Posttraumatic stress disorder and the nature of trauma

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The role of psychological trauma (eg, rape, physical assaults, torture, motor vehicle accidents) as an etiological factor in mental disorders, anticipated as early as the 19th century by Janet, Freud, and Breuer, and more specifically during World War I and II by Kardiner, was "rediscovered" some 20 years ago in the wake of the psychological traumas inflicted by the Vietnam war and the discussion "in the open" of sexual abuse and rape by the women's liberation movement. 1980 marked a major turning point, with the incorporation of the diagnostic construct of posttraumatic stress disorder (PTSD) into the 3rd edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III) *and the definition of its main diagnostic criteria (reexperiencing of the traumatic event, avoidance of stimuli associated with the trauma, and symptoms of increased arousal). Initially described as resulting from a onetime severe traumatic incident, PTSD has now been shown to be triggered by chronic multiple traumas as well. This "state-of-the-art" article discusses past and current understanding of the disorder, with particular emphasis on the recent explosive developments in neuroimaging and other fields of the neurosciences that have highlighted the complex interrelationships between the psychological, psychiatric, biological, and neuroanatomical components of the disorder, and opened up entirely new therapeutic perspectives on how to help the victims of trauma overcome their past.*

The human response to psychological trauma is one of the most important public health problems in the world. Traumatic events such as family and social violence, rapes and assaults, disasters, wars, accidents and predatory violence confront people with such horror and threat that it may temporarily or permanently alter their capacity to cope, their biological threat perception, and their concepts of themselves. Traumatized individuals frequently develop posttraumatic stress disorder (PTSD), a disorder in which the memory of the traumatic event comes to dominate the victims' consciousness, depleting their lives of meaning and pleasure.[1] Trauma does not only affect psychological functioning: for example, a study of almost 10 000 patients in a medical setting[2] reported that persons with histories of severe child maltreatment showed a 4 to 12 times greater risk for developing alcoholism, depression, drug abuse, and suicide attempts, a 2 to 4 times greater risk for smoking, ≥50 sex partners, and sexually transmitted disease, a 1.4 to 1.6 times greater risk for physical inactivity and obesity, and a 1.6 to 2.9 times greater risk for ischemic heart disease, cancer, chronic lung disease, skeletal fractures, hepatitis, stroke, diabetes, and liver disease.

Prevalence

Traumatic events are very common in most societies, though prevalence has been best studied in industrialized societies, particularly in the USA. Kessler et al[3]

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found that in the USA at least 15% of the population reported to have been molested, physically attacked, raped, or been involved in combat. Men are physically assaulted more often than women (11.1% vs 10.3%), while women report higher rates of sexual assault (7.3% vs 1.3%). Half of all victims of violence in the US are under age 25; 29% of all forcible rapes occur before the age of eleven. Among US adolescents aged 12 to 17, 8% are estimated to have been victims of serious sexual assault; 17% victims of serious physical assault; and 40% have witnessed serious violence.[4] Twenty-two percent of rapes are perpetrated by strangers, whereas husbands and boyfriends are responsible for 19%, and other relatives account for 38%. Men sustain twice as many severe injuries than women do. For women and children, but not for men, trauma that results from violence within intimate relationships is a much more serious problem than traumatic events inflicted by strangers or accidents: in 1994, 62% of the almost 3 million attacks on women in the USA were by persons whom they knew, while 63% of the almost 4 million assaults on males were by strangers. Four out of five assaults on children are at the hands of their own parents. Over a third of the victims of domestic assault experienced serious injury, compared with a quarter of victims of stranger assault.[5] This illustrates that an assault by someone "known" is not less serious than assault by a stranger. Domestic abuse and child abuse are closely related: in homes where spousal abuse occurs, children are abused at a rate 1500% higher than the national average (National Victim Center, 1993).[6]

Selected abbreviations and acronyms

ASR	*abnormal startle response*
CRH	*corticotropin-releasing hormone*
DESNOS	*Disorders of Extreme Stress Not Otherwise Specified*
EMDR	*eye movement desensitization and reprocessing*
HPA	*hypothalamo-pituitary-adrenocortical (axis)*
mCPP	*meta-chlorophenylpiperazine*
MHPG	*3-methoxy-4-hydroxyphenylglycol*
NE	*norepinephrine*
PTSD	*posttraumatic stress disorder*
SSRI	*selective serotonin reuptake inhibitor*

Many people experience horrendous events without seeming to develop lasting effects of their traumatization. The most common effects of traumatization are included in the symptom picture described in the diagnosis of PTSD. However, depression, increased aggression against self and others, depersonalization, dissociation, compulsive behavioral repetition of traumatic scenarios, as well as a decline in family and occupational functioning, may occur without victims meeting full-blown criteria for PTSD. The most common causes of PTSD in men are combat and being a witness of death or severe injury, while sexual molestation and rape are the most common causes of PTSD in women. The capacity of these events to produce PTSD varied significantly, ranging from 56% in patients who regain consciousness in the middle of surgical procedures, to 48.4% of female rape victims, and 10.7% of men witnessing death or serious injury. Women have twice the risk of developing PTSD following a trauma than men do.

The symptomatology of the trauma response

When people are faced with life-threatening or other traumatic experiences, they primarily focus on survival and self-protection. They experience a mixture of numbness, withdrawal, confusion, shock, and speechless terror. Some victims try to cope by taking action, while others dissociate. Neither response absolutely prevents the subsequent development of PTSD, though problem-focused coping reduces the chance of developing PTSD, while dissociation during a traumatic event is an important predictor for the development of subsequent PTSD.[7] The longer the traumatic experience lasts, the more likely the victim is to react with dissociation.

When the traumatic event is the result of an attack by a family member on whom victims also depend for economic and other forms of security, as occurs in victims of intrafamilial abuse, victims are prone to respond to assaults with increased dependence and with a paralysis in their decisionmaking processes. Thus, some aspects of how people respond to trauma are quite predictable, but individual, situational, and social factors play a major role in the shaping the symptomatology.

Rape victims, as well as children and women abused by male partners, often develop long-term reactions that include fear, anxiety, fatigue, sleep and eating disturbances, intense startle reactions, and physical complaints.

They often continue to dissociate in the face of threat, suffer from profound feelings of helplessness and have difficulty planning effective action. This makes them vulnerable to develop "emotion-focused coping," a coping style in which the goal is to alter one's emotional state, rather than the circumstances that give rise to those emotional states. This emotion-focused coping accounts for the fact that people who develop PTSD are vulnerable to engage in alcohol and substance abuse. Between a quarter and half of all patients who seek substance abuse treatment suffer from a comorbid PTSD diagnosis. The relationship between substance abuse and PTSD is reciprocal: drug abuse leads to assault, and, reciprocally, assault leads to substance use.

