

Robert Scaer's Neurobiological Model for PTSD and Psychosomatic Illness

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With his hypotheses about trauma and illness, neurologist Dr. Robert Scaer breaks or breaches enough conventions within both our field and the broader field of medicine and provokes just about everyone. While quite specific, his concepts and vocabulary operate in a space where psyche and soma, while distinct, are at the same time always part of an interactive continuum. In this sense, his work can be seen as a development of the dialectical unity and duality of psyche-soma proposed years ago by Wilhelm Reich (1973). Additionally Scaer's formulations, like Reich's, move across the boundaries of established specialties within medicine and in so doing, challenge established theory and practice. Scaer also borrows heavily from ethology in his formulations. In justification of drawing analogies between humans and sub-primate mammals when facing a life threat, he notes their common possession of a similar brain stem. This brain stem, or as Paul MacLean ('73) called it, "reptilian" unconscious part of our brain, is where the trauma-related somato-sensory cues that perpetuate the process of traumatization are stored. Finally, Scaer's formulation rests on a history of childhood traumatic abuse in the patient. As you know, there is controversy in our field regarding the accuracy of memories that are not accurately verified. Since these histories of physical, or sexual, or neglectful abuse are often not spontaneously supplied by the patients, Hillel might be the first to suggest the power of Dr. Scaer's suggestion in eliciting the data,. As if this were not enough for Hillel, many of these memories are apparently "recovered" (you will pardon the expression), in the course of these patients' trauma therapy. And to add a little more insult to anyone not already injured, Scaer, like the other popular fellow I just mentioned, Wilhelm Reich, is much more interested in the early researches of Breuer and Freud on hysteria than in Freud's later work in which his earlier theories of sexual abuse and incest in hysteria were recanted. In fact, I would say that Scaer views himself as continuing the neuropsychiatric tradition of Janet, Breuer and Freud. He credits these three for being the first in the emerging field of neuropsychiatry to give us a biopsychosocial understanding of trauma. In their 1893 series of case studies and papers, *Studies in Hysteria*, Scaer (2005) tells us that:

"... Breuer and Freud presented concepts that with uncanny accuracy reflect current concepts of dissociation and the manifestations of old traumatic experiences stored in procedural memory as sensations and motor behaviors. Indeed, they asserted that "hysterics suffer mainly from reminiscences" (Breuer & Freud, 1957, p. 7). They noted that these sensorimotor symptoms of hysteria actually reflect the physical experiences of an old, suppressed original trauma rather than only the physical experiences of the new "accident" or traumatic event that seemed to trigger the hysteria." (pp. 187-188)

Having said this, I find Scaer's work fascinating, and it has helped me to better comprehend, or at least get a glimpse of something cohesive in the otherwise disparate and obscure stories and

symptoms of a number of my patients. Since very little if any of his theory and practice has been proven correct by rigorous empirical studies, I, may be suffering from the illusion that I am less befuddled by some of my patients. But consider that if any of the far-reaching conclusions in his two books are accurate, you heard about it here first! Then again that may be one more problem some of you have with the books. There is very little that is not explained by his theories. He has ideas, some of which he acknowledges are more speculative than others, about the late effects of unresolved PTSD that include hypertension, cardiovascular disease and even immune disorders. Thankfully, today I am only trying to expose you to a vignette or two and his central formulation.

In his two books, Robert Scaer (2001, 2005), a practicing neurologist for 35 years, proposes the hypothesis that what he calls the “whiplash experience” (2001, p. 33) be taken as a model for traumatization, specifically for PTSD. As you may already have inferred, his hypothesis does not stay within the boundary of physical as distinct from psychological trauma. Rather it operates on a body/brain/mind continuum; he believes he has described a common set of neurophysiological and neurochemical processes which occur along a brain/mind/body continuum. His clinical insights culminate in positing a common underlying pathoneurobiology for both PTSD and a variety of poorly understood and difficult to treat somatic or medical illnesses. A short autobiographical section in his second book indicates that, like many notable figures in our field, Scaer’s professional interests and beliefs are inspired by his specific life story. A quick glance at the two outlines I have passed around will tell you that there is much more in the two books than I can report on today. I will limit myself to attempting to convey the essence of his hypothesis on the whiplash experience as a general model for posttraumatic illness.

