any conceivable head trauma, PTSD has been associated with substantial cognitive deficits of a severity and similarity sufficient to make the diagnosis of a head injury on neuropsychological test batteries (15,16). 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 (17,18,19). These cognitive deficits have a solid theoretical base in the neurohormonal changes triggered by trauma. Trauma has been shown to interfere with declarative memory, but not nondeclarative or procedural memory (1). This phenomenon appears to be related to the input of norepinephrine to the amygdala, allowing the brain to differentiate the emotional meaning of the incoming information. High activity within the amygdala inhibits storage and synthesis of declarative memory within the hippocampus while facilitating storage of memory linked to the norepinephrine-induced arousal (3). In addition, release of high levels of endorphins as part of the response to this arousal further interferes with memory consolidation (20). The result is a state of impaired declarative memory storage in the face of enhanced storage of trauma-related memory, most of it nondeclarative. In addition, high level of cortisol have a direct inhibitory, and in fact, neurotoxic effect on hippocampal structures, enhancing the inhibitory effect on declarative memory (21,22,23). The result is the exaggeration of trauma-related memories that in fact become intrusive at the expense of conscious declarative memory processes, and contribute to the documented memory deficits in PTSD (15).

As noted, endogenous opioids may also be involved in memory dysfunction in PTSD. Stress-induced analgesia is a well-known accompaniment of severe trauma. Soldiers with severe wounds have been noted to deny the need for morphine for some time after their injuries (24). This is consistent with previously mentioned studies that show that both norepinephrine and endorphins are

released after exposure to severe stress. Animal studies reveal that animals unable to escape a threatening situation and who exhibit withdrawal/despair- i.e. the freeze response- suffer from significant impairment of memory (20). In these animals, both the freeze response and panic interfered with memory processing, suggesting that both epinephrine and endorphins contribute to this deficit. van der Kolk and colleagues have postulated that the freeze/numbing response in animals exposed to prolonged severe and inescapable stress may be analogous to dissociation in humans exposed to trauma, and that dissociation may also be mediated by endogenous opiates (2). Excessive endorphin release may therefore play a role in the well-documented deficits in memory noted in patients with PTSD. Traumatic memory intrusion may also contribute to the documented deficits in attention, concentration, and multitask thinking in PTSD. In addition, arousal triggered by recurrent and kindled traumatic memories induces release of norepinephrine that once again activates the memory/arousal feedback circuit as well as the Hypothalamic/Pituitary/Adrenal axis, and sustains the cognitive deficits of PTSD. These concepts challenge the assumption in the Whiplash Syndrome that specific and documented cognitive dysfunction is by definition indicative of traumatic brain injury, especially in low velocity accidents.

TRAUMA AND THE POST CONCUSSION SYNDROME

Another matter of concern in the concept of concussion is the varied symptom complex commonly referred to as the post-concussion syndrome. This is a vague group of complaints which typically includes atypical headache, visual complaints usually involving blurring of vision, balance disturbance, tinnitus, dizziness and vertigo, orthostatic lightheadedness, and mood disturbances. These mood changes have been recognized to contain many of the symptoms of PTSD, and include irritability, stimulus sensitivity to noise and lights, sleep disturbance, fatigue and depression. Researchers have begun to recognize this association between PTSD and the elements of the postconcussion syndrome (18,25). In MVA-related concussion, all of the symptoms associated with post-concussion syndrome can be explained by the concept of traumatization. Autonomic activation perpetuated by a truncated freeze discharge facilitates the linking of procedural memory associated with various stimulated end organs to the kindled feedback circuit incorporating arousal and traumatic memory. The end organs involved include the extraocular muscles of the eyes, the vestibular apparatus of the inner ear and brainstem, the hearing apparatus of the inner ear, the autonomic nervous system and its control of peripheral blood vessels, and the muscles of the head, neck and shoulder girdle.

BINOCULAR DYSFUNCTION

As we have noted, under the influence of peripheral epinephrine and brainstem norepinephrine, the eyes will diverge at the moment of trauma or immediate threat, and the pupils of the eyes will dilate. This takes place by reflex activation of the extraocular muscles and the circular muscles that make up the iris of the eye. This response is as automatic as the stretch reflex, and serves to maximize the field of vision in the situation of threat or danger. This ocular reflex inevitably accompanies any arousal threat, including that of a MVA. As with other events associated with arousal, it is frozen in the event of the freeze response, and dissipated by its physiological discharge in the event of survival after freezing. Applying the concept that retention of traumatic arousal occurs because of impaired freeze discharge, the ocular and pupillary changes triggered by traumatic arousal will continue to be linked in the evolving feedback circuit between centers of arousal and those for procedural memory, the storehouse for motor skills and habits. Under these conditions, any arousal, whether linked to the other memories of the accident or even occurring in the stresses of everyday life will nonspecifically trigger ocular divergence and pupillary dilitation. In fact, these are basically the clinical findings that have been documented in examinations of patients with post-concussion vision abnormalities (See Chapter 3). Such patients almost inevitably are found to have impaired binocular movements and convergence insufficiency representing the persistence of variable tonic ocular divergence. In this model, however, these changes are not the result of brain injury per se, but rather the incorporation of the eye muscles in the PTSD neuromuscular/arousal/memory conditioned and kindled circuit.