Diagnostic issues

In 1980, the diagnosis of PTSD was constructed for inclusion in the *Diagnostic and Statistical Manual of Mental Disorders, 3rd edition (DSM-III)* in order to capture the psychopathology associated with traumatization in adults. Over the years, numerous studies have demonstrated that the diagnostic construct of PTSD is clinically relevant to individuals who have suffered single incident traumas such as rape, physical assaults, torture, and motor vehicle accidents. However, it has also become clear that in clinical settings most treatment-seeking patients have been exposed to a range of different traumatic events over their life span, and suffer from a variety of psychological problems, only some of which are covered in the definition of PTSD. These include affect dysregulation, aggression against self and others, amnesia and dissociation, somatization, depression, distrust, shame, and self-hatred. These other problems can either be conceptualized as comorbid conditions, or as part of a spectrum of trauma-related problems, that occur depending on the age at which the trauma occurred, the relationship to the agent responsible for the trauma, social support received, and the duration of the traumatic experience(s).

The diagnosis of PTSD is characterized by three major elements:

- ***The repeated reliving of memories of the traumatic experience.*** These tend to involve intense sensory and visual memories of the event, which are often accompanied by extreme physiological and psychological distress, and sometimes by a feeling of emotional numbing, during which there usually is no physiological arousal. These intrusive memories may occur spontaneously or can be triggered by a range of real and symbolic stimuli.
- ***Avoidance of reminders of the trauma,*** as well as of emotional numbing, detachment, and emotional blunting, often coexist with intrusive recollections. This is associated with an inability to experience joy and pleasure, and with a general withdrawal from engagement with life. Over time, these features may become the dominant symptoms of PTSD.
- ***A pattern of increased arousal*** is the third element of PTSD. This is expressed by hypervigilance, irritability, memory and concentration problems, sleep disturbances, and an exaggerated startle response. In the more chronic forms of the disorder, this pattern of hyperarousal and the avoidance may be the dominant clinical features. Hyperarousal causes traumatized people to become easily distressed by unexpected stimuli. Their tendency to be triggered into reliving traumatic memories illustrates how their perceptions become excessively focused on the involuntary seeking out of the similarities between the present and their traumatic past. As a consequence, many neutral experiences become reinterpreted as being associated with the traumatic past.

Secondary effects of developing PTSD

Once people develop PTSD, the recurrent unbidden reliving of the trauma in visual images, emotional states, or nightmares produces a constant reexposure to the terror of the trauma. In contrast to the actual trauma, which had a beginning, middle, and end, the symptoms of PTSD take on a timeless character. The traumatic intrusions themselves are horrifying: they interfere with dealing with the past, while distracting from being able to attend to the present. This unpredictable exposure to unbidden memories of the trauma usually leads to a variety of (usually maladaptive) avoidance maneuvers, ranging from avoidance of people or actions that remind them of the trauma, to drug and alcohol abuse, to emotional withdrawal from friends or activities that used to be potential sources of solace. Problems with attention and concentration keep them from being engaged with their surroundings with zest and energy. Uncomplicated activities like reading, conversing, and watching television require extra effort. This loss of ability to focus, in

turn, often leads to problems with taking one thing at a time and gets in the way of organizing one's life to get it back on track.

Disorders of extreme stress (DESNOS)

The *DSM-IV* Field Trial[8] demonstrated that it was not the prevalence of PTSD symptoms themselves, but depression, outbursts of anger, self-destructive behaviors, and feelings of shame, self-blame, and distrust that distinguished a treatment-seeking sample from a non-treatment seeking community sample with PTSD. The majority of people who seek treatment for trauma-related problems have histories of multiple traumas. One recent treatment-seeking sample[9] suffered from a variety of other psychological problems, which in most cases were the chief presenting complaints, in addition to their PTSD symptoms: 77% suffered from behavioral impulsivity, affective lability, and aggression against self and others, 84% suffered from depersonalization and other dissociative symptoms, 75% were plagued by chronic feelings of shame, self-blame, and feeling permanently damaged, and 83% complained of being unable to negotiate satisfactory relationships with others. These problems contribute significantly to impairment and disability above and beyond the PTSD symptoms.[10-12] Focusing exclusively either on PTSD, or on the depression, dissociation and character pathology prevent adequate assessment and treatment of traumatized populations.

As part of the *DSM-IV* Field Trial, members of the PTSD task force delineated a syndrome of psychological problems that have been shown to be frequently associated with histories of prolonged and severe interpersonal abuse. They called this "Complex PTSD," or "Disorders of Extreme Stress Not Otherwise Specified (DESNOS)."[8,13] This delineated a complex of symptoms associated with early interpersonal trauma: (i) alterations in the regulation of affective impulses, including difficulty with modulation of anger and being self-destructive; (ii) alterations in attention and consciousness leading to amnesias and dissociative episodes and depersonalizations; (iii) alterations in self-perception, such as a chronic sense of guilt and responsibility, chronically feeling ashamed; (iv) alterations in relationship to others, such as not being able to trust, not being able to feel intimate with people; (v) somatization—the problem of feeling symptoms on a somatic level for which no medical explanations can be found; and (vi) alterations in systems of meaning. These are now listed in the DSM under "Associated Features of PTSD."

The *DSM-IV* Field Trial of PTSD found that DESNOS had a high construct validity.[14] The earlier the onset of the trauma, and the longer the duration, the more likely people were to suffer from a high degree of all the symptoms that make up the DESNOS diagnosis.[8,15-17] These studies showed that interpersonal trauma, especially childhood abuse, predicts a high risk for developing DESNOS. Patients with DESNOS are high utilizers of crisis psychiatric care[16] and are usually refractory to conventional PTSD treatment.[17] Recent studies[18] showed that these patients may react adversely to current standard PTSD treatments and that effective treatment needs to focus self-regulatory deficits rather than "processing the trauma."

