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The Trauma Spectrum: The Interaction of Biological and Social Events in the Genesis of the Trauma Response

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When Kardiner first described the full syndrome of what is now called PTSD in 1941, he called the trauma response a "physioneurosis," that is, a mental disorder which affects both the soma and the psyche. Now, more than 40 years later much knowledge has been gained about the biological effects of traumatization. Based on the studies of disruptions of attachment bonds in non-human primates, the animal model of inescapable shock, and numerous studies of traumatized children and adults, we are beginning to understand the nature of the biological changes which underlie the psychological response to trauma. This paper will explore (1) the nature of the biological alterations in response to traumatization, (2) how these biological shifts depend on the maturation of the central nervous system (CNS), cognitive processes, and the social matrix in which they occur, (3) and how these alterations can influence psychopathological and interpersonal processes.

KEY WORDS: post-traumatic stress disorder; psychobiology; attachment; hyperarousal; memory; addiction to trauma.

INTRODUCTION

When Kardiner first described the full syndrome of what is now called PTSD (post-traumatic stress disorder) in 1941 (Kardiner, 1941), he called the trauma response a "physioneurosis," that is, a mental disorder that af-

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fects both the soma and the psyche. Noting that sufferers from PTSD continue to live in the emotional atmosphere of the traumatic event, he ascribed to them an enduring vigilance for and sensitivity to environmental threat. A stressor becomes traumatic when it overwhelms both psychological and biological coping mechanisms. This paper will examine how closely the psychological and biological dimensions of traumatization are intertwined and explore (1) the nature of the biological alterations in response to traumatization, (2) how these biological shifts depend on the maturation of the central nervous system (CNS), cognitive processes, and the social matrix in which they occur, (3) and how these alterations can influence psychopathological and interpersonal processes.

THE NATURE OF THE BIOLOGICAL SHIFTS ASSOCIATED WITH TRAUMA

Post-traumatic stress disorder has been described as phasic alteration between intrusive and numbing responses (Lindemann, 1942; Horowitz, 1976). This consistent pattern of hyperarousal alternating with numbing has been noticed following such a vast array of different traumas, such as combat, rape, kidnapping, spouse abuse, natural disasters, accidents, concentration camp experiences, incest, and child abuse, that it is reasonable to assume a biological substrate (van der Kolk, 1987). Contemporary research suggests that the intensity of the initial physiological response to the trauma is the most significant predictor of long-term outcome (Kilpatrick *et al.*, 1985; Holen, 1987; McCahill *et al.*, 1979). Freud (1919) may have been on the mark when he postulated that "the traumatic neurosis [is] a consequence of an extensive breach being made in the protective shield against stimuli." During the second world war, Grinker and Spiegel (1945) observed that the severity of "combat fatigue" was related to what we now understand to be symptoms of catecholamine depletion, such as masked face, cogwheel rigidity, and tremor. Kolb (1987) has recently reviewed the numerous studies which demonstrate increased autonomic arousal in combat veterans with PTSD. The enduring nature of this physiological hyperarousal has repeatedly been documented in response to sounds (Dobbs and Wilson, 1960; Malloy *et al.*, 1983; Kolb and Multipassi, 1982), symbols (Pitman *et al.*, 1987) and visual stimuli (van der Kolk and Ducey, 1985; Fish-Murray *et al.*, 1987) reminiscent of the trauma. Chronic increased reactivity has been demonstrated in other civilian trauma victims as well. Kilpatrick (Kilpatrick *et al.*, 1985) found persistent "generalized signs such as nervousness, tension and trembling . . panic attacks and apprehension and dread . . . which leads to avoidance and escape behavior in three follow-up studies of rape victims." In both a 1- and a 5-year follow up of survivors of an oilrig disaster in the North Sea, Holen (1987) found that the severity of the initial biologically based symptoms, both hyperarousal and dissociative phenomena, were the