VESTIBULAR AND AUTONOMIC DYSFUNCTION

The same theory applies to all of the remaining manifestations of the Whiplash Syndrome. Because of the intense forces of acceleration and deceleration in a MVA, the vestibular and proprioceptive sensory apparatus of the inner ears and musculotendenous system are subjected to massive sensory input. Once again, linking of arousal and memory to the conditioned activation of the vestibular system results in the appearance of vertigo, dizziness and balance disturbance under conditions of arousal. In addition, any quick movement of the head results in vestibular input. Since the particular and unique vestibular input at the time of the MVA is stored in procedural memory linked to arousal, movements of the head which coincidentally access the MVA-related movement pattern will trigger arousal and evoke the sensation of vertigo and loss of balance. So-called “cervical vertigo” may therefore arise from muscles of the neck involved originally in bracing patterns of the accident. Activation of bracing and movement patterns involving these muscles may arouse procedural memories of the accident which are also linked to coincident vestibular activation, thereby repeatedly inducing

vertigo with head movement. This may explain the absence of objective findings in many cases of positional vertigo in MVA's.

A labile and unstable autonomic nervous system is known to accompany PTSD. The primary measures in chronic cases involve documentation of unstable pulse and blood pressure responses to nonspecific arousal stimuli, sounds, pictures or even smells reminiscent of the trauma, or even guided imagery of the trauma. Electrodermal Skin Response has also been used with some success in identifying PTSD autonomic overresponders (26). Victims of PTSD are known to cycle in and out of arousal and dissociation, the former associated with adrenergic dominence, the latter with endorphinergic and possibly cholinergic tone. In the early stages after an MVA, an exaggerated sympathetic response may be at its most dramatic. I have documented many patients with systolic blood pressures over 170 mmHg with no history of hypertension, and with pulses of 120-140 at rest. Most of these patients have been in acute arousal and anxiety, with many symptoms of the arousal part of PTSD. On the other hand, those patients presenting with characteristic of sustained freeze and dissociation will generally present with low systolic blood pressure, occasional bradycardia, and frequent orthostatic hypotension and dizziness. Actual syncope is not uncommon in these individuals. Many of these dissociated patients will suffer from dramatic alternating constipation and diarrhea, and occasionally from symptoms of peptic acid disease. Autonomic lability, I believe, is a secondary, conditioned phenomenon linked to the arousal/memory feedback circuit of traumatization, with numbing and the dissociation of the freeze response often associated with cholinergic symptoms.

NEUROMUSCULAR DYSFUNCTION AND MYOFASCIAL PAIN

The other end organ system involved in traumatization in the Whiplash Syndrome, one that is totally ignored in consideration of MVA-related PTSD, is the neuromuscular system. Post-traumatic headache, cervical and lumbosacral spinal pain constitute the primary symptoms related to whiplash. This pain has been attributed to injuries to muscles, tendons, ligaments, intervertebral discs, joints and nerve roots (27). In the final analysis, however, the prolonged and intractable pain of whiplash is eventually attributable primarily to the condition of myofascial pain. The relationship of myofascial pain in the Whiplash Syndrome to the arousal/memory link in trauma is quite analogous to the involvement of other end organ systems with procedural memory for the traumatic event. At the instant of impact, selected muscles are stretched in a coordinated and synchronous pattern depending specifically on the direction of the initial acceleration of the body. Input from the stretched muscle spindle initiates contraction of alpha muscle fibers through the initiation of the gamma motoneuron system at the level of the spinal cord. At the instant of this selective grouped alpha muscle activation, proprioceptive input from the associated

musculotendinous units travels to the cerebellum via group I and/or group II afferent fibers. Input is then provided from cerebellar nuclei to the thalamus, brainstem vestibular nuclei and basal ganglia where reflex regulation of complex postural changes in response to the changes in velocity takes place through descending pathways affecting the anterior horn cell alpha and gamma motoneurons.

In addition, input from the thalamus to the amygdala facilitates assessment of the emotional content of the experience. Since the sudden velocity changes accompanying the MVA carry the implication of imminent life threat, arousal via brainstem norepinephrine pathways will accompany thalamic input to the amygdala, setting up the scenario of the full-blown fight/flight response. If physiologic freezing accompanies the completion of the MVA, as it often does, the experience of the complex neuromuscular response to the velocity forces of the accident will be incorporated into procedural memory in its exact form, just as will any learned motor skill. In addition, it will be reinforced by its association with high arousal, and therefore will be relatively permanent (28). Finally, it will be incorporated into the kindled arousal/memory circuitry of traumatization. Thereafter, memories of the accident, familiar stimuli reminiscent of the accident, dreams of the accident, arousal related to those memories, and eventually nonspecific arousal will tend to facilitate procedural neuromuscular memory of protective muscular bracing patterns from the MVA. Reflex activation of muscle groups will then be produced in a pattern mimicking that associated with the movements of the accident. The MVA victim will experience involuntary tightening of a selective group of regional muscles in a repetitive pattern, triggered by arousal, dreams, driving activities or memories of the accident. Repetitive use of those muscles in any other activities may trigger arousal and reflex muscle spasm. Since muscles are designed to contract briefly on a reciprocal basis with their opponents, involuntary sustained contraction of muscle groups on the basis of arousal-generated reflex input from postural centers of the brain sets up a condition of energy failure. This leads to an accumulation of metabolic waste products in muscle fibers, release of kinens and other chemical pain generators, and a condition of relative ischemia in the involved fibers. The end result is regional myofascial pain perpetuated by the same kindled feedback circuitry producing the symptoms of PTSD.