PTSD has become a common diagnosis for people who become patients in psychiatric hospitals. An examination of the records of the 384 000 Medicaid recipients in Massachusetts in 1997/98[19] revealed that PTSD, together with depression, was the most common psychiatric diagnosis. However, patients with a PTSD diagnosis spent 10 times as much time in the hospital than patients with the diagnosis of depression only. It is inconceivable that the 22 800 Medicaid recipients in Massachusetts who were admitted to psychiatric hospitals and diagnosed as suffering from PTSD were admitted following a one-time traumatic incident, such as a rape or motor vehicle accident. Most likely, they suffered from a complex constellation of symptoms. However, since the long-term psychiatric impact of chronic, multiple traumas receives the same diagnosis (PTSD) as would the effects of a onetime incident, this diagnosis fails to capture how convoluted the psychiatric problems of these patients are, and how complex their treatment is.

Historical background

Awareness of the role of psychological trauma as a contributory factor in psychiatric disturbances has waxed and waned throughout the past century. The study of the traumatic origins of emotional distress started during the last decades of the 19th century. At the Hôpital de la Salpêtrière in Paris, Jean Martin Charcot (1887)[20] first proposed that the symptoms of what was then called "hysterical" patients had their origins in histories of trauma. In his first four books, Charcot's student Pierre Janet described 591 patients, 257 of whom had a traumatic origin

of their psychopathology.[21,22] Janet was the first to propose that during traumatic events people experience "vehement emotions," which interferes with the integration of the overwhelming experience. Instead, the traumatic memories (and the actions related to them) are split off (dissociated) from everyday consciousness and from voluntary control: they are "unable to make the recital which we call narrative memory, and yet they remain confronted by (the) difficult situation."[23] Janet described how the memories of these traumas tended to return not as stories of what had happened: they were reenacted in the form of intense emotional reactions, aggressive behavior, physical pain, and bodily states that could all be understood as the return of elements of the traumatic experience.

Janet first observed that traumatized patients seemed to react to reminders of the trauma with responses that had been relevant to the original threat, but that currently had no adaptive value. Upon exposure to reminders, the trauma was reactivated in the form of images, feelings, and physical sensations related to the trauma.[21] He proposed that when patients fail to integrate the traumatic experience into the totality of their personal awareness, they seem to develop problems assimilating new experiences as well. It is . . . as if their personality has definitely stopped at a certain point, and cannot enlarge any more by the addition or assimilation of new elements."[23] "All (traumatized) patients seem to have had the evolution of their lives checked; they are attached to an insurmountable obstacle."[24] Janet proposed that the efforts to keep the fragmented traumatic memories out of conscious awareness eroded the psychological energy of these patients. This, in turn, interfered with the capacity to engage in focused action and to learn from experience. Unless the dissociated elements of the trauma were integrated into personal consciousness, the patient was likely to experience a slow decline in personal and occupational functioning.[25]

As a young physician, during the 1880s, Sigmund Freud did two clinical rotations at the Salpêtrière in Paris. Upon his return to Vienna he attached himself to an older internist, Jospeh Breuer, with whom he started to carefully study the symptoms of "hysterical" patients, and the origins of their symptoms, which were often characterized by marked motoric and sensory abnormalities. They summarized their first set of findings in a paper entitled *On the Physical Mechanisms of Hysterical Phenomena.*[26] Because of the astuteness of their observations it is useful to quote part of their account:

The ... memory of the trauma ... acts like a foreign body which long after its entry must be regarded as an agent that is still at work. At first sight it seems extraordinary that events experienced so long ago should continue to operate so intensely—that their recollection should not be liable to the wearing away process to which, after all, we see all our memories succumb. The following considerations may perhaps make this a little more intelligible. The fading of a memory or the losing of its affect depends on various factors. *The most important of these is whether there has been an energetic reaction to the event that provokes an affect.* By "reaction" we understand the whole class of voluntary and involuntary reflexes . . . in which . . . the affects are discharged. If this reaction takes place to a sufficient amount a large part of the affect disappears as a result. . . . If a reaction is suppressed [the affect] stays attached to the memory. The injured person's reaction to the trauma only exercises a complete "cathartic" effect if it is an adequate reaction—as, for instance, revenge. . . . Abreaction, however, is not the only method of dealing with the situation that is open to a normal person who has experienced a psychical trauma. But language serves as a substitute for action: with its help, an affect can be "abreacted" almost as effectively. . . . If there is no such reaction, in either deeds or words, any recollection of the event retains its affective tone. . . . A memory of such a trauma, even if it has not been abreacted, enters the great complex of associations, it comes alongside other experiences, which may contradict it, and is subjected to rectification by other ideas. . . . In this way a normal person is able to bring about the disappearance of the accompanying affect through the process of association. . . . *It may therefore be said that the ideas which have become pathological have persisted with such freshness and affective strength because they have been denied the normal wearing-away processes by means of abreaction and reproduction in states of uninhibited association* (italicized in original). We have become convinced that the splitting of consciousness . . . under the form of "double conscience" is present to a rudimentary degree in every hysteria and that a tendency to dissociation, and with it, the emergence of abnormal states of consciousness, is the basic phenomenon of this neurosis . . . in this view we concur with Janet . . . we must, however, mention another remarkable fact . . . namely, that these memories, unlike the memories of the rest of their lives, are not at the patients' disposal. On the contrary, *these experiences are*

> *completely absent from the patient's memory when they are in a normal psychical state, or are only present in a highly summary form*…. (1893, pp 7-11).[26]

Over time, Freud came to disbelieve the reality of his patients' tales of trauma. In his *Autobiographical Study* (1925),[27] he wrote:

> I believed these stories and consequently supposed that I had discovered the roots of the subsequent neurosis.… If the reader feels inclined to shake his head at my credulity, I cannot altogether blame him. I was at last obliged to recognize that these scenes of seduction had never taken place, and that they were only fantasies which my patients had made up (p 34).[27]

However, like Janet before him, Freud kept being fascinated with the issue of patients' apparent compulsion to arrange their lives in such a way that they would repeat their trauma over and over again. Freud proposed that the compulsion to repeat was a function of repression: because the memory is repressed the patient "is obliged to repeat the repressed material as a contemporary experience, instead of . . . remembering it as something belonging to the past" (p 18).[28]

In *Beyond the Pleasure Principle* (1920),[28] Freud described how patients suffering from traumatic neuroses often experienced a lack of conscious preoccupation with the memories of their accident. He postulated that "perhaps they are more concerned with NOT thinking of it." Yet, it appeared that Freud also was concerned with not thinking about the horrible real-life experiences that can destroy people's capacity to function. He did so by focusing on his patients' intrapsychic reality: interest in personal meaning making crowded out interest in the external reality that had given rise to these meaning systems. Psychiatry, as a discipline, came to follow Freud in his explorations of how the normal human psyche functioned: real-life trauma was ignored in favor of fantasy.