worst predictors of long-term outcome. Clinical experience suggests that the increased autonomic arousal is rather nonspecific, and may be seen in response to a large variety of stimuli. In fact, habaituation can occur to the original traumatic stimulus, while associated events are met with hyperreactivity (Strian and Klicpera, 1978). In PTSD, autonomic arousal is no longer a preparation for, but a precipitant of emergency responses which bear little relationship to the nature of the contemporary stimulus. This results in further autonomic arousal, anxiety, and even disorganization of thought processes (van der Kolk and Ducey, 1984; Fish-Murray *et al.*, 1987). In addition to hyperarousal, traumatized people have repeatedly been noted to have an all-or-nothing response to emotional stimuli. They have an impaired capacity to modulate the intensity of their emotional responses, be they anxiety, anger, or intimacy. Physiological changes can, in fact, account for most posttraulatic symptomatology: (1) the tendency to react to relatively minor stimuli as if there were a recurrence of the trauma: startle reactions and irritability, which interfere with an adequate cognitive assessment of current events and result in fight/flight/freeze reactions; (2) visual and motoric reliving experiences: nightmares, flashbacks, and behavioral reenactments generally are preceded by physiological arousal (Rainey *et al.*, 1987) which activates long-term potentiated memory pathways (Gold and Zornetzer, 1983); (3) persistent hyperarousal also can account for the avoidance and escape behavior characteristic of people with PTSD, psychologically as a defense, physiologically as downregulation of receptors in response to overstimulation (van der Kolk, 1987); (4) compulsive reexposure to circumstances reminiscent of the trauma may be related to both the neophobia seen in previously traumatized animals under stress (Mitchell *et al.*, 1985) and to the conditioned endogenous opioid release seen in traumatized animals and humans (Kelly, 1982; Christie and Chesher, 1982).

THE ANIMAL MODEL OF INESCAPABLE SHOCK

Since the biological building blocks of human beings and of our mammalian relatives are closely related, particularly in regard to such relatively uncomplicated reactions as fight, flight, and freeze behaviors to external danger, our knowledge of animal responses to life threatening situations has important implications for understanding the biological adaptation of people to inescapable stress. The animal model of inescapable shock (IS) provides an opportunity to understand the physiological response in PTSD and the biological substrate of its psychological effects. Exposure to inescapable aversive events has widespread behavioral and physiological effects in animals, including (1) deficits in learning to escape novel adverse situations, (2) decreased motivation for learning new contingencies, (3) evidence of chronic subjective distress (Maier and Seligman, 1976), and (4) increased tumor genesis and immunosuppression (Visintainer *et al.*, 1982). It has been estab-

lished that the helplessness syndrome seen after exposure to inescapable shock is due to the lack of control that the animal has in terminating shock and that the behavioral and biochemical sequelae of escapable shock tend to be in the opposite direction of those of inescapable shock (Weiss *et al.*, 1975).

Biochemical Changes following Inescapable Shock

IS results in a large variety of biochemical changes, including alterations in norepinephrine (NE) and dopamine (Anisman *et al.*, 1981), serotonin (Sherman and Petty, 1980), and endogenous opioid (Maier *et al.*, 1980) utilization. This altered biochemical response is thought to be due to the fact that, under severe stress, there first is a massive secretion, and a subsequent depletion of a variety of neurotransmitters, presumably because utilization exceeds synthesis (Anisman *et al.*, 1981). After repeated or prolonged stress, NE, opioid, and serotonin depletion become conditioned responses, which eventually can stimulate receptor hypersensitivity (van der Kolk *et al.*, 1985; Kraemer, 1985), leading to excessive responsiveness at times of subsequent stress.

