TOWARD A UNIFYING THEORY OF POST-TRAUMATIC STRESS DISORDER: Integrating Data from Studies of Post-Traumatic Behavior, Memory, Symptom Formation, Physiology, Cerebral Imaging, Psychoanalytic Findings and Evolutionary Theory

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Abstract

PTSD is considered a biologically altruistic, gene-pool survival enhancing reaction to life threat. Analogy is drawn to aspects of the cellular level of immune process. Information-laden circulating proteins act as signals replicating some of the molecular characteristics of the invaders. Though the individual cell may be handicapped by immune response, the community of cells usually profits. Similarly, when life-threat is perceived by the human individual, memory focuses on that threat and is occupied by producing replicas. These are intrusive involuntary processes: thoughts, flashbacks and nightmares with details of the life-threat. Behavioral sensorimotor memory enactments occur about details of the threat. Though individual behavior, memory and perceptual life are thus impoverished, survival of surrounding individuals and the gene-pool profit from the behaviorally transmitted information. This gene-pool value was present before the development of language. Therapeutic implications are discussed.

Introduction

Studies of post-traumatic disorders were a long-time focus for Freud, Breuer and Janet (Miller, 1991) and had a neurophysiologic conceptual basis. But in today's modern neurophysiological study climate, psychoanalysts and other theoreticians must consider much more than the scientific world Freud tried to encompass. Today we must deal with concepts of chaos theory, fractalization, neural membrane thresholds, kindling, neuro-endocrine aspects, potentiation, facilitation, downright neuronal deaths, dendritic pruning, loss of synapses, and gross atrophy of parts of the brain, to name a few sets of information. This essay will begin to consider such concepts and relevant data which must be unified in order to have a comprehensive theory of disorders resulting from psychological trauma.

Whenever there is an effort, such as this, to integrate diverse areas of data, the author's biases and frameworks should be immediately disclosed. Much of the data and previous theory have been developed by others and some data and theory by himself. The author's interests were at first physiological, for example his review of literature on catecholamines in infants, older humans and other mammalian species suggested a combined cognitive and psychophysiological etiology for the human infant's crisis of seven months "stranger anxiety" (Kliman, 1953). He has a long-standing interest in the psychoanalytically informed study of psychological emergencies as a potential area for illuminating pathogenesis with special clarity (Kliman, 1968). Since 1963 he has been engaged in clinical, forensic and research activities focused on traumas. These have emphasized psychoanalytic applications of research and service to catastrophically traumatized persons. Those persons are mostly young children and their families.

In 1963, the author founded a bereavement study project and recruited the collaboration of Martha Wolfenstein in the Albert Einstein College of Medicine Study of Young Children's Reactions to Death of a Parent (Kliman, 1968; Kliman, 1970; Wolfenstein and Kliman, 1965). In 1965, the author founded the Center for Preventive Psychiatry (Kliman, 1975; Zelman, 1996). That Center has in effect provided a clinical laboratory for the study of reactions to trauma, as well as the basis for a vigorous child analytic training program, feeding into the Association for Child and Adolescent Analysis. It has become the nation's largest center for the treatment of bereaved, molested and otherwise traumatized preschoolers. It also houses the nation's largest crises service for traumatized persons of all ages involved in a datable trauma. Almost 3,000 persons per year now come to that Center, mainly very young children.

As the Center for Preventive Psychiatry's service grew, it became possible to develop a few rigorous and systematic projects by which interventions may cast light on the etiologies of reactions to stress and trauma. One of the most scientifically rigorous is the delivery of controlled and manualized preventive services to populations of traumatized children. In particular, the author has been working on data from two projects with a combined total of over 800 consecutive children entering foster care, of whom about 90 who have been treated individually (Kliman, 1982; Kliman and Schaeffer, Kliman 1990).

In 1993 the author founded a Children's Psychological Trauma Center in San Francisco. Data from numerous children impressed me and changed my view of the etiology of PTSD and were consistent with emerging biological data. The current essay was begun when reporting (1992, 1994, 1995) on a series of ten children exposed to life-threatening events. Their diminished memory functioning was especially manifested by an inability to recall much quantity of early memories. But there was a qualitative change in memory functioning also, a change of quality to negative rather than pleasantly charged ones. The change of memory quality is manifested by a hyperfocus on sadistic and aggressive event memories, with minimal retrieval of tender, consoling, or pleasantly nurtured events.