Little attention was paid to the further exploration of "traumatic neuroses" until the outbreak of the second World War, when Abram Kardiner wrote up his experiences of treating World War I veterans in *The Traumatic Neuroses of War* (1941).[29] In this book, this psychoanalyst emphasized the psychobiological nature of traumatic stress. He noted that sufferers from "traumatic neuroses" develop an enduring vigilance for and sensitivity to environmental threat, and stated that ". . . the nucleus of the neurosis is a *physioneurosis*. This is present on the battlefield and during the entire process of organization; it outlives every intermediary accommodative device, and persists in the chronic forms. The traumatic syndrome is ever present and unchanged." He described extreme physiological arousal in these patients: they suffered from sensitivity to temperature, pain, and sudden tactile stimuli:

> These patients cannot stand being slapped on the back abruptly; they cannot tolerate a misstep or a stumble. From a physiologic point of view, there exists a lowering of the threshold of stimulation; and, from a psychological point of view a state of readiness for fright reactions (p 95). [29]

Central in Kardiner's thinking, as it had been for Janet and Freud, is that fact that:

> The subject acts as if the original traumatic situation were still in existence and engages in protective devices which failed on the original occasion. This means in effect that his conception of the outer world and his conception of himself have been permanently altered" (p 82). [29]

At the end of the second World War, Kardiner lamented that:

> . . . these conditions [traumatic neuroses] are not subject to continuous study . . . but only to periodic efforts which cannot be characterized as very diligent. Though not true in psychiatry generally, it is a deplorable fact that each investigator who undertakes to study [traumatic neuroses] considers it his sacred obligation to start from scratch and work at the problem as if no one has ever done anything with it before." [30]

This proved to be true for the subsequent 30 years until the issue of traumatic neuroses was rediscovered in the wake of the Vietnam war and the emergence of the women's movement. When the importance of trauma was rediscovered, starting around 1978, many of the early formulations that had long since been forgotten proved to be remarkably accurate. However, progress in understanding the function of attachment in shaping the individual and rapid developments in the neurosciences gave a new shape to these old insights.

The psychobiology of trauma

During the past two decades, important advances have been made in the understanding of the nature and treatment of PTSD. Probably the most important progress has been in the areas of the neurobiological underpinnings and treatment. Modern research has come to elucidate the degree to which PTSD is, indeed, a "physioneurosis," a mental disorder based on the persistence of biological emergency responses.

In order to understand how trauma affects psychobiological activity it is useful to briefly revisit some basic tenets of neurobiology. Paul McLean[31] defined the brain as a detecting, amplifying, and analyzing device for maintaining us in our internal and external environment. These functions range from the visceral regulation of oxygen intake and temperature balance to the categorization of incoming information necessary for making complex, long-term decisions affecting both individual and social systems. In the course of evolution, the human brain has developed three interdependent "subanalyzers," each with different anatomical and neurochemical substrates: (i) the brainstem and hypothalamus, which are primarily associated with the regulation of internal homeostasis; (ii) the limbic system, which is in charge of maintaining the balance between the internal world and external reality; and (iii) the neocortex, which is responsible for analyzing and interacting with the external world.

It is generally thought that the circuitry of the brainstem and hypothalamus is mostly innate and stable, that the limbic system contains both innate circuitry and circuitry modifiable by experience, and that the structure of the neocortex is most affected by environmental input.[32] If that is true, trauma would be expected to leave its most profound changes on neocortical functions, and least affect basic regulatory functions. However, while this may be true of the ordinary stress response, trauma, stress that overwhelms the organism, seems to affect people over a wide range of biological functioning, involving a large variety of brain structures and neurotransmitter systems.

The interrelation between regulatory functions

The brainstem, hypothalamus, limbic system, and neocortex in concert monitor relations with the outside world and assess what is new, dangerous, or gratifying. To accomplish this assessment, the brain needs to take in new sensory information, categorize its importance, and integrate it with previously stored knowledge. Most importantly, it needs to determine what is significant, and filter out irrelevant information. After the meaning of an incoming signal has been categorized, the brain (usually unconsciously) needs to "formulate" an appropriate plan of action, while attending to both short-term and long-term consequences. This evaluation then needs to lead to the initiation of an appropriate response, which needs to be terminated once the challenge is gone.[32,33] Moreover, in order to remain in a state of relative stability, people need to learn to engage in sustained activities without being distracted by irrelevant stimuli. The organism needs to learn from experience and be able to entertain a range of alternatives without becoming disorganized, or acting upon them. In order to do this, they need to learn to discriminate between relevant and irrelevant stimuli, and to only select what is appropriate for achieving one's goals. Much of evolution of the human brain has consisted in developing the capacity to form highly complex mental images and collaborative social relationships that allow complex thought in the context of social systems. In order for this to be successful, the organism needs to integrate its own immediate self-interest with a capacity to adhere to complex social rules.[34] People with PTSD usually have serious problems in carrying out a host of these functions. The degree of impairment is determined not only by the severity of their PTSD symptomatology, but also by the age at which the trauma occurred, the length of time that the traumatic event lasted, and the degree of social support that the individual received.

A century ago, the philosopher and psychologist William James (brother of the novelist Henry James)[35] noted that the power of one's intellect is determined by one's perceptual processing style. The ability to comprehend (grasp, hold together, take hold of—from the Latin cumprendere) depends on stimulus sampling and the formation of schematic representations of reality.[36] There seem to be qualitatively significant differences between the ways people with PTSD sample and categorize experience, and the ways in which nontraumatized people do so.[37,38] Failure to comprehend the experience (in other words, dissociation) plays a critical role in making a stressful experience traumatic.[39]

The apparent uniqueness of traumatic memories

A century of study of traumatic memories shows that: (i) they are primarily imprinted in sensory and emotional modes, though a semantic representation of the memory may coexist with sensory flashbacks[40]; (ii) these sensory experiences often remain stable over time and unaltered by other life experiences[1,41] (iii) they may return, triggered by reminders, at any time during a person's life

with a vividness as if the subject were having the experience all over again (*DSM IV*); and (iv) these sensory imprints tend to occur in a mental state in which victims may be unable to precisely articulate what they are feeling and thinking.[42,43]

While transformation of memories of day-to-day experiences is the norm, the flashbacks and other sensory reexperiences of PTSD seem not to be updated or attached to other experiences. Triggered by a reminder, the past can be relived with an immediate sensory and emotional intensity that makes victims feel as if the event were occurring all over again. Patients with PTSD seem to remain embedded in their trauma as a contemporary experience and often become "fixated on the trauma."[29] While most patients with PTSD construct a narrative of their trauma over time, it is characteristic of PTSD that sensory elements of the trauma itself continue to intrude as flashbacks and nightmares, altered states of consciousness in which the trauma is relived, unintegrated with an overall sense of self. Because traumatic memories are so fragmented, it seems reasonable to postulate that extreme emotional arousal leads to a failure of the central nervous system (CNS) to synthesize the sensations related to the trauma into an integrated whole.