The Relationships between Biochemical and Psychological Effects of IS

There are intriguing similarities between both the biochemical and the behavioral sequelae to traumatization of animals in response to IS and what we know about the biochemistry of the human response to overwhelming trauma. The behavioral sequelae of catecholamine depletion following IS in animals closely parallel the negative symptoms of PTSD in humans. Van der Kolk *et al.* (1985) and Kolb (1987) have proposed that the diminished motivation, decline in occupational functioning, and the global constriction seen in PTSD are correlates of a relative catecholamine depletion. The clinical symptomatology of hyperreactivity (with startle responses, explosive outbursts, nightmares, and intrusive recollections) coincides with the establishment of chronic noradrenergic hypersensitivity following transient catecholamine depletion after acute trauma in animals. Direct evidence of chronic noradrenergic hyperactivity in PTSD has recently been found (Mason *et al.*, 1985). Serotonin is thought to be the neurotransmitter most involved in modulating the actions of other neurotransmitters (Blier and de Montigney, 1985) and has been implicated in the fine tuning of emotional reactions. For example, it has been proposed that a relative lack of serotonin is responsible for the lack of buffering excessive noradrenergic responses to stress in people with manic-depressive illness (Bunney, 1984). Low serotonin in this illness may set the stage for the extreme swings in cerebrospinal fluid (CSF) NE found in that disorder (Bunney, 1984; Brown *et al.*, 1982). It also may be the reason why primates with early separation experiences

have sharply elevated CSF NE responses to stresses later in life (Kraemer *et al.*, 1984). The fact that medications that have been proven to be effective in bipolar iillness, such as Lithium Carbonate, and Carbamezapine, also positively affect the all-or-none response in people with PTSD (van der Kolk, 1983; Blier and de Montigney, 1985; Lipper *et al.*, 1986) lends further support for a role of serotonin in PTSD.

Intrusive Reexperiencing

Visual and motoric reliving experiences, nightmares, flashbacks, and reenactments generally are preceded by physiological arousal (Rainey *et al.*, 1987). We have postulated that a long-term augmentation of memory tracts following trauma underlies the repetitive intrusive reliving of the trauma under conditions of subsequent stress. Noradrenergic stimulation of locus coeruleus-hippocampal pathways at the time of the trauma can set up hyperpotentiated pathways which are reactivated at times of subsequent arousal, particularly during periods of cortical inhibition, such as during intoxication or sleep (Delaney *et al.*, 1983; Squire, 1987).

The immediacy with which trauma victims react to stress is familiar to all clinicians who deal with this population: Rape victims experience intense anxiety in situations reminiscent of the trauma; Vietnam veterans may misinterpret the movements of a sleeping bed partner as a Viet Cong attack and react accordingly (van der Kolk *et al.*, 1984); mild noises played into the rooms of sleeping people with PTSD may precipitate nightmares in which old traumatic occurrences are recreated in exact detail (Kramer *et al.*, 1984). An illustration of how autonomic arousal is associated with flashback phenomena was provided by a former parachutist who had a three month period of symptoms of PTSD after his second parachute failed to open until he was a few hundred feet above the ground. Five years later the only remaining symptom of PTSD is a flashback of this event after exposure to another severe stress such as a near car accident.

PTSD and the Endogenous Opioid System

Animals exposed to inescapable shock develop Stress Induced Analgesia (SIA) when reexposed to a subsequent stressor within a brief period of time. This analgesic response to prolonged or repeated stress is mediated by endogenous opioids, and is readily reversible by the opioid receptor blocker naloxone (Kelly, 1982). In humans, elevations of enkephalins and plasma beta endorphins have been reported following a large variety of stressors (e.g., Janal *et al.*, 1984; Colt *et al.*, 1981; Cohen *et al.*, 1982). In testing the generalizability of the phenomenon of SIA to people, we found that Vietnam veterans with PTSD, when viewing a movie depicting Vietnam combat demon-

strate analgesia equivalent to a 8 mg injection of morphine, which can be blocked with naloxone (Pitman *et al.*, submitted for publication; van der Kolk *et al.*, unpublished data). Thus reexposure to traumatic situations seems to evoke an endogenous opioid response analogous to that seen in animals in response to even mild shock subsequent to inescapable shock. This means that reexposure to stress in some people can have the same effect as the temporary application of exogenous opioids, and may provide a similar relief from stress.

