

Session 4

Trauma and memory

BESSEL A. VAN DER KOLK MD

Boston University School of Medicine, Boston MA, USA

Abstract

The study of traumatic memories challenges several basic notions about the nature of memory: (i) that memory always is a constructive process; (ii) that memory is primarily declarative (i.e. that people can articulate what they know in words and symbol) (iii) that memory is present in consciousness in a continuous and uninterrupted fashion; and (iv) that memory always disintegrates in accuracy over time. A century of study of traumatic memories shows that (i) semantic representations may coexist with sensory imprints; (ii) unlike trauma narratives, these sensory experiences often remain stable over time, unaltered by other life experiences; (iii) they may return, triggered by reminders, with a vividness as if the experience were happening all over again; and (iv) these flashbacks may occur in a mental state in which victims are unable to precisely articulate what they are feeling and thinking. The present paper reviews the literature on traumatic memories and discusses the recent neuroimaging studies which seem to clarify the neurobiological underpinnings of the differences between ordinary and traumatic memories.

Key words

dissociation, neuroanatomical model, posttraumatic stress disorder, traumatic memory.

INTRODUCTION

“Traumatic memory plays an important role in certain neuroses and psychoses. While some doctors never trouble their heads about traumatic memories, and are not even aware of the fact that they exist, and while others fancy them everywhere, there is room for people to take a middle course, and to detect the existence of traumatic memories in specific cases”.¹

The nature and reliability of traumatic memories has been controversial in psychiatry for over a century. Traumatic memories are difficult to study because the profoundly upsetting emotional experiences that give rise to posttraumatic stress disorder (PTSD) cannot be approximated in a laboratory setting; even viewing a movie depicting actual executions fails to precipitate posttraumatic symptoms in normal college students (RK Pitman, pers. comm., 1994). If trauma is defined as an inescapably stressful event that overwhelms people's existing coping mechanisms, it is questionable whether findings of memory distortions in normal subjects exposed to videotaped stresses in the laboratory can serve as a meaningful guide to understanding traumatic memories. Clearly there is

little similarity between witnessing a simulated car accident on a TV screen, and being the responsible driver in a car crash in which one's own children are killed. While response to stress evokes homeostatic mechanisms that lead to self-conservation and resource re-allocation,² PTSD involves a unique combination of hyperarousal, learned conditioning and shattered meaning propositions. Shalev has proposed that this complexity is best understood as the co-occurrence of several interlocking pathogenic processes including (i) an alteration of neurobiological processes affecting stimulus discrimination (expressed as increased arousal and decreased attention), (ii) the acquisition of conditioned fear responses to trauma-related stimuli, and (iii) altered cognitive schemata and social apprehension.

Without the option of simulating trauma in the laboratory, there are only limited options for the exploration of traumatic memories: (i) collecting retrospective reports from traumatized individuals, (ii) post-hoc observations, or (iii) provoking of traumatic memories and flashbacks in a laboratory setting. Surprisingly, since the early part of this century, there have been very few published systematic studies that explore the nature of traumatic memories based on patient reports. Provocation studies of traumatic memories have been done in psychophysiology laboratories,^{3,4} and in tests where patients with PTSD are given drugs that alter neurotransmitter function that seem to promote access to trauma-related memories.⁵

Correspondence address: Bessel A. van der Kolk, MD, 16 Braddock Park, Boston, MA 0211-5804, USA.

Parts of this study were published earlier (van der Kolk B. A. & Fisler R. Dissociation and the fragmentary nature of traumatic memories. *J. Traumatic Stress* 1995; 9, 505–525).

This review will explore the studies that have collected data on people's memories of highly stressful and traumatic experiences, and will examine the differences between these two types of experiences. We then will review the evidence implicating dissociation as the central pathogenic mechanism that gives rise to PTSD, which causes traumatic memories to be retrieved, at least initially, in the form of mental imprints of sensory and affective elements of the traumatic experience. We then will present the research findings of alteration in brain structure and function in PTSD that seem to play a role in these abnormal memory processes and conclude with a discussion about the nature of traumatic memories, as contrasted with memories of ordinary events, and the implications of these differences for treatment.