The availability of neuroimaging studies of patients with PTSD has provided an opportunity to determine which brain structures are affected by traumatic experiences and, hence, how these structures are mobilized differently in response to traumatic reminders, compared with their response to neutral stimuli. This has facilitated a rapid increase in our understanding of the potential mechanisms of PTSD and promoted the exploration of new therapeutic techniques.

Psychophysiological effects of trauma

One of the principal contributions of trauma research to psychiatry has been the clarification that the development of a chronic trauma-based disorder is qualitatively different from a simple exaggeration of the normal stress response.[44] It also has become clear that PTSD is not an issue of simple conditioning: many people who do not suffer from PTSD, but who have been exposed to an extreme stressor, will again become distressed when they are once again confronted with the tragedy. Pitman[45] has pointed out that the critical issue in PTSD is that the stimuli that cause people to overreact may not be conditional enough: a variety of triggers not directly related to the traumatic experience may come to precipitate extreme reactions.

Abnormal psychophysiological reactions in PTSD occur on two very different levels: (i) in response to specific reminders of the trauma; and (ii) in response to intense, but neutral stimuli, such as loud noises, signifying a loss of stimulus discrimination.

Conditional responses to specific stimuli—kindling

PTSD sufferers experience heightened physiological arousal in response to sounds, images, and thoughts related to specific traumatic incidents. A large number of studies have confirmed that people with PTSD, but not controls who did not develop PTSD, respond to such reminders with significant increases in heart rate, skin conductance, and blood pressure.[46-48] The highly elevated autonomic responses to reminders of traumatic experiences that happened years, and sometimes decades, ago illustrate the intensity and timelessness with which these memories continue to affect current experience.[45] Post and his colleagues[49,50] have shown that life events play a critical role in the first episodes of major affective disorders, but become less pertinent in precipitating subsequent occurrences. This capacity of triggers with diminishing strength to produce the same response over time is called kindling.

Medications that decrease autonomic arousal, such as β-adrenergic blockers and benzodiazepines, tend to decrease traumatic intrusions, while drugs that stimulate autonomic arousal may precipitate visual images and affect states associated with prior traumatic experiences in people with PTSD, but not in controls. For example, in patients with PTSD, the injection of drugs such as lactate[51,52] and yohimbine[53] tends to precipitate panic attacks, flashbacks (exact reliving experiences) of earlier trauma, or both. In our own laboratory, approximately 20% of PTSD subjects responded with a flashback of a traumatic experience when they were presented with acoustic startle stimuli.

Hyperarousal to intense, but neutral stimuli—loss of stimulus discrimination

Excessive stimulation of the CNS at the time of the trauma may result in permanent neuronal changes that have a negative effect on learning, habituation, and stimulus discrimination. These neuronal changes do not

depend on actual exposure to reminders of the trauma for expression. The abnormal startle response (ASR) characteristic of PTSD[54] is one example of this phenomenon. Several studies have demonstrated abnormalities in habituation to the ASR in PTSD.[55,56] Interestingly, people who previously met criteria for PTSD, but no longer do so now, continue to show failure of habituation of the ASR (van der Kolk et al, unpublished data; Pitman et al, unpublished data).

The failure to habituate to acoustic startle suggests that traumatized people have difficulty evaluating sensory stimuli and mobilizing appropriate levels of physiological arousal.[57] Thus, the problems that people with PTSD have with properly integrating memories of the trauma and their getting mired in a continuous reliving of the past is mirrored physiologically in the misinterpretation of innocuous stimuli as potential threats. To compensate, they tend to shut down. However, the price for shutting down is decreased involvement in ordinary, everyday life.

The hormonal response in posttraumatic stress disorder

In a well-functioning organism, stress produces rapid and pronounced hormonal responses. However, chronic and persistent stress inhibits the effectiveness of the stress response and induces desensitization.[58] PTSD develops following exposure to events that overwhelm the individual's capacity to reestablish homeostasis. Instead of returning to baseline, there is a progressive kindling of the individual's stress response. Initially, only intense stress is accompanied by the release of endogenous, stress-responsive neurohormones, such as cortisol, epinephrine and norepinephrine (NE), vasopressin, oxytocin, and endogenous opioids. In PTSD, even minor reminders of the trauma may precipitate a full-blown neuroendocrine stress reaction: it permanently alters how an organism deals with its environment on a day-to-day basis, and it interferes with how it copes with subsequent acute stress.

While acute stress activates the hypothalamo-pituitary-adrenocortical (HPA) axis and increases glucocorticoid levels, organisms adapt to chronic stress by activating a negative feedback loop that results in: (i) decreased resting glucocorticoid levels in chronically stressed organisms[59]; (ii) decreased glucocorticoid secretion in response to subsequent stress[60,61]; and (iii) increased concentration of glucocorticoid receptors in the hippocampus.[62] Corticotropin-releasing hormone (CRH), produced by the hypothalamus, controls the secretion of adrenocorticotrophic hormone from the pituitary. It has substantial anxiogenic properties and has become the focus of intense interest in recent years.

Yehuda and associates (see review by Yehuda, 1997[63]) have comprehensively examined the HPA axis in PTSD, the neuroendocrine system controlling the stress hormone cortisol. Despite the fact that one would predict high cortisol as part of the stress response, the available evidence has consistently demonstrated low levels of serum cortisol. Careful examination of this issue has demonstrated that people with PTSD suffer from a disorder of the circadian cortisol modulation. Numerous studies have now demonstrated that the administration of low-dose dexamethasone results in supersuppression of cortisol release in patients with PTSD, but not in other disorders. Yehuda has suggested that increased concentration of glucocorticoid receptors could facilitate a stronger glucocorticoid negative feedback, resulting in a more sensitive HPA axis and a faster recovery from acute stress.[61]

In a study by Resnick et al,[64] the investigators collected blood samples from 20 acute rape victims and measured their cortisol response in the emergency room. Three months later, a prior trauma history was taken, and the subjects were evaluated for the presence of PTSD. Victims with a prior history of sexual abuse were significantly more likely to have developed PTSD 3 months following the rape than rape victims who did not develop PTSD. Cortisol levels shortly after the rape were correlated with histories of prior assaults: the mean initial cortisol level of individuals with a prior assault history was 15 µg/dL compared to 30 µg/dL in individuals without. These findings can be interpreted to mean either that prior exposure to traumatic events results in a blunted cortisol response to subsequent trauma, or in a quicker return of cortisol to baseline following stress.