Scaer dedicates his first book (2001) to George Engel, his teacher, who he says “taught [him] that the diagnostic truth lies more in the uninterrupted story told in its entirety by the patient than in a dozen diagnostic tests’.

SCAER (2001), FIRST VIGNETTE

When Beth first came to my office, she brought her best friend, a nurse, “to interpret for her.” Beth was quite guarded, almost suspicious, with wide, frightened eyes. She sat with her arms held tightly at her side, holding a handkerchief, her head forward, and her shoulders raised and tense. Her forehead was furrowed, and at twenty-five, she looked closer to forty. She said that she had suffered whiplash in an accident, and was not getting any better. A friend had referred her to me because of my interests in motor vehicle accident-related injuries. Her insurance company was beginning to question her treatment, since the accident had occurred at speeds of 5 to 10 mph. No damage occurred to either car, and the driver of the other car suffered no injuries. Beth, on the other hand, continued to suffer from terrible neck and shoulder pain, numbness of her right arm, disabling headaches, and temporomandibular joint syndrome. Six months of chiropractic treatment and massage therapy brought her only temporary relief from pain, and a dental splint only slightly helped her morning headaches. Recently she developed severe pain in her right hip and buttock, with the pain radiating down the back of her leg. X rays and MRIs failed to show any cause for her spinal or leg pain. (p. xvii)

Let me break in to wonder if your differential diagnosis at this point includes malingering, factitious disorder, or one of the somatoform disorders such as conversion disorder, hypochondriasis or somatization disorder? But Scaer has more information for us:

Since the accident, Beth had also experienced worsening problems of distraction and an inability to concentrate. Her memory was terrible, and she would constantly go somewhere and forget what she had intended to do. She had recently started to drive again, and would travel to some familiar place in town, and on arriving, realize that she could not remember how she got there. She would stumble over words, say the wrong word for what she meant, and then feel stupid. She even had developed a strange stutter whenever she was stressed. She constantly made errors in her checkbook.

Worst of all, since the accident, she had become panicky when traveling in a car, especially as a passenger. Her heart would pound for no reason. She had become edgy and irritable, and jumped at every loud sound. Thoughts and images of the accident kept popping into her mind, producing distraction and anxiety. Although she finally was able to fall asleep without drugs, she kept waking fully

aroused, sometimes with a racing heart, sometimes with dreams of the accident or of being threatened with no means of escape. During the day she was exhausted by every physical and mental effort, and had dropped out of her master's program in clinical social work. Her extreme sensitivity to almost any stimulus had resulted in her isolation from almost all of her prior social activities.

She admitted that at times she was worried that she might have a brain tumor. She experienced dizziness when she moved her head too quickly, kept losing her balance and bumping into things, and noticed that her vision blurred whenever she moved her focus from one object to another. Reading caused blurring of vision and a headache. Only an MRI of her head reassured her that she had no diseases of the brain. (pp. xvii-xviii)

Let me break in to note that we have not been told if there are external incentives for the patient to be feigning illness. Some of you may be thinking that hypochondriasis is sounding less likely, and that she has some of the complaints seen in post-concussion syndrome. But you are also thinking that Beth's symptoms fulfill the DSM-IV criteria for a Posttraumatic Stress Disorder (309.81). She re-experiences, she avoids, and she is hyperaroused. A puzzling feature, however, in her story is the apparent trivial nature of the MVA in which she was involved. As we know, the DSM-IV requires that the individual "experienced, witnessed, or was confronted with an event or events that involved actual death or serious injury, or a threat to the physical integrity of self or others." (Desk ref. DSM-IV, p. 209B) the person's response to that event should involve "intense fear, helplessness, or horror"(desk ref. DSM-IV, p.209 It was the regular occurrence of a chronic, disabling syndrome such as Beth's following apparently minor MVAs, that led Dr. Scaer to the hypothesis that what he calls the "whiplash experience" be taken as a model for traumatization per se, and specifically for PTSD.