THE STABILITY AND ACCURACY OF MEMORIES OF STRESSFUL EVENTS

At least since 1889, when Pierre Janet first wrote about the relationship between trauma and memory,⁷ it has been widely accepted that what is now called declarative or explicit memory is an active and constructive process. What a person remembers depends on existing mental schemata; once an event or a particular bit of information is integrated into existing mental schemes it will no longer be available as a separate, immutable entity, but be distorted both by associated experiences and by the emotional state at the time of recall.^{7,8} As Schachtel defined it:

"Memory as a function of the living personality can be understood as a capacity for the organization and reconstruction of past experiences and impressions in the service of present needs, fears, and interests".⁹

However, accuracy of memory is affected by the emotional valence of an experience; studies of people's subjective reports of personally highly significant events generally find that their memories are unusually accurate and that they tend to remain stable over time.¹⁰⁻¹³ It appears that evolution favors the consolidation of personally relevant information. For example, Yuille and Cutshall interviewed 13 out of 22 witnesses to a murder 4-5 months after the event.¹⁴ All witnesses had provided information to the police within 2 days of the murder. These witnesses were found to have very accurate recall with little apparent decline over time. The authors concluded that emotional memories of such shocking events are 'detailed, accurate and persistent'.¹⁴ They suggested that witnessing real 'traumas' leads to 'quantitatively different memories than innocuous laboratory events'.

Researchers also have studied the accuracy of memories for culturally significant events, such as the

space shuttle Challenger and the murder of President Kennedy. Brown and Kulik first called memories for such events 'flashbulb memories'.¹⁵ While people report that these experiences are etched accurately in their minds, research has shown that even those memories are subject to some distortion and disintegration over time. For example, Neisser and Harsch found that people changed their recollections of the space shuttle Challenger disaster considerably after a number of years.¹⁶ However, these studies did not measure the personal significance that their subjects attached to this event. Clinical observations of people who suffer from PTSD suggest that there are significant differences between flashbulb memories and the posttraumatic perceptions characteristic of PTSD. As of early 1995, I could find no published accounts in the scientific literature of intrusive traumatic recollections of traumatic events in patients suffering from PTSD that had become distorted over time, naturally or by manipulation, either in an experimental or in a clinical setting.

THE COMPLEXITY OF MEMORY SYSTEMS

Contemporary memory research has demonstrated the existence of a great complexity of memory systems within each individual, with multiple components, most of which are outside of conscious awareness. Each one of these memory functions seems to operate with a relative degree of independence from the others. To summarize, declarative (also known as explicit) memory refers to conscious awareness of facts or events that have happened to the individual.¹⁷ This form of memory functioning is seriously affected by lesions of the frontal lobe and of the hippocampus, which also have been implicated in the neurobiology of PTSD.¹⁸ Non-declarative, implicit, or procedural memory refers to memories of skills and habits, emotional responses, reflexive actions, and classically conditioned responses. Each of these implicit memory systems is associated with particular areas in the central nervous system.¹⁹ Schacter has referred to the scientific descriptions of traumatic memories, like those by Pierre Janet, as examples of implicit memory.²⁰

THE APPARENT UNIQUENESS OF TRAUMATIC MEMORIES

The DSM definition of PTSD recognizes that trauma can lead to extremes of retention and forgetting; terrifying experiences may be remembered with extreme vividness, or totally resist integration. In many instances, traumatized individuals report a combination of both. While people seem to easily assimilate familiar and expectable experiences and while memories of

ordinary events disintegrate in clarity over time, some aspects of traumatic events appear to get fixed in the mind, unaltered by the passage of time or by the intervention of subsequent experience. For example, in our own studies on posttraumatic nightmares, subjects claimed that they saw the same traumatic scenes over and over again without modification over a 15-year period.²¹ For the past century, many students of trauma have noted that the imprints of traumatic experiences seem to be qualitatively different from memories of ordinary events. Starting with Janet, accounts of the memories of traumatized patients consistently mention that emotional and perceptual elements tend to be more prominent than declarative components.^{22–24} These recurrent observations of the apparent immutability of traumatic memories have given rise to the notion that traumatic memories may be encoded differently than memories for ordinary events, perhaps via alterations in attentional focusing, perhaps because of extreme emotional arousal interferes with hippocampal memory functions.^{7,11,18,25–30}

AMNESIA AND THE RETURN OF DISSOCIATED MEMORIES

Global memory impairment

While amnesia following adult trauma has been well-documented, the mechanisms for such memory impairment remains insufficiently understood. This issue is even more complicated when it concerns childhood trauma, since children have fewer mental capacities to construct a coherent narrative out of traumatic events. More research is needed to explore the consistent clinical observation that adults who were chronically traumatized as children suffer from generalized impairment of memories for both cultural and autobiographical events. It is likely that the combination of autobiographical memory gaps and continued reliance on dissociation makes it very hard for these patients to reconstruct a precise account of both their past and current reality. The combination of a lack of autobiographical memory, continued dissociation and of meaning schemes that include victimization, helplessness and betrayal, is likely to make these individuals vulnerable to suggestion and to the construction of explanations for their trauma-related affects that may bear little relationship to the actual realities of their lives.