Yehuda[63] has proposed that cortisol basically functions as an "antistress" hormone, shutting off the other biological reactions that were initiated by the stress response. Simultaneous activation of catecholamines and glucocorticoids stimulates active coping behaviors, while increased arousal in the presence of low glucocorticoid levels would provoke undifferentiated fight or flight reactions.

Catecholamines

Release of norepinephrine also plays an important role in the acute stress response. It leads to increased glucose metabolism and heightened awareness and concentration. Increased NE is correlated with heightened emotional arousal and mediated by right amygdala activation. Emotional arousal correlates with accuracy of recall and the consolidation of emotional memory.[65] Neuroendocrine studies of Vietnam veterans with PTSD have found good evidence for chronically increased sympathetic nervous system activity in PTSD.[66] This leads to compensatory downregulation of adrenergic receptors.[67] Southwick et al[68] used yohimbine injections (0.4 mg/kg) to study noradrenergic neuronal dysregulation in Vietnam veterans with PTSD. Subjects responded with substantially larger increases in plasma 3-methoxy-4-hydroxyphenylglycol (MHPG) than controls. Yohimbine precipitated significant increases in all PTSD symptoms, as well as panic attacks in 70% of subjects and flashbacks in 40% (for a more extensive discussions of catecholamines in PTSD, see Marburg[69]).

Other neurotransmitter abnormalities

While the role of serotonin in PTSD has received less systematic attention than the corticosteroids, the potential importance of serotonin in PTSD is illustrated by the fact that inescapably shocked animals are found to have decreased CNS serotonin levels[70] and that serotonin reuptake blockers are singularly effective pharmacological agents in the treatment of PTSD. Decreased serotonin in humans has repeatedly been correlated with impulsivity and aggression.[71-73] The literature tends to readily assume that these relationships are based on genetic traits. However, studies of impulsive, aggressive, and suicidal patients seem to find at least as robust an association between those behaviors and histories of childhood trauma (eg, refs 74-76). In order to test serotonergic contributions to trauma-related symptomatology, Southwick et al[77] administered meta-chlorophenylpiperazine (mCPP), a serotonin (5-HT) agonist, to 26 Vietnam veterans with PTSD. Thirty-one percent of the subjects experienced a panic attack, and 27% a flashback. These figures are comparable to the effects of the injection of yohimbine, which acts solely on the noradrenergic system. There was almost no overlap between the subjects who had these reactions to mCPP and those who did on yohimbine. This suggests that multiple neurotransmitters are involved in these complex PTSD symptoms.

Trauma and the central nervous system

The disintegration of experience

In a series of studies, we have demonstrated that traumatic memories initially have few narrative elements: when PTSD patients have their flashbacks, the trauma is relived as isolated sensory, emotional, and motoric imprints of the trauma, without a storyline. We have shown this in victims of childhood abuse,[40] assaults and accidents in adulthood,[78] and in patients who gained awareness during surgical procedures.[79] These studies support Janet's 1889 observations[21] and confirm the notion that what makes memories traumatic is a failure of the central nervous system to synthesize the sensations related to the traumatic memory into an integrated semantic memory. Sensory elements of the experience are registered separately and are often retrieved without the patient appreciating the context to which this sensation or emotion refers.

These observations lead to the notion that in PTSD the brain's natural ability to integrate experience breaks down. A variety of CNS structures have been implicated in these integrative processes: (i) the parietal lobes are thought to integrate information between different cortical association areas[80]; (ii) the hippocampus is thought to create a cognitive map that allows for the categorization of experience and its connection with other autobiographical information[81]; (iii) the corpus callosum allows for the transfer of information by both hemispheres,[82] integrating emotional and cognitive aspects of the experience; (iv) the cingulate gyrus is thought to play the role of both an amplifier and a filter that helps integrate the emotional and cognitive components of the mind[83]; and (v) the dorsolateral frontal cortex, which is where sensations and impulses are "held in mind" and compared with previous information to plan appropriate actions. The frontal lobes, in general, are thought to function as a "supervisory system" for the integration of experience.[84] Recent neuroimaging studies of patients with PTSD have suggested a role for most of these structures in the neurobiology of PTSD.

Neuroimaging studies in PTSD

As of 1999, there have been seven published studies utilizing neuroimaging of patients with PTSD.[85-91] Four studies have used magnetic resonance imaging (MRI) to measure hippocampal volume in individuals with PTSD, and three studies have used positron emission tomogra-

phy (PET)[85,88,91] to measure differential activation of the CNS in response to traumatic and nontraumatic scripts in patients with PTSD.

Hippocampal volume

Three different studies have shown that people with chronic PTSD have decreased hippocampal volumes, ranging from 8%[87,92] to 26%.[86] The fact that the only prospective study of acutely traumatized individuals, Shalev et al (ref 93 and personal communication, 1999) failed to find a correlation between hippocampal volume and PTSD severity suggests that this hippocampal shrinkage is a function of chronicity. Recent research suggests that the hippocampal changes may not be irreversible.[94-96] However, work of Sheline[97] strongly suggests some irreversible shrinkage of hippocampus in recurrent depression. In animals, decreased hippocampal functioning has been shown to cause behavioral disinhibition[98] and makes animals more likely to define incoming stimuli in the direction of emergency (fight/flight) responses. If the same is true for humans, this might contribute to the problems of PTSD patients with "taking in" and processing arousing information, and to learn from such experiences. The decreased size of the hippocampus might play a role in the ongoing dissociation and misinterpretation of information in the direction of threat. Their altered biology would make them vulnerable to react to newly arousing stimuli as a threat, and to react with aggression, or withdrawal, depending on their premorbid personality.[99]

Symptom provocation studies

Rauch, van der Kolk, and colleagues[85] conducted a PET scan study of patients with PTSD in which they were exposed to vivid, detailed narratives of their own traumatic experiences. During exposure to the script of their traumatic experiences these subjects demonstrated heightened activity only in the right hemisphere, specifically, in the areas that are most involved in emotional arousal—the amygdala, insula, and the medial temporal lobe. During exposure to their traumatic scripts there was a significant decrease in activation of the left inferior frontal area—Broca's area—which is thought to be responsible for translating personal experiences into communicable language. Shin et al's study,[91] utilizing a slightly different paradigm, essentially confirmed these findings in a different trauma population. In another study, Lanius et al (submitted) exposed 6 subjects with PTSD and 6 controls to a traumatic script and measured their responses with functional magnetic resonance imaging (fMRI) scans, and consistently found decreased activation of the thalamus and of the dorsolateral prefrontal cortex in PTSD patients during exposure to their trauma scripts.