It was predicted in my 1992 presentations (Horowitz Seminar and IACAPAP that formal psychological testing of earliest memory would be a means to show impoverishment of early childhood memory among adults abused as children. Since then, as a result of formal early memory testing studies at the Wayne State Department of Psychiatry, there is now evidence that age of onset of early childhood memory is later among traumatized adults than among other psychiatric patients. Further, there are other "negative" phenomena involving cognition among PTSD patients. In addition to the early childhood memory range deficit there are short term attentional deficits and I.Q. deficits. (Bremner JD, Scott TM, Delaney RC, et al (1992) These are all greater among adults psychologically abused as children than among control patients. The list of negative phenomena of posttraumatic stress disorder should now be considered to include focal hippocampal atrophy, short-term memory deficits, impoverishment of retrievable early childhood memories, and diminished cognitive abilities. The cognitive deficits are both attentional and information retrieving (Sutker PB, Winstead DK, Galina ZH, et al,1991).

Observations about Childhood Memory Following Catastrophic Life-threat

Most of my series of videotaped interviews can be viewed by scholars who visit The Children's Psychological Trauma Center. The great majority of over 100 consecutively videotaped families have given permission for scientific and educational review. The tapes show assessments of and data concerning early memory and current behavior. Interviews are on hand concerning the following ten undisputedly and catastrophically life-threatened children.

1. a child severely burned at 15 months, and interviewed at two and a half years.

2. a kidnapped and raped six year old, now age ten.

3. & 4. two physically uninjured toddlers both of whose parents were killed instantly in a car in which he was a passenger, interviewed five and seven years later.

5. a physically crushed toddler whose mother was killed instantly in the car in which he was a passenger, interviewed two years later.

6. a boy whose penis was feloniously amputated, and left for dead at age seven, interviewed at age ten.

7. a child who swallowed lye at age 18 months, interviewed at age six.

8. a homicidally assaulted two year old, now age five.

9. the older sister of the second child, a percipient witness to her disappearance, interviewed five years later at age 12.

10. a child who narrowly escaped an explosion of her automobile, and witnessed a charred corpse, interviewed two years later at age 10.

The children, to varying degrees, all show phenomena I consider abnormal for their current ages:

A. Pre-traumatic Amnesia:

This is an impoverishment of the quantity of memories in the period before age five and/or at least the period within one to two years prior to the trauma.

B. Retrospective templating of memory by trauma:

Impoverishment of benevolent memories, specifically, so that malevolent, sadistic, masochistic, painful, or terrifying events predominate in the bank of what is recalled prior to age five.

Some of the children also show a related phenomenon: traumatic impoverishment of social perception. They are vigilant for more traumatization, and view many ordinary social transactions as malevolent. They are inattentive to benign transactions, nurturant opportunities

are not cultivated, and they are unreceptive to new complex benevolent experiences. Learning is secondarily inhibited by a disinterest in benevolent transactions with authorities.

What needs to be encompassed in a unifying theory of Post-Traumatic Reaction to Catastrophic Events

A. Definition and Features of the Disorder

Post-Traumatic Stress Disorder is a diagnostic entity well defined in DSM-III-R and DSM-IV (1993). For purposes of my unifying theory, I regard the pathologies as comprising three categories: (1) intrusive phenomena, (2) avoidant or numbed behavior and experiences, and (3) physiologically aroused phenomena.

Intrusive pathologies are also conceptualized as "positive." They include anxiety, nightmares, and involuntarily retrieved memories such as flashbacks and unsought, unwelcomed or repetitive trauma-related thoughts and behavioral enactments. In children, the behavioral enactments are particularly clear and common.

In contrast to intrusion, the avoidant pathology is "negative," with constriction of attention and memory, amnesias for parts of the etiologic events, reduced social and recreational functioning, constriction of interests and desires, and loss of conviction of being fully present in the here and now. Dissociation is considered an avoidant or numbing manifestation. Loss of interest in customary hobbies, work and recreation as well as in customary personal relationships are symptoms of avoidant/numbing type. Oppositionalism (Wayrnenen and Kliman, 1992) is also a "negative" or constricting behavior.