Self-harm, Dissociation, and Endogenous Opioids

If the analogy with other mammals is correct, early exposure to severe, prolonged, environmental stress causes increases in both autonomic and endogenous opioid response to subsequent stressors. This can produce not only a long-term enhanced autonomic stress response but also an increased endogenous opioid response, with both dependence and withdrawal phenomena akin to the application of exogenous opioids. The secretion of endogenous opioids in response to stress in previously traumatized people could explain the frequency with which childhood trauma is associated with subsequent self-destructive behavior. Green (1978) found that 41% of a sample of abused children engaged in such self-destructive acts as headbanging, biting, burning, and cutting. Studies and case reports of adult self-mutilators consistently report physical and sexual abuse in the childhoods of these patients (e.g., Bach-y-Rita, 1974; Simpson and Porter, 1981; Pattison and Kahan, 1983). Self-mutilation is reported as a response to abandonment in these people, and is accompanied by both analgesia and an altered state of consciousness. Self-harm provides relief and return to normalcy. A recent study found raised metenkephalins in patients who habitually mutilate themselves (Coid *et al.*, 1983). Opioid receptor blockade has been found to decrease self-mutilation (e.g., Richardson and Zaleski, 1983). The relationship between endogenous opioids, dissociation, and self-mutilating behavior is a subject for further investigation.

ATTACHMENT AND MATURATION OF THE CENTRAL NERVOUS SYSTEM (CNS)

People are biological organisms that develop from the moment of birth in a social context. Much of the maturation of the central nervous system (CNS) occurs after parturition. In the past few decades research about primate development has demonstrated that ongoing neurobiological development is strongly influenced by the social matrix, particularly by the quality of the attachments to caregivers. Studies by Bowlby (1969, 1973, 1984), Spitz

(1945), Emde (1982), Reite *et al.* (1981), Cicchetti (1984), and many others have started to demonstrate the profound psychobiological effects of disruption of the human mother-infant attachment bond. In non-human primates, the work of Harlow and his heirs has shown in vastly greater detail the lasting neurobiological changes that follow disruptions of attachment during infancy. Starting with Bowlby, and supported by numerous subsequent investigations, the need for a "safe base" from which a child can venture out to explore the world and to which it can safely return has been amply demonstrated. Continuity of attachment between mother and child is maintained with a complex interactional system: In all mammalian species, abandoned pups emit a distress cry, which stimulates complementary behaviors in mothers (and other caregivers) that promote secure development (MacLean, 1985). In a variety of animal species, including man, infants tend to seek increased attachment in the face of any external danger. This occurs even when the threat emanates from the attachment object itself: thus, attachment may increase even when the caregiver no longer provides effective protection and nurturance (Rajecki *et al.*, 1978). Lack of parental response to separation results in a biphasic protest/despair response (Bowlby, 1969) which most likely is correlated with hyperactivity or underactivity of a variety of neurotransmitter systems. The protest signals are aimed at bringing the primary attachment figure back, and they cease upon the adult's return. Failure to return results in a despair response.

There is a remarkable parallel between the protest and the despair phases of an infant's response to parental separation and the hyperarousal and numbing states found in PTSD. Field (1985) has suggested that normal play and exploratory activity in children requires the presence of a familiar attachment figure who modulates their physiological arousal by providing a balance between soothing and stimulation: In the absence of the mother, an infant experiences extremes of under- and overarousal that are physiologically disorganizing. Unresponsive or abusive caregivers may cause hyperarousal that has long-term effects on the child's ability to modulate strong emotions. After repeated separations these changes persist over time even though there are no readily observable behavioral concomitants of this chronic increase in physiological arousal (Coe, *et al.*, 1983). Field notes: "on a continuum from low to high physiological arousal there is an optimal level for every organism. The shape of an individual's optimal stimulation curve may depend on the level of stimulation received during early experience." Field has proposed that the mother serves as a mediator of soothing and arousal in the child, and that failure in the developent of the "the essential synchronicity between mother and child" leads to physiological disorganization which leads to extremes of under- and overarousal (Field, 1985). This physiological instability, in turn, can interfere with the capacity to assimilate and accommodate new information, and sets the stage for an increased vulnerability to develop PTSD in response to subsequent stressors.

In recent years, evidence has accumulated about the biological substate of attachment and separation behaviors (for review, see Field and Reite, 1985; van der Kolk, 1987). Studies on a variety of species suggest that the distress call is mediated by endogenous opioids, as is the maternal response to the distress call: Both are abolished by low-dose morphine, and no other known psychotropic agent (Newman *et al.*, 1982; Panksepp *et al.*, 1985). Low levels of endogenous opioids are thought to be released upon ventral to ventral contact. Opiate receptor blockade in monkeys enhances the need for social attachment (Fabre-Nys *et al.*, 1982). Social isolation during critical stages of development directly affects the number or sensitivity of brain opiate receptors in animals (Panksepp, 1980).