Research on traumatic amnesia and delayed memory retrieval

While the vivid intrusions of traumatic images and sensations are the most dramatic expressions of PTSD,

the loss of recollections for traumatic experiences is well documented. The complexity of memory loss for trauma, and the psychological elaboration of the experience itself is illustrated by a very early case in the psychiatric literature; Charcot described the case of Lellog who was in a traffic accident with a horse-drawn wagon, after which his legs were paralyzed.³¹ Although he fell against the ground, and was unconscious, there was no neurological sign indicating a somatic cause of the paralysis. Instead it was discovered that as he fell, just before losing consciousness, he saw the wheels of the wagon approaching him, and strongly believed that he would be run over. This fantasy was dissociated and gave rise to his paralysis.³² Janet describes a case of a woman who re-enacted her traumatic experience without having any conscious recollection of what had happened to her.^{8,33} Early in my work with traumatized patients I described the case of a woman who had lost all memory of having been involved in the Coconut Grove fire, but who kept re-enacting her experience on its anniversary,³⁴ and of a Vietnam veteran who set up the police to re-create a shoot-out with him on the anniversary of his friend's death, for which he had no conscious recollection.³⁵

For over 100 years, there have been numerous descriptions of traumatized patients who suffered from amnesia for traumatic experiences. In the detailed case reports the role of dissociation in amnesia usually is easy to detect. For example, in his book, *The Traumatic Neuroses of War*, Kardiner describes a patient who had a complete amnesia for all the events preceding an accident.²³ The story was reconstructed by the patient from fragments related to him. During the month of unconsciousness he was said to have set fire to the hospital several times. Since then, the patient had been subject to lapses of unconsciousness lasting from 12 h to 11 days. He was later told that he was taken to a hospital and that he was fully awake during these lapses, was active, smoked, read and talked but was not his conscious self. He was also told that he did not appear to be of his 'right mind'. These major lapses of consciousness occurred at intervals of 5 years. Since that time he had only minor ones. They usually began with a feeling of paralysis in one extremity, either an arm or a leg. Sometimes it was only an attack of vertigo.²³ Fragments of dissociated memory often returned in dissociative fugue states; for example, triggered by a sensory stimulus, Kardiner described patients who became agitated, assaultive, using language that would have been appropriate to being caught up in the middle of a military assault. He described how many patients, while riding the subway in New York City, had flashbacks of being back in the trenches, especially upon entering a tunnel. In other

cases, people had panic attacks in response to stimuli reminiscent of the trauma, while failing to make a conscious connection between how they felt, and the prior traumatic experience.

Today it is generally accepted that the memory system is made up of networks of related information; activation of one aspect facilitates the retrieval of associated memories.^{36,37} Emotions and sensations seem to be the critical cues for the retrieval of information along these associative pathways. This means that the emotions attached to any particular experience play a major role in determining what cognitive schemes will be activated. In this regard, it is relevant that many people with trauma histories, such as rape, spouse battering and child abuse, seem to function relatively well as long as feelings related to traumatic memories are not stirred up. However, after exposure to specific emotional or sensory triggers, they may feel or act as if they were traumatized all over again. These triggers are not necessarily intrinsically frightening; any affect or sensation related to a particular traumatic experience may serve as a cue for the retrieval of associated sensations, including fear, longing, intimacy and sexual arousal.

Amnesia of traumatic experiences, with delayed recall for all or parts of the trauma, has been noted following natural disasters and accidents,^{7,34,38,39} war-related trauma,^{22,23,40-47} kidnapping, torture and concentration camp experiences,⁴⁸⁻⁵⁰ physical and sexual abuse,⁵¹⁻⁵⁴ and after committing murder.⁵⁵ A recent general population study by Elliot and Briere showed that total amnesia of traumatic events occurred in a certain proportion of victims after every conceivable traumatic experience (except for witnessing the death of one's child), and that, in addition, a substantially higher proportion of victims had significant amnesia of particular details of these traumatic experiences.⁵⁶ For reasons that are not at all clear, childhood sexual abuse seems to have the highest proportion of total amnesia prior to memory retrieval, with figures ranging from 19 to 38%.^{53,54} Amnesia of emotional and cognitive material seems to be age- and dose-related; the younger the age of the trauma, and the more prolonged, the greater the likelihood of significant amnesia.^{51,57,58}

Trauma and dissociation

Christianson described how, when people feel threatened, they experience a significant narrowing of consciousness, and remain merely focused on the central perceptual details.⁵⁹ As people are being traumatized this narrowing of consciousness seems to sometimes evolve into a complete amnesia of the experience. More than 80 years ago, Janet claimed:

"Forgetting the event which precipitated the emotion...has frequently been found to accompany intense emotional experiences in the form of continuous and retrograde amnesia... They are an exaggerated form of a general disturbance of memory which is characteristic of all emotions".⁶⁰

He claimed that when people become too upset, memories cannot be transformed into a neutral narrative; a person is 'unable to make the recital which we call narrative memory, and yet he remains confronted by (the) difficult situation'.¹ This results in 'a phobia of memory' that prevents the integration ('synthesis') of traumatic events and splits off the traumatic memories from ordinary consciousness.¹ Janet claimed that the memory traces of the trauma linger as what he called 'unconscious fixed ideas' that cannot be 'liquidated' as long as they have not been translated into a personal narrative. When this occurs they instead continue to intrude as terrifying perceptions, obsessional preoccupations and as somatic re-experiences such as anxiety reactions.

Similar observations have been made by other clinicians treating traumatized individuals. For example, in 1945 Grinker and Spiegel noted that some combat soldiers develop excessive responses under stress and that these are responsible for the transformation of stress into a permanent disorder.

"Fear and anger in small doses are stimulating and alert the ego, increasing efficacy. But, when stimulated by repeated psychological trauma the intensity of the emotion heightens until a point is reached at which the ego loses its effectiveness and may become altogether crippled....".²²

Grinker and Spiegel described that these soldiers suffered from severe anxiety states, which was accompanied by confusion, mutism and stupor. In civilian trauma victims Horowitz described an 'acute catastrophic stress reaction', characterized by panic, cognitive disorganization, disorientation and dissociation.⁶¹ Recent research has shown that having dissociative experiences at the moment of the trauma is the most important long-term predictor for the ultimate development of PTSD.⁶²⁻⁶⁴ Carlson and Rosser-Hogan found a strong relationship between severity of the trauma, dissociative symptoms and PTSD in Cambodian refugees.⁶⁵ Bremner *et al.* found that Vietnam veterans with PTSD reported having experienced higher levels of dissociative symptoms during combat than men who did not develop PTSD.⁶⁶ Koopman *et al.* found that dissociative symptoms early in the course of a natural disaster predicted PTSD symptoms 7 months later.⁶⁷ A prospective study of 51 injured trauma survivors in Israel found that peri-traumatic dissociation explained 30% of the variance in the 6

months follow-up PTSD symptoms, over and above the effects of gender, education, age, event-severity, and intrusion, avoidance anxiety and depression that followed the event.⁶⁸ Peri-traumatic dissociation was the strongest predictor of PTSD status 6 months after the event.

While dissociation may be adaptive under extreme conditions, the lack of integration of traumatic memories is thought to be the pathogenic agent that leads to the development of the complex biobehavioral changes, of which PTSD is the clinical manifestation. This observation was first made by Janet, and is confirmed by a subsequent century of clinical and research data. Janet proposed that intense arousal ('vehement emotion') seems to interfere with proper information processing and the storage of information into narrative (explicit) memory. Janet, and subsequent students of this issue, noted that during conditions of high arousal 'explicit memory' may fail. The individual is left in a state of 'speechless terror' in which words fail to describe what has happened. However, while traumatized individuals may be unable to make a coherent narrative of the incident, there may be no interference with implicit memory; traumatized people may 'know' the emotional valence of a stimulus and be aware of associated perceptions, without being able to articulate the reasons for feeling or behaving in a particular way. Janet proposed that traumatic memories are split off (dissociated) from consciousness, and, instead, stored as sensory perceptions, obsessional ruminations, or as behavioral reenactments.^{8,69,70} Janet's student Piaget described how an active failure of semantic memory leads to the organization of memory on somatosensory or iconic levels.⁷¹ He pointed out:

"It is precisely because there is no immediate accommodation that there is complete dissociation of the inner activity from the external world. As the external world is solely represented by images, it is assimilated without resistance (i.e., unattached to other memories) to the unconscious ego".

These and subsequent observations of other traumatized populations suggest that what may most complicate the capacity to communicate about traumatic experiences is that memories of trauma may have no verbal (explicit) component whatsoever. Instead, the memory may have been organized on an implicit or perceptual level, without an accompanying narrative about what happened. Recent symptom provocation neuroimaging studies of people with PTSD support the clinical observation; during the provocation of traumatic memories there is a decrease in activation of Broca's area, the part of the brain most centrally involved in the transformation of subjective experience into speech (Rauch *et al.*⁷²). Simultaneously, the areas

in the right hemisphere that are thought to process intense emotions and visual images had significantly increased activation.