The third category, autonomic arousal, is also "positive" and could be collapsed with the intrusive phenomena. It includes excessive perceptual vigilance, excessive wakefulness, startling and physiologic reactivity when exposed to stimuli that symbolize or resemble aspects of the original danger. There is intense psychological arousal accompanying the physiological arousal, and conversely, as well as great distress upon exposure to events that symbolize or resemble some part of the danger-event, and such distress is in proportion to the amount of physiological reaction. The physiologic reactions are primarily catecholamine outpourings, and arousal of the pituitary-adrenocortical axis, together with central nervous system arousal. The central nervous system arousal is emphatically limbic, especially of the hippocampus and particularly of certain nuclei such as the locus ceruleus. Imaging studies show that when the disorder is chronic, these over-aroused areas become atrophied—particularly the right hippocampus (Bremner, 1995) and, in addition, there is some less significant frontal lobe atrophy. The negative PTSD phenomena I consider associated with the brain atrophy, especially in the right hippocampus, are reduced short term memory and reduced memory for early periods of childhood. As I pointed out in earlier presentations, there are diminished quantity and quality of early childhood memory amongst child survivors of catastrophic traumas.

How do these negative phenomena occur, leading to impoverishment of the very basic functions of attention and retrieval of memory? It seems likely there are several mechanisms at work, minimally. Probably there is some exhaustion effect on neurotransmitters.

1 - Neurotransmitter exhaustion, resistance, and toxicity:

The chronic overactivity of noradrenergically rich areas, such as the locus coeruleus, leads to complex effects, possibly including receptor-resistance, down-regulating, and arboreal depletion with some nuclear atrophy of the involved cells. Thus the patients who have chronic autonomic arousal, chronic intrusive thoughts, elevated pulse rate and hypervigilance, ultimately suffer some atrophy of the involved limbic system. This end-result, atrophy, can be seen in MRI studies. But the functional overactivity of the areas which are ultimately atrophic can best be seen in PET imaging studies (Bremner 1997) in which PTSD patients are exposed to trauma-specific scripts. Their limbic systems light up in ways not matched by other psychiatric patients and controls.

2 - The kindling, potentiating, facilitating, and then apoptotic effects of chronic neuronal stimulation:

In PTSD, there is a cycle of self-perpetuating reactions to reminders of the original life-threat. These involuntarily and repeatedly practiced and self-perpetuating states produce a form of constant physiologic readiness for fight or flight. The catecholamines, cortisol, dopamine, opioids and other substance poured out improve survival prospects in aggression-loaded situations, such as combat or assault by a predator. The catecholamines, in particular, increase the circulating platelets, improve the heart's efficiency, reduce bleeding and promote healing. (Kliman, 1952) The endorphins released increase tolerance for pain from wounds and ready the body's responses should life-threatening events re-occur (van der Kolk, B., 1994).

Bodily memory in the form of autonomic hyperreactivity to reminders of the original trauma is well documented (5). Years after a danger-event has passed, there is often chronic circulatory readiness for flight or fight, chronic hypervigilance for similar events, and chronic physiologic hyperreactivity to events that resemble as aspect of the original traumatic event. Sleep is light, allowing for easy awakening by danger.

Apoptosis is a phenomenon possibly induced by the hypercortisolemia of PTSD. Apoptotosis, or neuronal death, has been noted as an effect of high cortisol levels in animals.

Effects on Memory

Semantically accessible memory is disordered in various ways. Patients have verbalizable preoccupations with a danger-event. Patients are extraordinarily easily led to remember the danger-event. Memory of the danger-event intrudes into dream and markedly into behavior. Short term and long-term memory are deficient in PTSD according to systematic studies. Annecdotal findings show an impoverishment of pre-traumatic semantic memory, with a simplification of narratives concerning the pre-traumatic epochs. (Kliman, 1992) Among holocaust survivors and others who live after massive trauma, much of pre-traumatic life is irretrievable by conscious efforts at recall (Krystal, Tauber, 1996).