In his thirty-five years as medical director of a rehabilitation clinic, Scaer has of course seen patients with documented ruptured cervical discs, nerve root injuries, and ruptured neck ligaments - patients who suffered impacts in excess of thirty miles per hour - and who generally recovered rapidly with no long-term disability. Beth, however, represents a group of MVA patients whose accident involved minimal, in some cases, no impact. These patients showed a delay in onset and a worsening over time in their symptoms. Their wide variety of problems included both spinal and neurological symptoms and typical emotional symptoms suggestive of trauma, dissociation and PTSD.

In these patients, rather than physical and structural damage to the spine, jaw and brain as the basis of whiplash-based injury, Scaer proposes the whiplash experience as an experienced-based model of traumatization. Specifically, he (in line with the "intense fear, helplessness, or horror"

stipulated in DSM-IV) invokes the meaning (experience of helplessness) of any event to an individual in determining whether it proves to be traumatic for them. Furthermore, Dr. Scaer points out that PTSD is generally considered to be a purely psychiatric diagnosis. He notes that:

Much of the medical literature tends to ignore that often-dramatic physical symptoms, including bowel disorders, myofascial pain and cognitive impairment, accompany PTSD in a fashion often identical to that experienced in the whiplash syndrome. We therefore seem to be dealing with a syndrome affecting all aspects of a person's being, including body, mind and spirit. This posttraumatic syndrome is produced by threat, shock or injury that occurs in a state of helplessness, and the neurophysiological changes triggered by these experiences are somehow stored in a person's unconscious (procedural) memory in a cumulative manner along with other such life experiences. Accumulation of traumatic life experiences then leads to a condition of increasing vulnerability and decreased resiliency to further trauma. (pp. 20-21)

But, in line with Scaer's suggestion that the patient's story has a somatic or bodily dimension, I have a treat for all of us, we are going to return to Beth, the puzzling MVA patient and pretend that we are doctors who work in a psychosomatic or somatopsychic manner that harkens back to whenever it was that neuropsychiatrists did neurological exams on their patients:

When I examined Beth, she was quite guarded and defensive, exhibiting what one might call "exaggerated pain behavior". She manifested "give-away" weakness of her right arm and leg, and "stocking and glove" anesthesia of these extremities, as well as loss of sensation to pin prick over the entire right side of her head and face. Remarkably, however, her right hand was slightly cooler to touch than the left, and patterns of hair growth revealed subtle but definite thinning over the right side of her scalp. Her neck was quite resistant to passive movement, and neck extension immediately caused her to become nauseated. Tightness, tenderness, and trigger points were prominent in her neck, jaw, and shoulder girdle muscles, and straight leg-raising caused pain in her right hip and tingling of her right foot.

So, as Beth's body tells more of her story, the physical, neurological part of the story, which George Engel would have the patient tell us in its entirety, are you more or less satisfied with somatization as a diagnosis? Should we settle for her having internalized or converted some sort of emotional/psychological issue into physical symptoms? Are we in an unaccustomed place? As psychiatrists, we normally get a report from the internist, neurologist, gastroenterologist, that no adequate medical condition/explanation has been found for the patient's complaints, and we may conclude that the patient is somatizing, as we put it.

Dr. Scaer offers an explanation for these findings on Beth's physical exam as both patterns of arrested reflex muscular bracing from the moment of a traumatic event and then later cyclical regional, autonomic changes in various end organs of the body which provided sensory information about a perceived life threat - information which then became part of a self-reinforcing arousal/memory circuit stored in procedural memory.

In this situation, however, our neurologist, Dr. Scaer, is being difficult. He is not satisfied that either whiplash or post-concussion syndrome adequately describes Beth's problem. He leans toward a diagnosis of a complicating PTSD, but with van der Kolk, he stresses ongoing somatic consequences of exposure to traumatic stress. I must tell you though that although he feels that Beth's symptoms are more than can be explained by her MVA, per se, Dr. Scaer has not called on our expertise, because he does not believe in the diagnosis of somatization or, for that matter, somatiform disorders as they are currently defined. As you know, somatization disorder is defined in DSM-IV as a diagnosis of exclusion. A specific number of pain, gastrointestinal, sexual, and pseudoneurological symptoms that have resulted in significant impairment of function, cannot be fully explained by a known general medical condition or the direct effects of a substance. Or, if there is a related general medical condition, the physical complaints or resulting impairment are in excess of what would be expected from the medical work-up. The DSM-IV does not conjecture as to the cause of the disorder, but it is generally accepted that painful emotion, stress, or psychological issues are somehow internalized or expressed as physical symptoms. The pathophysiology is considered to be unknown, so boldly or foolishly, Scaer (2001) is stepping into this vacuum regarding MVA patients, somatization disorder patients, and another group of patients with poorly understood medical conditions – all of whom account for a substantial share of our national health care costs- and proposing a pathophysiology. He says:

We believe that these conditions (somatiform disorders) , as subset of dissociation, are actually defined by demonstrable vasomotor changes in the dissociated regions of the body. It is not enough to say that somatization reflects expression of an emotion in a somatic symptom, or that conversion represents a symptom in the absence of a medical condition. Rather, psychosomatic syndromes appear to reflect subtle but demonstrable physiological changes in dissociated regional end organs based on changes in autonomic tone, dependent in turn on trauma-related central and autonomic nervous system kindled responses, and mediated by vasoconstriction. (p. 125)

Let me say that, even if some of Scaer's proposed pathoneurophysiological mechanisms for the wide-variety of conditions he discussed in the two books turn out to be validated empirically, it is not likely to be easy to change neural circuitry operating at the level of procedural memory.

Do not be impatient with me if I only now give you another piece of the patient's story, the anamnesis. Perhaps I wanted you to empathize with Dr. Scaer (2001) who, for years, was baffled by patients like Beth, until he began to ask in detail about past traumatic life experiences:

Similar to many of my patients, she was very open and candid and willing to share with me what must have been a hellish nightmare of her childhood. She was supported in this discussion by the confidante and friend who had accompanied her. From the age of six through twelve, she was visited twice each week after bedtime by her brother, who was older by seven years. As if by ritual, he would painfully twist her right arm behind her back as she lay in bed on her stomach, pull down her panties, and rape her. Any sound by her would result in his beating her, or covering her head with a pillow as he abused her. She never told her parents for fear of punishment by her verbally abusive alcoholic father. At age nineteen, her brother ran away from home and the abuse stopped. During college, memories and panic related to the abuse resurfaced, and she underwent years of counseling, which enabled her to "put it all behind her." She obtained her degree, and, prior to the accident, had been pursuing a graduate degree with the goal of becoming a social worker (p. xviii).

Let me attempt to summarize the neurophysiological-neurochemical model of traumatization that Scaer is proposing. As Scaer sees it, MVA patients such as Beth, and others who present with symptoms of PTSD following a variety of traumatizing experiences, have a specifically relevant history of been abused physically, sexually or otherwise, as children. The original experience of helplessness, (i.e. no effective way to fight or flee) led to a freeze or dissociative response, mediated by endorphins and predominantly parasympathetically driven. Depending on the details of the abuse, the sensory messages and aborted motor responses to the threat were encoded in the child's procedural memory. These memories, involving the sensorimotor and autonomic nervous systems are conditioned responses. They are critical to the unconscious, survival-based behavior of the organism. Linked to a threat to life and occurring in a state of high arousal, they become conditioned quickly and are resistant to extinction, often lasting a lifetime. Concepts such as kindling and irradiation help to explain why with time, these conditioned, trauma-based procedural neural circuits will be triggered by ever less specific internal and external cues. We are all familiar with the patient whose panic attacks are triggered by the sensation of his heart beating vigorously while he jogs. This patient would be understood by Scaer to have cued, via his own strong heartbeat, a conditioned procedural circuit linked to the high arousal and fear of a prior traumatic experience. What may be less familiar to you is "Somatic Dissociation" (pp. 97-126) – a concept that Scaer (2001) invokes to explain a great variety of otherwise poorly understood syndromes. His focus is on the part of the body whose sensori-motor input has been incorporated

in the arousal response (often the end organ (skin, muscles, eyes),, and then in the kindled circuitry of procedural memory.

SOMATIC DISSOCIATION

This material bears a bit of repetition and further illustration. Paraphrasing Scaer (2001):

In this model, the end organs involved in receiving the necessary information for reflex self-protective behavior in an MVA are primarily those sensory centers of the head and neck. These sources of sensory input include the extraocular muscles of the eyes, the vestibular apparatus of the inner ear and brainstem, the hearing apparatus of the inner ear, the a.n.system and its control of peripheral blood vessels and the muscles of the head, neck, and shoulder girdle. SENSORY INPUT FROM THESE END ORGANS AT THE TIME OF A WHIPLASH WILL BE INCORPORATED IN THE AROUSAL RESPONSE AND THEN IN THE KINDLED CIRCUITRY INVOLVING AROUSAL AND PROCEDURAL MEMORY FOR THE EVENT.