ONGOING DISSOCIATION IN TRAUMATIZED PEOPLE

People who have learned to cope with trauma by dissociating often continue to do so in response to minor stresses. The continued use of dissociation as a way of coping with stress means that people remain emotionally constricted and inadequately respond to subsequent tasks. Traumatized people who have dissociated their traumatic memories seem to not be able to experience a full range of affects within the same ego state. The severity of ongoing dissociative processes (usually as measured with the DES, Bernstein and Putnam⁷³) has been correlated with a variety of psychopathological conditions that are thought to be associated with histories of trauma and neglect: severity of sexual abuse in adolescents, somatization,⁷⁴ bulimia,⁷⁵ self-mutilation⁷⁶ and Borderline Personality Disorder.⁷⁷ The most extreme example of this is people who suffer from dissociative identity disorder (multiple personality disorder), who have the highest DES scores of all populations studied and in whom separate identities seem to contain the memories related to different traumatic incidents.⁷⁸

The perceptual organization of traumatic experience

Numerous studies on trauma note that trauma is organized in the memory on a perceptual level.^{7,8,23,24} Having listened to the narratives of traumatic experiences from hundreds of traumatized children and adults over the past 20 years, we also keep hearing both adults and children describe how traumatic experiences initially are organized on a non-verbal level. Clinical experience and reading a century of observations by clinicians dealing with a variety of traumatized populations led us to postulate that 'memories' of the trauma tend to, at least initially, be experienced as fragments of the sensory components of the event; as visual images, olfactory, auditory, or kinesthetic sensations, or intense waves of feelings that patients usually claim to be representations of elements of the original traumatic event. What is intriguing is that patients consistently claim that their perceptions are exact representations of sensations at the time of the trauma. For example, when Southwick and his group injected yohimbine into Vietnam veterans with PTSD, half of their subjects reported trauma-related perceptions that they reported to be 'just like it was' (in Vietnam).⁶

In a recent study an instrument was designed, the Traumatic Memory Inventory (TMI), that allowed for a detailed examination of the nature of traumatic and non-traumatic memories, and provided a structured way of recording whether and how memories of traumatic experiences are retrieved differently from memories of personally significant, non-traumatic events.⁷⁹ In order to examine the retrieval of traumatic memories in a systematic way, we specifically inquired about sensory, affective and narrative ways of remembering, about triggers for unbidden recollections of traumatic memories, and ways of mastering. The TMI describes (i) nature of trauma(s), (ii) duration, (iii) whether subject has always been aware that trauma happened, and if not, when and where subject became conscious of trauma, (iv) circumstances under which the subject first experienced intrusive memories; and circumstances under which they occur presently, (v) sensory modalities in which memories were experienced as a story, as an image (what did you see?), in sounds (what did you hear?), as a smell (what did you smell?), as feelings in your body (what did you feel? where?), and as emotions (what did you feel, what was it like?). These data were collected for how subjects remembered the trauma initially, while the subject was most bothered by them, and currently. The TMI also describes (vi) the nature of flashbacks, (vii) the nature of nightmares, (viii) precipitants of flashbacks and nightmares, (ix) ways of mastering intrusive recollections (e.g., by eating, working, taking drugs of alcohol, cleaning, etc. and (x) confirmation (records: court or hospital, direct witness, relative went through same trauma).

We then asked our subjects the same question about a highly significant experience, such as a wedding, the birth of a child, etc., and collected the same information. Subjects considered most questions related to the non-traumatic memory nonsensical; none had olfactory, visual, auditory, kinesthetic re-living experiences related to such events as high school graduations, birthdays, weddings, or births. They denied having vivid dreams or flashbacks about these events. The subjects claimed not to have periods in their lives when they had amnesia of any of these events; none claimed to have photographic recollections of any of these events. Environmental triggers did not suddenly bring back vivid and detailed memories of these events, and no subject felt a need to make special efforts to suppress memories of these events.

When asked about the traumatic memory, all of these subjects reported that they initially had no narrative memory of the event; they could not tell a story about what had happened, regardless of whether they always knew that the trauma had happened, or whether they retrieved memories of the trauma at a later date.

All these subjects, regardless of the age at which the trauma occurred, claimed that they initially 'remembered' the trauma in the form of somatosensory flashback experiences. These flashbacks occurred in a variety of modalities: visual, olfactory, affective, auditory and kinesthetic, but initially these sensory modalities did not occur together. As the trauma came into consciousness with greater intensity, more sensory modalities were activated, and a capacity to tell themselves and others what had happened emerged over time.