B. General Biological Considerations

The cellular level is a good place to begin this consideration. Throughout all known species, from bacteria to higher organisms, there appears to be some generalization of cellular protein stress responses. Trauma leads to strengthening of the assaulted cell, and often to communication with other cells. Cells given a mild heat shock, or exposed to heavy metals, alcohols and various other metabolic poisons respond with such similar changes in gene expression that researchers started referring to the accompanying products as "stress proteins" (Welch, 1993). Animal cells given a mild heat shock "one sufficient to increase the levels of the stress proteins—become better protected against a second heat treatment that would otherwise have been lethal. Moreover, those thermotolerant cells were also less susceptible to other toxic agents...the protein stress response somehow protected cells against varied environmental insults." Ischemic damage, for example, is better resisted...it turns out that the stress proteins created by numerous bacteria and parasites which invade human beings are often the major antigens that the immune system uses to remember, recognize and destroy the invaders. The human immune system is thus aided in its constant vigilance for alien forms of stress proteins (Welch, 1993). Welch could well have added that communication occurs among cells regarding dangers, and that there is knowledge spread among cells by the creation of protein responses to intruders. Cells "learn" to "know" and "avoid" trauma.

It is my thesis that this vigilance, this repetitive searching for and communication about patterns signifying danger, and the communicative aspects of response are also part of the higher levels of animal behavioral response to perceived life-threatening trauma. That is especially true when the trauma is life-threatening.

Just as in immune responses, psychological responses can be overdone. When overdone, neurophysiological vigilance tends to become harmful to the individual. However, I postulate that while the individual may suffer from excessive recruitment of its behavior by vigilant search and communications about patterns of danger, there are benefits not only to individual survival but also to members of the victim's gene pool. The price to the individual is a gross impoverishment—as I will detail below—of action, adaptation, memory, and intrapsychic life. The price is altruistically paid, and the gene pool benefits with a higher survival rate than it would have without the information made available by the post-traumatic behavioral narrative.

Towards a Unified Theory of Post-traumatic Stress Disorder

All of the above data must ultimately be taken into account by a unifying theory. The theory we develop here will conclude that while the individual suffers from the pathology following life-threatening events, the species profits. The features of the disorder can be understood as a communicative process, well-suited to pre-linguistic animals and to pre-linguistic epochs of our species. All of the following are pre-linguistic communications which are part of post-traumatic stress reactions: specific danger-associated avoidance features, specific trembling and sweating-on-reminder associations, olfactory consequences of fear-specific sweating, agitation, irritability in trauma-related circumstances, hypervigilance for specific threats, and the specific teaching of

others by sweaty avoidance of formerly dangerous places. All of these behaviors have a socially signaling and informative value, providing danger-alerting data for the community. The post-traumatic syndrome is inclusive of a kind of pantomimic story-telling process, carrying survival-related memory into action, and is advantageous to the viewers' survival.

It is considered probable by the author that post-traumatic behavioral communication evolved because it helped the gene pool and should not be considered simply a disease. Failure to consider the "bearing witness" value of specific behaviors could lead to miscarriage of treatment. Similarly, treatment procedures have to take into account the handicapping secondary deficits of benevolent memory and perception of social benevolence which occur when the victim has become reduced into a living icon, who confines himself too much to communicating about the specific danger experience.

Psychoanalytic theory of post-traumatic reactions has not considered much about the evolutionarily useful value of danger reactions, although Freud was fully cognizant of the value of fear and the signal function of anxiety for the individual's survival (Freud,). Psychoanalytic consideration of trauma-induced disorders began largely with Janet and Freud, and was heavily neurophysiologically based at first. The modernity of Freud's ideas that hysterical conversion symptoms were a behavioral memory of traumatic experiences is not sufficiently appreciated. Freud was highly interested in neurology and neurophysiology, evident in his work on cerebral palsy and his invention of a gold stain for the fine structures of neurons (Miller, 1991). Freud's first theories of psychological trauma contained concepts which are still relevant to modern neurophysiological findings and concepts. He postulated a threshold of neuronal stimulation by traumatic experience beyond which damage is done to the mental apparatus, something like today's kindling concept.