CEPHALIC MYOFASCIAL PAIN

In any MVA, the muscles of the head, neck and jaw are invariably the most involved in residual long-term regional myofascial pain. Whether the mechanism of the accident involves a rear-end, head-on or rollover impact, neck pain, headache and jaw pain usually are inevitable. In the velocity-change model of physical injury to these structures, the cause is felt to lie in the pendular effect of the skull on the neck, rendering these structures more vulnerable to damaging

forces. This does not, however, explain the same phenomenon in low velocity accidents, or when the head and neck were not subjected to unusual forces based on the particular dynamics of the accident. Another explanation lies in the intimate neural association of the locus ceruleus with sensory end organs and especially joint proprioceptors of the head and cervical spine which provide this critical center of arousal with information about environmental threat through positional orientation of the head and its sensory apparatus. The orienting reflex, a gradual sided to side rotation of the head allowing scanning of the environment for information utilizing all of the sense organs of the head, is a basic and universal instinct in all species. Muscles of the head and neck are therefore intimately involved in sensory information access in all situations, both with regard to feeding and to fight/flight survival. Activation of cephalic and cervical muscles with associated bracing in response to threat renders them uniquely vulnerable to the conditioned inclusion of this bracing in the arousal feedback circuit, with subsequent persistent cervical myofascial pain. Jaw clenching is a primitive arousal reflex, with its roots lying deeply in the instinctual pattern involving use of the teeth as both offensive and defensive weapons in animals. Unconscious bruxing after an MVA therefore is incorporated into the nonspecific protective muscular bracing patterns linked to unresolved arousal, leading to the well- known but perplexing condition of TMJ syndrome.

CONCLUSIONS

Clinical experience suggests that many victims involved in MVA's experience dissociation, or freezing, at the moment of the accident. Dissociation is known to be a major predictor of eventual development of PTSD, and is felt to be the equivalent of the freeze response seen in animals. For reasons as yet unclear, the human species, unlike creatures in the wild, tends not to go through the stereotyped and instinctual neuromuscular discharge of the autonomic arousal of the freeze response in the face of trauma.

The physiologic model of kindling presents a compelling rationale for the symptom complex of PTSD. Stored autonomic energy from a truncated freeze response might well provide the impetus and fuel for development of kindling in trauma. The experimental model of kindling, of course, entails the application of an external stimulus to trigger development of the resulting self-perpetuating circuitry. In this case, the external stimulus is experiential related to sensory input rather than to external application of an electrical impulse. Involvement of arousal and procedural memory circuitry is clearly implicit in the model of traumatic kindling. Linking of these centers with those of the sensory end organs involved in the traumatic experience would predictably result in a cyclical and kindled repetition of somatic symptoms representative of the intense sensory input experienced at the moment of trauma. This process provides a unitary hypothesis for the myriad symptoms of whiplash. It also provides a model for the concept of somatization, oneof the more prominent comorbid conditions seen in PTSD.

In this model, myofascial pain derives from arousal-activated descending motor input to the anterior horn cell from reflex motor centers of the brain, linked to sustained arousal associated with unresolved discharge of the freeze response. The specific distribution of the involved muscles is linked to anatomical and physiological procedural memory patterns of the neuromuscular defensive bracing response to the MVA. Those muscle groups activated by the stretch reflex to contract in response to the velocity changes in the accident would continue to brace in the face of arousal or memory input related to the trauma. As a phenomenon of central rather than peripheral origin, this hypothesis would explain the remarkable lack of consistency or specificity of findings with investigation of the peripheral portion of the motor unit in myofascial pain using electrophysiological, chemical, and biopsy studies. It would also explain the frequent regional persistence of myofascial pain, and its variable lack of response to peripheral forms of treatment.

Similarly, vestibular, auditory, visual and autonomic symptoms of the post-concussion syndrome may represent somatic experiences of the MVA linked to procedural memory and arousal circuitry, and perpetuated by any head movement reminiscent of the trauma, by nonspecific arousal or by declarative memory of the accident. Although focal brain or end organ injury certainly may contribute to some of these symptoms, their frequently delayed onset and occurrence in low velocity accidents supports their etiology in traumatization.

Although minor traumatic brain injury may be a cause of specific and sometimes persisting cognitive deficits in MVA's, cognitive impairment in the Whiplash Syndrome is also explainable by the mechanisms of dissociation, attention deficit and thought intrusion seen in PTSD. In very low velocity accidents, these physiologic events associated with trauma are more than likely the primary etiology for significant cognitive symptoms and impairment.

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