Maternal separations have been shown to affect other neurotransmitter systems as well. In monkeys, even brief separations cause highly elevated plasma cortisol levels; repeated maternal separations lead to chronically elevated plasma cortisol (Coe *et al.*, 1978). Elevated cortisol leads to an increase in tryptophan hydroxylase, which, in turn, results in a decrease in serotonin, the neurotransmitter most implicated in the modulation of affect and aggression (Brown *et al.*, 1982). Kraemer and McKinney have demonstrated that monkeys with early separation experiences have much higher CSF NE responses to subsequent social stressors, and to amphetamines (Kraemer *et al.*, 1984). They become much more aggressive when exposed to strangers, and are more susceptible to abuse alcohol than monkeys with safe early bonding experiences (Kraemer, 1985).

Like animals who have been exposed to inescapable shock, infants who are separated from their mothers show changes in hypothalamic serotonin, adrenal gland catecholamine synthesizing enzymes, plasma cortisol (Konner, 1982; Tennes, 1982), and immunoresistance (Laudenslager *et al.*, 1985). These changes are not minor, and they persist over time. This suggests the psychological effects of early separation rest on long-term neurobiological alterations. Since any given behavior is undoubtedly influenced by a large variety of interacting neurochemical systems, any attempt to establish connections between a particular neurotransmitter and a specific type of behavior is bound to result in an oversimplification.

CLINICAL PRESENTATION: A FUNCTION OF DEVELOPMENTAL LEVEL AND SEVERITY OF THE TRAUMA

Trauma and Psychiatric Illness

In recent years there have been consistent reports that at least 50% of psychiatric patients have a childhood history of severe chronic physical and/or

sexual abuse (Carmen *et al.*, 1983; Herman, 1985; Beck and van der Kolk, 1987; Bryer *et el.*, 1987). Despite the fact that so many current psychological symptoms and maladaptive behaviors obscure the significance of the childhood traumatic stressors, most of these patients show syndromes which are reminiscent of the posttraumatic sequelae in adults, such as physiological hyperreactivity, a subjective sense of loss of control, chronic passivity alternating with uncontrolled violence againt the self or others, and sleep disturbances. However, these patients do not meet DSM III criteria for PTSD, principally because the relevance of the original traumatic stressor has been lost in the current clinical picture, both to the patients and to the clinicians. Data from these studies indicate that the hallmark of psychiatric patients with chronic childhood trauma is the multiplicity of clinical presentations which they exhibit over time, the variety of diagnoses that are given, and the number of different medications received.

In a recent study (Herman *et al.*, submitted for publication) we found that 21 of 23 patients with a diagnosis of borderline personality disorder reported a history of severe physical and/or sexual abuse, compared with only one in the control group of bipolars and none in the schizoid controls. The severity of the borderline characteristics of affective instability, violence against self and others and drug abuse was directly proportional to the severity of the childhood trauma and the disorganization of early attachment bonds (van der Kolk *et al.*, 1987). Also striking was the degree of improvement of most of our subjects since they first entered this study, between 3 and 6 years prior to our traumatic antecedent interviews. All subjects ascribed this improvement to feeling secure with their therapists. Thus, while we consistently find histories of severe trauma in many psychiatric patients, strictly speaking, they do not have PTSD: Their traumas have become integrated into the totality of their personality organization. While they continue to experience life the way Kardiner described adult trauma victims: either on constant alert for trauma, or as having given up all hope of activity being able to influence the outcome of their lives, the historical referents for this stance usually have been lost.