Psychoanalytic theory of post-trauma reactions — especially a hypothesized process called "turning passive into active," better called "the repetition compulsion"—has been partly operationalized in at least one effort (Kliman, 1993). Following this operationalizing, an intervention was mounted to interfere with that measurable form of the repetition compulsion. That measurable form of unconsciously organized and driven behavior is called "repetitive bouncing." Prior trauma or rejection or abandonment leads to "bouncing" (high transfer rate) among foster homes. Transfer rate data was gathered concerning carefully matched pairs of foster children, treated and not treated. These treated children were given brief, intensive, structured therapies to increase their sense of the continuity of personal history, and to facilitate healthy mourning for lost caregivers. From these studies, a personal life history book method has evolved which is now a replicable and manualized method of psychotherapy for traumatized children. The largest study of 225 children as well as a more fully controlled study (20 matched pairs) both utilize that personal life history method (Bondy, 19xx, Kliman, 1988), indicating that personal narrative functions can be enhanced. This effect of a personal narrative enhancement is thought to cast light on the etiology of the repetition compulsion, indicating that the cause includes a remediable deficient memory of personal history.

Instead of simply enacting their stories of prior abandonments, the children are led from sensorimotor enactments into semantic forms of communication, and their memories are given practice in recalling benevolent experience while creating the personal life history book.

The "bouncing" manifestation of repetition compulsion is widespread among foster children. Foster children who are ejected or rejected from their biological families bring templates of rejection-related behavior into their new homes. Largely through their stereotypic or icon-like behaviors they induce one rejection after another. They bounce from home to home at an approximate 25% rate in the first year of care. Yet each form of treatment given in our several studies dramatically reduces this rate, whether the treatment is psychoanalysis five times a week by a trainee, or an application of psychoanalysis five times a week in a therapeutic nursery setting, or individual psychotherapy, or parent guidance, or educational psychotherapy, or a therapist administering 30 sessions of a personal life history book treatment, or treatment in groups (Kliman, 1988; Bond, 1988).

Thus, we find that the repetition compulsion, which so harmfully narrows a traumatized child's behavioral repertoire, can be stopped. And the methods which stop it have in common a tendency to deliberately expand the child's personal historical narrative so as to include many nontraumatic events, ranging from remote past to benevolent transference experiences.

Another factor in our own studies influencing our theory is the finding of behavioral simplification in various forms among traumatized children. Their behavior is not only repetitious, it is reduced in repertoire of qualities. Cruel experiences are the main qualitative category.

Richness of experience is often vigorously avoided and made further unlikely by the post-traumatic state of oppositionalism. I made this an area of close scrutiny. Findings in a study of a behavioral scale of oppositionalism among sexually abused children showed a highly significant presence of that trait, differentiating them sharply from other children (Wayrynen, 1993; Kliman and Wayrynen, 1994).

Oppositional behavior is considered to be part of a generally harmful constriction or narrowing of behavioral repertoire. It has a survival purpose, teleologically speaking, an apparent defensive purpose of reducing complexity of tasks when in a survival mode, and reducing opportunities for reception of the harmful input of others. Unfortunately, it reduces learning conspicuously, especially about benevolent experiences.

Chronic endangerment by physical abuse in childhood is often responded to by general numbing of reactivity, and a numbing of cognitive life. It is useful to compare the physiological responses of physically abused children to different stimuli with responses of children in a reference group and to correlate the physiological responses with intellectual and personality functioning. Abused children were compared by Carry (1995) to a reference group on two batteries of tests that were administered on separate days. In one session, children were shown slides with emotional or cognitive content while heart rate, pulse height, skin conductance, electromyography, and skin temperature were measured. In the other session, intellectual and personality functioning was measured using the WISC-R, Quick Neurological Screening Test, and the Junior Eysenck personality inventory. Results showed that abused children had smaller changes in pulse height in the first two stimulus conditions presented ("No Signal" and "Math"), but their electrodermal responses were lower throughout all stimulus conditions. Abused children also had higher

introversion and lower Verbal and Full Scale IQ, higher introversion and lower Verbal and Full Scale IQ scores. Verbal and Full Scale IQ scores were inversely related to the severity of abuse that had been experienced. When these variables were used in a discriminant function analysis, children were assigned to the correct group 86 percent of the time. These findings support a model that describes the effects of abuse as substantially reducing cognitive development (Carrey NJ et al, 1995).