This study suggested that there is a dramatic difference between the ways in which people experience traumatic memories versus other significant personal events. It supports the idea that is in the very nature of traumatic memory to be dissociated, and to be initially stored as sensory fragments that have no linguistic components. All of the subjects in this study claimed that they only came to develop a narrative of their trauma over time. Five of the subjects who claimed to have been abused as children were even unable to tell a complete narrative of what had happened to them as adults. They merely had fragmentary memories that supported other people's stories and their own intuitive feelings that they had been abused.

The traumatic experiences initially were not categorized into a narrative, and they seemed to serve no communicative function. It appears that as people become aware of more and more elements of the traumatic experience they construct a narrative that 'explains' what happened to them. This process of weaving a narrative out of disparate sensory elements of an experience is probably not all that dissimilar from how people automatically construct a narrative under ordinary conditions. However, when people have day-to-day, non-traumatic experiences, the sensory elements of the experience are non-registered separately in consciousness, but are automatically integrated into a personal narrative.

This study supports Piaget's notion that when memories cannot be integrated on a semantic/linguistic level, they tend to be organized in more primitive ways of information processing: as visual images or somatic sensations. Even after considerable periods of time, and even after acquiring a personal narrative for the traumatic experience, most subjects reported that these experiences continued to be come back as sensory perceptions and as affective states. The persistence of intrusive sensations related to the trauma after the construction of a narrative contradicts the notion that learning to put the traumatic experience into words will reliably help abolish the occurrence of flashbacks, which seems to be a central assumption in a variety of treatment modalities.

PSYCHOBIOLOGICAL ISSUES REGARDING TRAUMATIC MEMORIES

Trauma, neurohormones and memory consolidation

When people are under stress, they secrete endogenous stress hormones that affect the strength of memory consolidation. Based on animal models it has been widely assumed that massive secretion of neurohormones at the time of the trauma plays a role in the ways that traumatic experiences are consolidated into long-term memory. The critical issue here is the long-term potentiation (LTP) of memory traces.^{8,29} Mammals have memory storage mechanisms that modulate how strongly a memory is consolidated according to the strength of the accompanying hormonal stimulation.^{80,81} This capacity helps organisms evaluate the importance of sensory input in proportion to how strongly the associated memory traces are consolidated; emotionally significant material, consolidated in states of high arousal, is accessed more easily in subsequent states of high arousal. In traumatized organisms, the capacity to access relevant memories appears to have gone awry; they become too sensitive to access memory traces related to the trauma and, hence, they tend to 'remember' the trauma too easily, namely when it is irrelevant to current experience (Pitman *et al.*, *Black Hole of Trauma*).

The norepinephrine input to the amygdala determines how strongly a memory trace is consolidated.^{82,83} The role of norepinephrine in memory consolidation has been shown to have an inverted U-shaped function; both very low and very high levels of CNS norepinephrine activity interfere with memory storage.^{80,81} Physiological arousal in general can trigger trauma-related memories, while, conversely, trauma-related memories precipitate generalized physiological arousal. It is likely that the frequent re-living of a traumatic event in flashbacks or nightmares cause a re-release of stress hormones which further kindle the strength of the memory trace.⁸⁴

Traumatic memories are state dependent

Research has shown that, under ordinary conditions, people with PTSD often have a fairly good psychosocial adjustment. However, they do not respond to stress in the way other people do. Under pressure, they may feel, or act as if they were traumatized all over again. Thus, high states of arousal seem to selectively promote retrieval of traumatic memories, sensory information, or behaviors associated with prior traumatic experiences (APA 1987, 1994). Traumatized animals also have been shown to revert to irrelevant emergency be-

haviors in response to minor stresses. Rhesus monkeys with histories of severe early maternal deprivation, but not normally raised monkeys, become markedly withdrawn or aggressive in response to emotional or physical stimuli (such as exposure to loud noises, or the administration of amphetamines).⁸⁵ In mice, Mitchell *et al.* found that their response to novel situations depended on whether or not they had been previously exposed to high stresses.⁸⁶ In states of low arousal, animals tend to be curious and to seek novelty. Under ordinary conditions, an animal will choose the most pleasant of two alternatives. However, during states of high arousal, they avoid novelty, and revert back to what is familiar, regardless of the outcome. Mitchell *et al.* found that animals who have been locked in a box in which they were exposed to electric shocks and then released, return to those boxes when they are subsequently stressed. Mitchell *et al.* concluded that this return to familiar patterns of behavior is non-associative (i.e. uncoupled from the usual reward systems).