The evidence is clear that prior trauma predisposes adults to develop full-blown PTSD in response to later life stresses (Burgess and Holstrom, 1979; Hendin *et al.*, 1983; Helzer, 1987). Although no studies have as yet been done to precisely map out the differential effect of trauma on people at varying stages of development, clinical evidence points in the direction that as people mature, stressful life experiences are either more likely to be effectively processed and overcome, or to be walled off and to affect only isolated aspects of functioning. Depending on the age of the victim, predisposing personality factors and the nature and severity of the trauma, fragments of the trauma can be reexperienced in a large variety of ways: as somatic sensations, as visual recollections in the form of flashbacks and nightmares,

as behavioral reenactments of aspects of the trauma, as hypermnesias, or even as experiences of fully formed distinctly different personalities, as in Multiple Personality Disorder (Brett and Ostrow, 1985; Putnam *et al.*, 1986; van der Kolk, 1987). Thus, there seems to exist a spectrum of trauma related disorders, ranging from phobias and panic reactions related to distinct (early?) traumatic events to borderline and multiple personality disorder, where extreme and chronic violation prior to adolescence is integrated into the totality of a person's personalty organization. PTSD, as defined by the DSM IIIR is only one possible outcome of traumatization. Recent studies of burned children (Stoddard, 1985) and of adult survivors of natural and man-made disasters (Shore *et al.*, 1986; Holen, 1987) have shown that, over time, recurrent recollections of the trauma abate, but that chronic anxiety, without conscious reference to the trauma, persists in a large percentage of individuals.

Information Processing and Memory Storage

Both a person's developmental level and the degree of physiological arousal affect the way in which information is processed and memories are stored. Developmental psychologists have identified three modes of information processing: enactive, iconic, and symbolic/linguistic (White and Pillemer, 1979; Neisser, 1967). These three modes of representation closely parallel Piaget's notions of sensorimotor, preoperational, and operational thinking, which bear relationships with the development of the central nervous system (Piaget, 1970). Over the course of development of the child, there is a shift from sensorimotor (motoric action), to perceptual representations (iconic), to symbolic and linguistic modes of organization of mental experience. During periods of stress, people tend to revert to earlier modes of representation. The esssence of the trauma experience is that it leaves people in a state of "unspeakable terror." The experience does not fit into existing conceptual schemata: it overhwelms. This precludes accommodation and assimilation of the experience; leaving the experience to be organized on a sensorimotor or iconic level—as horrific images, visceral sensations, or as fight/flight/freeze reactions. Under ordinary circumstances, memories for these experiences are often difficult to retrieve, but they can be reactivated by affective, auditory, or visual cues. This is analogous to the concept of state-dependent learning where information is acquired in an aroused, or intoxicated state and is not available under normal conditions, but returns when the altered state of consciousness is reintroduced (Eich, 1980).

Children are particularly vunerable to physiological disorganization in the face of stress, and they rely principally on their caregivers for modulation of arousal. The acquisition of cognitive schemata in which to frame current life experience plays a crucial role in the capacity to modulate physiological arousal in the face of threat. Terr (1979, 1983) has described

the degree to which kidnapped children were preoccupied with creating cognitive schemes in which to encompass their traumatic experience. Cognitive schemata serve as a buffer against being overwhelmed: e.g., and emergency medical technician is much less likely to react to physical trauma with a freeze or flight reaction than people whose cognitive frame leaves them unprepared for specific responses in the face of mutilation and death. Thus, the cognitive preparedness (development) of an individual interacts with the degree of physiological disorganization to determine the capacity for mental processing of potentially traumatizing experiences (Kihlstrom, 1984; van der Kolk, 1987). Traumatic experiences will be stored in memory, but the somatosensory elements may override linguistic representation, and be expressed as anxiety attacks and panic disorders. Our current understanding is that children are much more prone than adults to react to trauma on a somatosensory level (Pynoos and Eth, 1985), and often have little cognitive awareness of the origin of these reactions, which result in increased clinging and neophobia.