Again, another psychoanalytically derived intervention is relevant to our theory-building. We also had the impression, better documented by Carrey (1995) that cognitive deficit is unusually common among traumatized preschoolers. An intervention by our "Cornerstone Method" with a variety of traumatized preschool child patients treated in therapeutic nurseries shows that measurable cognitive functional deficits can be significantly reversed. The Cornerstone Method, a very intensive application of psychoanalytic technique daily in a therapeutic nursery for traumatized preschoolers showed a marked cognitive advantage for the treated children. Their I.Q.'s rose an average of 12 points when serial testing was performed on 42 preschoolers treated by this method, versus a lack of I.Q. gain for 10 children treated by other method. The children who gained most were those with moderate trauma. Those with severe trauma histories were already relatively inaccessible. Also, children who were three and four years old gained more I.Q. points than older preschoolers, suggesting there are unfortunate developmental limits to recovery from trauma-induced
cognitive defects.

Mechanisms of divided attention and emotional flooding during the initial traumatic experiences may explain some of memory findings in PTSD, particularly partial psychogenic amnesia. Such difficulty registering and/or processing segments of long-term memory of the event is a product of initial hyperarousal with resultant attention deficit. Later concentration and short-term memory difficulties are secondary to chronic anxiety and distraction by intrusive imagery. Cognitive research reveals that blocked focal attention leads to impaired explicit processing, yet intact implicit recall. The hippocampal region is crucial for explicit memory processing, involving a subsequent consolidation process that is postulated to make memories "permanent" in the cortex. Treatment approaches are expected to lead to resolution of traumatic states and processing of impaired memory concerning the trauma. Success in such therapeutic endeavors may depend upon rehabilitation of impaired hippocampal memory processing. (Siegal DJ, 1995). The narrative practice and reconstruction of benevolent memories advocated in our Personal Life History Book method is a form of such deliverate rehabilitation, focusing on the avoided positively toned past, trying to compensate for the reduced access to such danger-irrelevant pathways.

Findings in a series of ten catastrophically, life-threateningly stressed children seen in forensic interviews are of special interest to us and have necessarily biased our theory-building (Kliman, 1996). Videotapes of the catastrophically stressed children, even year after the life-threatening events, show a marked tendency to behavioral use of the child's self as an icon, a living sign of what happened. To the great detriment of the individual child victim, the life-threatening event can be viewed repeatedly by family and others in proximity to the child. While the onlookers gain survival-useful information about a threat the child has experienced, the child's behavior is maladaptively constricted by the iconic behavioral replaying of the dangerous event.

Pre-traumatic Amnesia

A further part of data from the forensic videotaped interviews studied is that the catastrophically traumatized, life-threatened child victims suffer from remarkable impoverishment of pre-traumatic memories. Childhood amnesia seems to spread excessively over the pre-traumatic epochs of the victims memories. Benevolent experiences are particularly lacking among the pre-traumatic memories. It is therefore our theory that a hippocampally or otherwise limbically mediated memory filtering or template has taken over pre-traumatic memory. As part of the response to life-threat, memory retrieval is limited and simplified it to fit the task of response to life-threat. The task-oriented response causes exclusion of benevolent-content information.. When the mechanism become chronic, as in chronic limbic arousal, impoverishment of quantity of pre-traumatic memory is accompanied by shifting of retrieval to those memories which have danger related qualities. What might appear to be a libidinal or psychosexual regression to sadomasochistic preoccupations is thus also a pathology of memory retrieval due to a permanent state of emergency memory processing (Kliman, 1992, 1994, 1995, 1996).

Anatomic and Physiologic Aspects of Memory Encoding

Traumatic experience may become encoded into memory differently than non-traumatic experiences. Neuroanatomic studies of post-traumatic stress disorder patients show atrophies of areas in the hippocampus and frontal lobes, consistent with findings of disorders of memory. I believe these atrophies are part of a chain of pathophysiologic adaptations, in which the high norepinephrine production and high cortisol levels cause synaptic loss and even neuronal deaths, leading to the atrophies noted. In turn the atrophies contribute to impoverishment and simplification of long-term as well as short-term memory retrieval. Animal studies show that those same brain areas are atrophied in hypercortisolemia.