In people, analogous phenomena have been documented; memories (somatic or symbolic) related to the trauma are elicited by heightened arousal.⁸⁷ Information acquired in an aroused, or otherwise altered state of mind, is retrieved more readily when people are brought back to that particular state of mind.^{88,89} State dependent memory retrieval may also be involved in dissociative phenomena in which traumatized persons may be wholly or partially amnesic for memories or behaviors enacted while in altered states of mind.^{8,70,78}

Contemporary biological researchers have shown that medications that stimulate autonomic arousal may precipitate visual images and affect states associated with prior traumatic experiences in people with PTSD, but not in control subjects. In patients with PTSD the injection of drugs such as lactate⁵ and yohimbine⁶ tends to precipitate panic attacks, flashbacks (exact reliving experiences) of earlier trauma, or both. I believe that these are examples of state-dependent memory retrieval. 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; heightened arousal led to the retrieval of sensory elements of the trauma that were ordinarily inaccessible.

Functional and neuroanatomical correlates of PTSD, and their possible relationship to traumatic memory processes

Recent brain imaging studies of patients with PTSD have shown significant limbic system abnormalities. These may provide the beginnings of an understanding of both the memory storage and retrieval problems in

these patients. Two sets of significant findings have now been demonstrated in the laboratory: (i) decreased hippocampal volume in people with PTSD, and (ii) abnormal activation of the amygdala and related structures, abnormal lateralization, and decreased Broca's area activity when patients are induced to re-experience their traumas.

Three different studies, done in three different laboratories, have shown that people with PTSD have decreased hippocampal volumes, compared with matched controls. Bremner found that Vietnam combat veterans with PTSD had an 8% reduction in the volume of their right hippocampus compared with veterans who suffered no such symptoms.⁹⁰ Stein *et al.* found a 7% reduction in hippocampus volume in women with PTSD who had suffered repeated childhood sexual abuse.⁹¹ Gurvitz *et al.* found that Vietnam veterans with the most intense combat exposure and with the most severe PTSD had an average shrinkage of 26% in the left hippocampus and 22% in the right hippocampus, compared with veterans who saw combat but had no symptoms.⁹² The severity of their PTSD was directly proportional to the degree of hippocampal shrinkage.

I recently have been involved in PET scan studies of patients with PTSD in which they were exposed to vivid, detailed narratives of their own traumatic experiences.⁴ For example, a firefighter who had recovered the charred remains of bodies, and a woman who accidentally caused the death of her own two children by going through a red traffic light. These narratives were read to the patients to trigger their PTSD symptoms while PET scans were made. For comparison, the patients also wrote narratives that invoked a neutral scene. During exposure to the script of their traumatic experiences these subjects demonstrated heightened activity only in the right hemisphere; in the areas that are most involved in emotional arousal, the parts of the limbic system most intimately associated with the amygdala. Because these structures are central sites for the experience of anxiety, they have been called the 'worry circuit'. Activation of these structures was accompanied by heightened activity in the right visual cortex, reflecting the flashbacks that these patients reported. Perhaps most significantly,

Broca's area, the part of the left hemisphere responsible for translating personal experiences into communicable language, 'turned off'. We believe this to reflect the speechless terror experienced by these patients, and their tendency to experience emotions as physical states, rather than as verbally encoded experiences.

These findings indicate that the PTSD patient's difficulties in putting feelings into words is reflected in actual changes in brain activity. Striking in this study was the marked lateralization towards the right hemisphere, which is thought to be in charge of evaluating the emotional significance of incoming information and in regulating the autonomic and hormonal responses to that information. In contrast, Broca's area had a significant decrease in oxygen use during exposure to traumatic reminders. This probably means that during activation of the traumatic memory, the brain is 'having' its experience; the person may feel, see, or hear the sensory elements of the traumatic experience. He or she may be physiologically impaired 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'. Their bodies may respond as if they are being traumatized again, with the secretions of the various neurohormones that are mobilized on those occasions, but this is dissociated from subjective experience, and the victim cannot 'own' what is happening, and thus cannot take steps to do anything about it.

A neuroanatomical model for the failure of integration of traumatic memories

How can we interpret the significance of these findings for understanding the nature of PTSD? Table 1 provides a schematic presentation of the interrelationships between various brain structures involved in the interpretation, storage, and retrieval of information, and provides a scheme of what may occur when people suffer from PTSD. Sensory information enters the CNS via sensory organs (e.g. eyes, nose, skin, ears) which pass information on to the thalamus, where some of this information is integrated. The thalamus, in turn,

Table 1. The interrelationships between various brain structures involved in the interpretation, storage, and retrieval of information