Scaer (2001) explains the predominant traumatic impact at the cephalic or head end of the organism:

These sensory receptors provide... information about environmental threat through positional orientation of the head and its sensory apparatus. The orienting reflex, a gradual side-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 instinctual motor pattern in all species. Muscles of the head and neck are therefore intimately involved in all sensory information access in all situations, both with regard to feeding and to fight/flight survival. (p. 53)

I would add that not just the head and neck, but any part of the body, such as:

1. The skin or gastrointestinal tract affected by high autonomic arousal, or
2. Any localized muscle group involved in bracing against and reacting to physical threat/abuse, can become part of the above self-reinforcing arousal/memory circuit stored in procedural memory.

Additionally, while Scaer arrived at this trauma model from motor vehicle accident patients, I find that he has given the field of somatic psychotherapy a model for psychosomatic illness that begins to do justice to the complexity of the human organism. In future publications I will address the integration of his work with my work on cephalic shock (Lewis, 1976, 1984, 1986, 1998, 2007). I will also address the application of Scaer's highly specific and nuanced pathoneurobiology to the patients whose trauma has not been gross and dramatic, but rather the cumulative poorly

attuned, moderately seductive parental handling which has hard-wired a sustained vigilance to their own internal body sensations and reflex motor behavior. Such a patient will not, for instance, relate a dramatic story of having been on many occasions nearly suffocated inadvertently as a small child by his drunken and obese mother. The triggering and early traumas of Scaer's patients related in his first book (2001) are by and large single and dramatic. His second book (2005), however, stresses the lifelong impact of cumulative adversity. He tells us that:

If a sensory message is repeated often enough in this group of people, the message itself is likely to kindle and become incorporated into the vast assembly of procedural memories of body movements, sensations, and experiences that are linked to threat. Any repetitive movement of the body that is similar in any way to past thwarted defensive body movement will be especially likely to trigger reflexive bracing. (p. 234)

The above paragraph suggests to me an additional specific and enriching perspective to that given us by Reich (1949) and Lowen (1975). In the latter model the spastic, chronically braced musculature is holding conflicted impulse and intolerable affect. The integration of these perspectives is, however, beyond the scope of this paper.

SCAER (2001), SECOND VIGNETTE

One such patient was referred to me because of partial vision loss in her right eye following an auto accident two months prior. She had not experienced a blow to her head. The patient had lost control of her car on an icy mountain road, and as the car spun out of control, it slid off of the right side of the road into a shallow ditch. On the other side of the ditch was a cliff. As she watched the edge of the cliff approaching from her right side (perceived in slow motion of course), her car was stopped by impacting a tree, and came to rest. Thereafter, she developed persistent blurring of vision in her right eye. Ocular refraction showed a consistent refractive error that appeared to be correctable, and she was fitted with a lens that provided clear vision in that eye. Within minutes after putting on the glasses with the new lens, she developed nausea, palpitations, flashback memories of the accident, and panic. Any further attempts to restore or correct the measurable refractive error in her right eye continued to produce attacks of panic and re-experiencing of the traumatic event through flashbacks. Her visual impairment was diagnosed as being hysterical in nature.

This remarkable patient presents an example of this group of symptoms. The measurable but physically unexplained refractive error in her right eye was clearly a somatic, dissociative response driven by the moment of life-threatening danger perceived in that eye at the time of the MVA. Visual images perceived in her right eye activated procedural memory for the terrifying events of the potentially fatal accident and triggered emotionally valenced flashbacks and panic. Blurring of vision in that eye became a protective dissociative phenomenon. The unusual feature of this case, of course, is that the refractive error was consistently measurable and reproducible. Dissociation had resulted in a probably temporary but physiologically measurable alteration in the dissociated end organ. The occurrence of this type of visual distortion in MVAs usually appears related to the association of visual cues accompanying the threat at the moment of the accident. (pp. 104-5)