These early neuroimaging studies of patients with PTSD present us with a range of surprising findings that force us to reevaluate our previous concepts of the pathophysiology of PTSD. Of the various findings, increased activation of the amygdala in response to traumatic scripts is the least surprising. After all, it has been well established that the amygdala is centrally involved in the interpretation of the emotional valence of the incoming information and that confrontation with feared stimuli activates the amygdala and related structures.[100] Exposure to traumatic scripts frequently provokes autonomic activation of patients with PTSD (eg, refs 48 and 101), and this is likely mediated by activation of the amygdala and related structures. It is well understood that the information evaluated by the amygdala is passed on to areas in the brainstem that control autonomic and neurohormonal response systems. By way of these connections, the amygdala transforms sensory stimuli into emotional and hormonal signals, thereby initiating and controlling emotional responses.

High levels of stimulation of the amygdala can also interfere with hippocampal functioning.[102,103] Thus, extreme emotional arousal may prevent the proper evaluation and categorization of experience by interfering with hippocampal functions. It is possible that, when this occurs, sensory imprints of experience are stored in memory, but because the hippocampus is prevented from fulfilling its integrative function, these various imprints are not combined into a unified whole.[104] The experience is laid down, and later retrieved, as isolated images, bodily sensations, smells, and sounds that feel alien and separate from other life experiences. Decreased hippocampal functioning is likely to interfere with the localization of incoming information in time and space and cause continued fragmentation of experience. The recent findings of decreased dorsolateral frontal cortex activation would further provide a neurobiological explanation why people with PTSD plunge into reexperiencing their trauma with limited consciousness that they are simply remembering elements of experiences belonging to the past. In our pilot study, using single photon emission computed tomogra-

phy (SPECT) as an outcome measure of eye movement desensitization and reprocessing (EMDR) treatment, subjects had increased activation of the dorsolateral prefrontal cortex following effective treatment.

Hemispheric lateralization

The finding of hemispheric lateralization in subjects exposed to their personalized trauma scripts indicates that there is differential hemispheric involvement in the processing of traumatic memories. This may have important implications for the understanding of the nature of PTSD. The right hemisphere, which developmentally comes "on-line" earlier than the left hemisphere, is involved in the expression and comprehension of global, nonverbal emotional communication (tone of voice, facial expression, visual/ spatial communication), and allows for a dynamic and holistic integration across sensory modalities.[105] This hemisphere is particularly integrated with the amygdala, which assigns emotional significance to incoming stimuli and helps regulate the autonomic and hormonal responses to that information. While it is exquisitely sensitive to emotional nuances, it has, at best, a rudimentary capacity to think or communicate analytically, to employ syntax, or to reason.[106,107]
In contrast, the left hemisphere, which mediates verbal communication and organizes problem-solving tasks into a well-ordered set of operations and processes information in a sequential fashion,[107] seems to be less active in PTSD. It is in the area of categorization and labeling of internal states that people with PTSD seem to have particular problems.[108,109] It is conceivable that failure of left hemisphere function during states of extreme arousal is responsible for the derealization and depersonalization reported in acute PTSD.[7,110]

New directions for treatment

For over a century, it has been understood that traumatic experiences can leave indelible emotional memories. Contemporary studies of how the amygdala is activated by extreme experiences dovetail with the laboratory observation that "emotional memory may be forever."[111] The accumulated body of research suggests that patients with PTSD suffer from impaired cortical control over subcortical areas responsible for learning, habituation, and stimulus discrimination. The concept of indelible subcortical emotional responses, held in check to varying degrees by cortical and hippocampal activity, has led to the speculation that delayed-onset PTSD may be the expression of subcortically mediated emotional responses that escape cortical, and possibly hippocampal, inhibitory control.[1,45,112]
The early neuroimaging studies of PTSD showed that, during exposure to a traumatic script, there was decreased Broca's area functioning and increased activation of the right hemisphere. This would imply that it is difficult for traumatized individuals to verbalize precisely what they are experiencing, particularly when they become emotionally aroused. They may experience physiological arousal and fragments of memories may be activated, but they often seem to be too hyperaroused or hypoaroused to be able to "process" and communicate what they are experiencing. A relative decrease in left hemispheric representation provides an explanation for why traumatic memories are experienced as timeless and ego-alien: the part of the brain necessary for generating sequences and for the cognitive analysis of experience is not functioning properly. Our research[85] can be interpreted as showing that during activation of a traumatic memory, the brain is "having" its experience. The person may feel, see, or hear the sensory elements of the traumatic experience, but he or she may be physiologically prevented from being able to translate this experience into communicable language. When they are having their traumatic recall, victims may suffer from speechless terror in which they may be literally "out of touch with their feelings." Physiologically, they may respond as if they were being traumatized again. Particularly when victims experience depersonalization and derealization, they cannot "own" what is happening, and thus cannot take steps to do anything about it.
In order to help traumatized individuals process their traumatic memories, it is critical that they gain enough distance from their sensory imprints and trauma-related emotions so that they can observe and analyze these sensations and emotions without becoming hyperaroused or engaging in avoidance maneuvers. The serotonin reuptake blockers seem to be able to accomplish exactly that. Studies in our laboratory have shown that selective serotonin reuptake inhibitors (SSRIs) can help PTSD patients gain emotional distance from traumatic stimuli and make sense of their traumatic intrusions.[113]
The apparently relative decrease in left hemisphere activation while reexperiencing the trauma suggests that it is important to help people with PTSD find a language in which they can come to understand and communicate

their experiences. It is possible that some of the newer body-oriented therapies, dialectical behavior therapy, or EMDR may yield benefits that traditional insight-oriented therapies lack.