Recent research by Nadel and others (e.g., Jacobs and Nadel, 1985) on state-dependent learning sheds some light on the greater vulnerability of children to respond to trauma with anxiety reactions. They found that the hippocampus, which serves a mapping function for locating memories for experiences in space and time, does not fully mature till the third or fourth year of life. However, the maturation of the taxon system, which subserves memories related to the quality (feel and sound) of things occurs much earlier. Thus, in the first few years of life only the quality of events, but not their context can be remembered. During subsequent human development, the locale system remains vulnerable to disruptions. Stress disrupts the functioning of the hippocampally based locale system, and potentiates the taxon system, i.e., it leads to context-free fearful associations which are hard to locate in space and time (Squire, 1987). The hippocampus is rich in corticosterone receptors, which are selectively activated during stress (Pfaff, Silva, & Weiss, 1971; Sapolsky, Krey, & McEwen, 1984). Severe or prolonged stress, with its accompanying increase in corticosteroid levels can result in a suppression in hippocampal functioning and thus amnesia for traumatic experiences (Squire, 1987). Lacking localization in space and time, they are encoded in sensorimotor form and therefore cannot be easily translated into the symbolic language necessary for linguistic retrieval. Clinical experience shows that many traumatized adults are aware of fragments of the traumatization, but are unable to recall specifics and put them into autobiographical context.

Freud (1920) already had noted that when traumatic material gains only partial representation, it is accompanied by anxiety which can then trigger defensive maneuvers of suppression, avoidance, and flight. This anxiety can be viewed as a partial reliving of affective and somatosensory components of the traumatic memories, without the symbolic and linguistic representations necessary to place the trauma in its historical context. Overwhelmed by generalized somatic and affective reactions without context, traumatized

adults often regress to earlier stereotyped emotional and behavioral patterns, including infantile dependency, obsessive compulsive behavior, and difficulties in modulating the intensity of aggression (Krystal, 1968). Freud (1920) recognized that while conscious memories and feelings connected with trauma are forgotten, they may return as intrusive recollections, feeling states (such as overwhelming anxiety and panic unwarranted by current experience), as fugues, delusions, depersonalization states, and finally as behavioral reenactments. He proposed that origins of the repetition compulsion were founded in repression of the trauma and, like Lindemann (1942), he did not think that amnestic dissociation ever remained adaptive: His premise was that when a person represses important events "he is obliged to repeat the repressed material as a contemporary exprience instead of . . . remembering it as something belonging to the past."

EFFECTS ON INTERPERSONAL FUNCTIONING

The increased physiological arousal of traumatized individuals decreases their capacity to adequately assess the nature of current challenges, and interferes with resolution and integration of the trauma (Strian and Klicpera, 1978). It interferes with their ability to make calm and rational assessments of stressful situations and as a result, many traumatized people go immediately from stimulus to response, relying on action, rather than thought to meet new challenges. These responses to traumatization, including dissociative phenomena, sensation seeking, emotional constriction, and drug and alcohol abuse all cause difficulty in recognizing the traumatic etiologies and reenactments in the symptomatology of many psychiatric patients. During periods of emotional constriction, when amnesia for the traumatic event, overinvolvement in work, and emotional distance may be the only symptoms, it is even more difficult to make the correct diagnosis.

The biochemical alterations secondary to trauma also may play a role in the other psychological defects which have been described to accompany trauma: (1) Depression, learned helplessness and catatenoid reactions (Horowitz, 1976; Krystal, 1984); (2) poor-affect tolerance, loss of ability to articulate specific and differentiated emotions, and a tendency to experience emotions as physical states (Krystal, 1984); (3) fixation on (Kardiner, 1941) and addiction to the trauma (van der Kolk *et al.*, 1985).

Depression and Learned Helplessness

A disturbance in the hormones necessary to prepare for stress, such as chronic noradrenergic hypersensitivity, chronic low serotonin, and high cortisol may contribute to the decreased capacity of many trauma victims

to feel actively in control of their lives. The loss of feeling that they can influence the course of their lives leads many trauma victims to position themselves at either extreme of the dependence-independence spectrum, i.e., they lose their capacity to modulate intimacy and dependency (e.g., Lindy, 1987). In practice, this means that many trauma victims either become intensely dependent on their caregivers, which is accompanied by a loss of personal initiative, or take a counterdependent stance, with lack of involvement with others, often accompanied by excessive involvement in work. Traumatized people generally have difficulty in modulating intimacy: They develop a disorder of hope. They often seem to have difficulties in knowing what are appropriate, or inappropriate demands. They may blame themselves for obvious physical or emotional abuse by a partner, but are enraged about minor disappointments coming from others. Unable to appropriately assess their own and others' contributions to interpersonal tension, they often continue to see many social transactions as further victimization. Lack of emotional involvement in actual relationships diminishes the meaning of life since the trauma, and thus further perpetuates the central role the trauma plays in their lives (van der Kolk and Ducey, 1984).