This patient illustrates what Scaer (2001) refers to as the rupturing of boundaries by traumatic stress. He defines boundaries in this context as:

Our unconscious proprioceptive awareness of a spatial zone containing a zone of safety and wholeness, not only as it relates to emotions and relationships but also to the tangible, as well as the intangible, material world. (p. 118)

As specialists in reenactment, we are probably not surprised to hear that Scaer's MVA patients are very prone to be involved in further MVAs. But, as always, Scaer's (2001) clinical detail and inferences are intriguing:

This increased risk in traffic probably is related to distraction resulting from dissociation, as well as suppression of attention and visual perception in regions of boundary rupture. These areas of boundary rupture are usually defined and regional, and often are identifiable as developing in the perceptual region where the threat was first identified... The localized area of the body that first experienced an impact of blow, the surrounding area from which the person heard the threatening noise, or the direction in which the body was forcibly thrust might all potentially create an area of impaired boundary continuity. (pp118-119)

SUMMARY DISCUSSION

As I've said, I believe that even a small part of what Scaer has addressed in these two books, makes a worthwhile presentation. The case vignettes and clinical commentary reflect his extensive experience and his clinical theory is informed by an impressive grasp of current neurophysiology. As my own work is centered in a somatic psychotherapeutic approach, I particularly appreciate Scaer's rather pioneering attempt to shed light on the complex and still too mysterious mind/brain/body continuum.

Having said this, I am still concerned with two aspects of his hypothesis. The first is that his data set is too heavily sampled with patients who have experienced dramatic, relatively isolated traumatic events, whether MVAs or incidents of childhood abuse. The second is a central part of his theory, which I have mentioned, called the freeze or immobility response. He has borrowed this from ethology, and it refers to the prey animal that collapses when death in the jaws of its predator is immanent. Commonly known as 'playing possum' (and technically known as tonic immobility), this is a complex state in which the animal becomes limp, immobile, frozen. There is a dramatic drop in heart rate and blood pressure, and the muscles collapse. The animal is inert, but its parasympathetic and endorphinergic activity is high. Scaer, building on the work of Van der Kolk (2001, p. 17) and Peter Levine (1997), postulates this state as analogous to PTSD in humans, specifically to somatic dissociation. When the predator does not kill such animals, we are told that they instinctively dissipate the stored energy of their aborted fight or flight response in various stereotyped somatic and autonomic responses, including trembling, perspiring, and deep breathing. Traumatized human beings, on the other hand, tend not to dissipate their frozen response following the traumatic event, and this in Scaer's theory is a crucial factor in the perpetuation of sensory-motor messages of life threat, first acquired to warn the unwitting victim of the original trauma, but now triggering useless reflexive self-protective motor responses, pain and much more. I have no reason to doubt Scaer's suggestion that he has been personally helped to reduce his own trauma-induced vulnerabilities via a therapy that guided him towards the discharge and completion of an aborted "involuntary motor response" which, analogously to the prey animal, was the reflexive attempt at self protection during the traumatic event.

While this borrowed ethological model is compatible with some of the ways in which I work, I find that it lacks the complexity required to capture the situation of most of the patients I see in my practice. Many of them - even those who suffer from the somatic symptoms and conditions to which Scaer refers - do not remember episodes of crude and dramatic abuse; or if they do, these episodes are imbedded in and emerge from a complex matrix of chronically impaired parenting. This ethological model seems to have led Scaer to some fascinating propositions about PTSD and the aforementioned otherwise poorly understood illnesses. While no model is as complex as reality,

I doubt if an ethologically-based view can address the disorganizing experience with which many of my patients struggle - that is, of having been betrayed both by their primary attachment figures and their own DNA. I refer to the survival instinct that programmed them as children to seek proximity to an unsafe, that is, insecure parent.

But finally, Scaer's insights suggest strongly that many of the procedural memories that are at the crux of posttraumatic syndromes are sensory messages and motor reflexes. Cognitive-behavior therapy tells us that through repeated exposure in a benign environment, habituation progressively diminishes the fear/arousal response in PTSD. Scaer (2001) tells us that:

Ultimately, one must gain access to the insidious conditioned trauma response from a physiological and unconscious reflexive approach in order to extinguish, desensitize, inhibit, or quench it (p. 161)

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