P300 wave activity in the EEG is generally considered an indication of event processing. P300 has been recorded in 26 subjects (15 women) one month after a traumatic aggression which occurred without organic brain complications. Among the sample, 16 subjects fulfilled DSM-III-R criteria for PTSD and 10 did not. P300 amplitude was significantly lower in the 16 PTSD subjects as compared to the 10 subjects without PTSD. This study supports a theoretic view that information processing is disturbed in PTSD. (Charles G, 1995).

Studies of memory confirm there are pathologies of short-term memory retrieval and long-term memory retrieval as well. In the long-term memory pathology, there is a significant loss of childhood memories, accompanied by a moving of childhood amnesia into later parts of childhood. PTSD patients scored significantly lower on the total recall, long-term storage, long-term retrieval, and delayed recall measures of the verbal component of the Selective Reminding Test and on the recall, long-term storage, long-term retrieval, and continuous long-term retrieval measures for the visual component of the Selective Reminding Test (Bremner et al, 1993).

As Friedman (199) states, "the neurobiological basis of some of the symptoms of PTSD can be understood in the context of neural circuits and neural mechanisms of anxiety and fear.

Memories and previously learned behaviors influence responses to fear-inducing stimuli via such neural mechanisms as fear conditioning, extinction, and sensitization. Although with the medial temporal lobe memory system, emotional responsiveness (amygdala) and memory (hippocampus) may be separately organized, there is considerable interaction between storage and recall of memory and affect. This is exemplified by the critical role of the amygdala in conditioned fear acquisition, sensitization, extinction, and the attachment of affective significance to neutral stimuli. The hippocampal memory system is essential to short-term memory. However long-term memory storage may be organized such that, as time passes, with subsequent additional retrieval opportunities and the acquisition of related material, the role of the hippocampus diminished until it may no longer be necessary for memory. The repository of long-term memory may be in the same areas of cortex where the initial sensory impressions take place. The persistence of intrusive memories may be due to the strength of neuronal interactions between cortical region where many such memories are stored and subcortical region, such as the amygdala, which serve to attach affect to the memories. The psychological distress and physiological responses to trauma reminders involve the mechanisms of fear conditioning and extinction." (Friedman, p. 281).

The hippocampus is involved in control of neuroendocrine systems, learning and memory and emotional states dysfunction is thought to contribute to the symptoms of depression. Chronic stress leads to alteration of the structure and survival of cells in this brain region. Repeated stress causes atrophy of certain populations of stress-sensitive neurons, the CA3 pyramidal cells. The atrophy or death of these neurons results from several factors, included sustained elevation of adrenal-glucocorticoid levels and decreased levels of brain derived neurotrophic factor (BDNF). BDNF and other nerve growth factors are critical for maintaining the normal function, as well as the survival, of neurons. (Duman, 1996).

Summary and Conclusion

My unifying theory is that post-traumatic reduction of complexity in memory is part of the process by which the PTSD survivor becomes a living icon, communicating about and bearing witness to danger for the sake of his kin and community. Using mechanisms which are archaic and pre-linguistic in their evolutionary sources, the PTSD patient is doing a job to save others, and is doing that job with ancient tools and at great personal expense. He is reduced to pantomime, autonomic expression, and behavioral sign behavior, compulsively enacting his danger experiences. He displays diminished recall of or cultivation of benevolent experiences. He busily concentrates on stereotypic communication of danger. His potentialities for development as a person are stopped short and kept simple. He cannot recall much to learn a nontraumatic perspective from his past, and is limited in what he learns from his future. Perception of social reality is altered in the same survival-templated way as memory. Perception, including social perception, becomes vigilant, but impoverished and templated to filter out what does not fit the prelinguistically communicated story of the life-threatening trauma. The victim behaves energetically by seeking present and future dangers to avoid or re-enact the past. Both his avoidant vigilance and behavioral reenactments are informative to others. Like a gun-shy animal who jumps at every loud noise and trembles when he avoids the smell of human hunters, his pre-linguistic but information-loaded behavior is informing others of his species on the scene.

His avoidant and enacting behavior involuntarily inform his gene pool of details of the life-threatening events which templated his mind. Through reenactments of the content and general story of the danger, he shows what and where to avoid being near, thus teaching others to survive.

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