Making meaning of the traumatic experience usually is not enough. Traumatized individuals need to have experiences that directly contradict the emotional helplessness and physical paralysis that accompany traumatic experiences. In many people with PTSD, such helplessness and paralysis become a habitual way of responding to stressful stimuli, further weakening their feelings of control over their destiny. The critical steps in treating PTSD can be summarized as follows (for more details see ref 114):

- ***Safety***. When people's own resources are inadequate to deal with threat, they need to rely on others to provide them with safety and care. After having been traumatized, it is critical that the victim reestablishes contact with his or her natural social support system. If this system is inadequate to ensure the safety of the patient, institutional resources need to be mobilized the help the patient find a place to recover.
- ***Anxiety management***. After the patient's safety has been assured, there may be a need for a variety of psychological interventions. Patients need to learn to *name* the problems they face, and learn to formulate appropriate solutions. Assault victims must learn to distinguish between the real-life threats, and the haunting, irrational fears that are part of the disorder PTSD. If anxiety dominates, victims need to be helped to strengthen their coping skills. Practical anxiety management skills training may include deep muscle relaxation, breathing control, role-playing, covert modeling, thought stopping, and guided self-dialogue.
- ***Emotional processing.*** In order to put the event(s) in perspective, the victim needs to reexperience the event without feeling helpless. Traditionally, following Freud's notion that words can substitute for action to resolve a trauma (1893),[115] this has been done by helping people to talk about their entire experience.[13,64,116] They are asked to articulate what they think happened, and what led up to it; their own contributions to what happened, their thoughts and fantasies during the event, what was the worst part of it, and their reactions to the event in detail, including how it has affected their perceptions of themselves and others. Such exposure therapy is thought to promote symptom reduction by allowing patients to realize that: (i) remembering the trauma is not equivalent to experiencing it again; (ii) that the experience had a beginning, middle, and end, and that the event now belongs to one's personal history.

In recent years, a variety of new techniques have been developed that have the potential of desensitizing patients with PTSD without requiring them to fully engage in a verbal reliving of the traumatic experience. Of these treatments, EMDR has been best studied.[117] Although traditional exposure therapy can be very helpful in overcoming traumatic intrusions, it needs to be applied with care. Some patients, on recalling their trauma, may become flooded with both the traumatic memories and memories of previously forgotten traumas. Increased activation of traumatic memories may be associated with increased shame, guilt, aggression, and increase in alcohol and drug use.

Conclusions

The rediscovery of trauma as an etiological factor in mental disorders is only about 20 years old. During this time, there has been an explosion of knowledge about how experience shapes the central nervous system, and the formation of the self. Developments in the neurosciences have started to make significant contributions to our understanding of how the brain is shaped by experience, and how life itself continues to transform the ways biology is organized. The study of trauma has probably been the single most fertile area within the disciplines of psychiatry and psychology in helping to develop a deeper understanding of the interrelationships between emotional, cognitive, social, and biological forces that shape human development. Starting with PTSD in adults, but expanding into early attachment and coping with overwhelming experiences in childhood, our field has discovered how certain experiences can "set" psychological expectations and biological selectivity. Research in these areas has opened up entirely new insights in how extreme experiences throughout the life cycle can have profound effects on memory, affect regulation, biological stress modulation, and interpersonal relatedness. These findings, in the context of the development of a range of new therapy approaches, are beginning to open up entirely new perspectives on how traumatized individuals can be helped to overcome their past. ❑

El trastorno de estrés postraumático y las características del trauma

El papel del trauma psíquico (ej. violación, asalto, tortura, accidentes de vehículos motorizados) como un factor etiológico en los trastornos mentales fue anticipado por Charcot, Freud y Breuer en el siglo XIX, y luego más específicamente durante la I Guerra Mundial por Kardiner. Hace 20 años este papel etiológico fue "redescubierto" en el contexto de los traumas psicológicos producidos por la guerra de Vietnam y también a raíz de la discusión pública que ha realizado el movimiento de liberación de la mujer sobre el abuso sexual y las violaciones. El año 1980 marcó un hito importante ya que se incorporó el constructo diagnóstico del trastorno de estrés postraumático (TEPT) en la tercera edición del Manual Diagnóstico y Estadístico de los Trastornos Mentales (DSM-III) *y se definieron sus principales criterios diagnósticos (re-experimentar el acontecimiento traumático, evitación de estímulos asociados con el trauma y síntomas de hiperalerta). Inicialmente se describió que el TEPT se producía por un acontecimiento traumático aislado; sin embargo, actualmente se ha observado que el TEPT puede ser gatillado también por múltiples traumas crónicos. Este artículo que constituye una puesta al día sobre el TEPT discute las comprensiones pasadas y las actuales acerca de este trastorno, con especial énfasis en el explosivo desarrollo reciente de las técnicas de neuroimágenes y otros campos de las neurociencias. Estos estudios han iluminado las complejas interrrelaciones entre los componentes psicológicos, psiquiátricos, biológicos y neuroanatómicos de este cuadro, y han abierto perspectivas totalmente novedosas acerca de la formo de ayudar a las víctimas de traumas para superar el pasado.*

Etat de stress post-traumatique et nature du traumatisme

Le rôle des traumatismes psychologiques (tel que viols, agressions physiques, tortures, accidents de la circulation) en tant que facteurs étiologiques dans l'apparition de certains troubles mentaux, pressenti dès le 19e siècle par Charcot, Freud et Breuer, et de façon plus spécifique au cours des première et deuxième guerres mondiales par Kardiner, fut "redécouvert", il y a 20 ans, à la suite des traumatismes psychologiques infligés par la guerre du Vietnam et de la discussion "sur la place publique" par le mouvement de libération des femmes des abus sexuels et des viols. L'année 1980 a représenté un tournant important avec l'intégration de la catégorie diagnostique de l'état de stress post-traumatique (ESPT) dans la troisième édition du Manuel Diagnostique et Statistique des Troubles Mentaux (DSM-III) *et la définition de ses critères diagnostiques principaux (événement traumatique constamment revécu, évitement des stimulus associés au traumatisme et présence de symptômes persistants traduisant une activation neurovégétative). Initialement décrit comme survenant à la suite d'un incident traumatique sévère unique, il a été démontré que l'ESPT pouvait également être provoqué par des traumatismes chroniques multiples. Cet article de synthèse passe en revue les connaissances passées et actuelles sur cette pathologie, en insistant tout particulièrement sur les développements récents et accélérés en neuro-imagerie et dans les autres domaines des neurosciences. Ces développements ont permis de souligner les interactions complexes existant entre les composantes psychologiques, psychiatriques, biologiques et neuroanatomiques de cette pathologie, et ouvert des perspectives thérapeutiques totalement nouvelles sur la façon d'aider les victimes de traumatismes à surmonter leur passé.*

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