Poor-affect Tolerance

Traumatized people generally continue to have a poor tolerance for arousal. They have a tendency to respond to stress in an all-or-nothing fashion: they are prone to react either with unmodulated anxiety, which is often accompanied by motoric discharge, or with social and emotional withdrawal. The decreased capacity to modulate physiological arousal combined with the reduced ability to utilize symbols and fantasy to cope with stress leave traumatized individuals vulnerable to experience subsequent stresses primarily as somatic states, rather than as discreet historical events which require specific solutions (Krystal, 1984). The tendency to experience stress as a somatic reaction may take the form of psychosomatic symptoms, panic attacks, rage reactions, or behavioral reenactments. Lacking the proper historical references, i.e., a conscious understanding about the reason for the intensity of these reactions which are out of proportion to the severity of the current stressors, they lack verbal or symbolic control over their reactions, and often need psychopharmacological agents or biofeedback to help modulate the intensity of these somatic responses.

Fixation on (Addiction to) the Trauma

A certain proportion of traumatized patients remain centrally preoccupied with their trauma, at the expense of their life experiences (van der Kolk)

et al., 1985; Shoularsh, 1987). In this article we have discussed our preliminary findings about how the endogenous opioid system may be involved in this compulsive reliving of the trauma. Reliving may take the socially and psychologically useful form of sublimated preoccupation by being of assistance to other victims, or of "bearing witness." However, other trauma victims continue to recreate the trauma in some form for themselves, or for others. War veterans may enlist as mercenaries, incest victims may become prostitutes, victims of child physical abuse may provoke subsequent abuse in foster families, victims of child abuse may grow up to become self-mutilators (Pattison and Kahan, 1983). Still others recreate the trauma by identifying with the aggressor, and perpetrating the same acts on others that were once exercised upon them (Burgess *et al.*, 1987). The clinical impression is that what these people have in common is a vague sense of apprehension, emptiness, boredom, and anxiety when not involved in activities reminiscent of the trauma. There is little evidence that reenacting the trauma will eventually lead to mastery and resolution. In fact, psychotherapy may at times reinforce preoccupation with and fixation on the trauma. Much remains to be learned about the treatment of people who compulsively repeat the trauma on themselves or on others. This may be the area in which understanding the human response to traumatization eventually may yield the greatest dividends.

TREATMENT IMPLICATIONS

Unlike the panic attacks and chronic anxiety which have their origin in traumatic childhood events, acute post-traumatic anxiety in adults is quite amenable to verbal therapies. In fact, verbalizing the contextual elements of the trauma is the essence of the treatment of acute post-traumatic reactions. Pharmacological support not only can be immensely helpful to decrease the degree of physiological hyperarousal, but also can facilitate the capacity to retrieve memories of the traumatic events which can be understood as historical, rather than as contemporary experiences. Hypnosis may be a helpful adjunct in retrieving memories (Spiegel, 1981). Once the traumatic experiences have been located in time and place, a person can start making distinctions betwen current life stresses and past trauma, and decrease the impact of the trauma on present experience. Traumatized patients are frequently very difficult to engage in psychotherapy. This probably is related both with a fear of attachment which reawakens the risk of abandonment, and with reluctance to remember the trauma itself. After intense efforts to ward off reliving the trauma, therapists cannot expect that the resistances to remember will suddenly melt away under their empathic efforts. The trau-

ma can only be worked through when a secure bond is established with another person; this then can be utilized to hold the psyche together when the threat of physical disintegration is reexperienced (van der Kolk, 1987). Both the etiology and the cure of trauma-related psychological disturbance depend fundamentally on the security of interpersonal attachments. Failures to approach trauma-related material very gradually lead to intensification of the affects and physiological states related to the trauma, leading to increased somatic, visual, or behavioral reexperiences.

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