Traumatic memory	Narrative memory
Images, sensations, affective and behavioral states	Narrative: semantic and symbolic
Invariable – does not change over time	Social and adaptive
Highly state-dependent	Cannot be evoked at will. Evoked at will by narrator
Automatically evoked in special circumstances	
No condensation in time	Can be condensed or expanded, depending on social demands

passes this raw sensory information on for further evaluation both to the amygdala and to the prefrontal cortex. The amygdala interprets the emotional valence of the incoming information; it attaches emotional significance to what is coming in. The information evaluated by the amygdala is passed on to areas in the brainstem that control behavioral 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.²⁶

LeDoux proposes that because input from the thalamus arrives at the amygdala before information from the neocortex, this earlier arrived sensory input from the thalamus 'prepares' the amygdala to process the later arriving information from the cortex. Thus, the emotional evaluation of sensory input precedes conscious emotional experience; people may become autonomically and hormonally activated before having been able to make a conscious appraisal of what they are reacting to. Thus, a high degree of activation of the amygdala and related structures can generate emotional responses and sensory impressions that are based on fragments of information, rather than full-blown perceptions of objects and events.²⁶ LeDoux points out that emotion itself can be a memory, and he advocates that emotion be treated as a memory process rather than as a process that simply influences memory.²⁶

Once the amygdala has assigned emotional significance to sensory input, it passes this evaluation on to other brain structures, including the hippocampus, whose task it is to begin organizing and categorizing this information with previously existing information about similar sensory input. The strength of the hippocampal activation is affected by the intensity of input from the amygdala; the more significance assigned by the amygdala, the stronger the input will be attended to and the memory retained. However, this interaction has an inverted U-shaped function; in animals, high level stimulation of the amygdala interferes with hippocampal functioning.^{17,93} This means that very high levels of emotional arousal may prevent the proper evaluation and categorization of experience by interfering with hippocampal function. One can hypothesize that when this occurs sensory imprints of experience are stored in the memory but because the hippocampus is prevented from fulfilling its integrative function, these various imprints are not united into a unified whole. The experience is consolidated, and later retrieved, as isolated images, bodily sensations, smells and sounds that feel alien, and separate from other life experiences. Because the hippocampus did not play its usual role in helping to localize the incoming information in time and space, these fragments continue to

lead an isolated existence. Traumatic memories are timeless, and ego-aient.

Effects of high levels of amygdala activation and decreased hippocampal volume

Both LeDoux and Ademas *et al.* have shown that one-time intense stimulation of the amygdala will produce lasting changes in neuronal excitability and enduring behavioral changes in the direction of either fight or flight.^{94,95} In kindling experiments with animals, Ademas *et al.* have shown that permanent changes in limbic physiology can cause lasting changes in defensiveness and in predatory aggression.⁹⁵ An animal's pre-existing 'personality' had a major effect on how high levels of amygdala stimulation affected them; 'fearless' animals become more aggressive, while shy and withdrawn animals become even more shy and withdrawn. In animals, previous personality characteristics can be exaggerated by trauma; it is likely that this is true for people.

The twice replicated finding that people with chronic PTSD have decreased hippocampal volume might explain some of the behavioral abnormalities seen in people with chronic PTSD. In animals, decreased hippocampal functioning causes behavioral disinhibition; it is likely that this is the stage for the animal to interpret incoming stimuli in the direction of deserving emergency (fight/flight) responses. If the same is true for people, this might explain why patients with PTSD have difficulties 'taking in' and processing arousing information, and to learn from such experiences. 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. Their decreased Broca's area functioning during this time would make it difficult for them to 'understand' what is going on; they experience intense emotions without being able to name their feelings. Their bodies are aroused, and fragments of memories may be activated, but they are unable to form a clear mental construct of what they are experiencing. Needing to re-establish their internal homeostasis, they use their muscles; discharge via smooth muscles leads to psychosomatic reactions, while discharge through the striated muscles leads to action. Both are likely to have adverse consequences. Neither solution allows much chance to learn from experience.

CONCLUSIONS

When people receive sensory input they generally automatically synthesize this incoming information into the large store of pre-existing information. If the event is personally significant they generally will transcribe

these sensations into a narrative, without conscious awareness of the processes that translate sensory impressions into a personal story. Our research shows that in contrast with the way people seem to process ordinary information, traumatic experiences initially are imprinted as sensations or feeling states, and are not collated and transcribed into personal narratives. Both my interviews with traumatized people, and my brain imaging studies of them, seem to confirm that traumatic memories come back as emotional and sensory states, with little capacity for verbal representation. This failure to process information on a symbolic level, which is essential for proper categorization and integration with other experiences, is at the very core of the pathology of PTSD.

We have earlier written about Janet's clear distinctions between traumatic and ordinary memory.⁸ According to Janet, traumatic memory consists of images, sensations, affective and behavioral states, that are invariable and do not change over time. He suggested that these memories are highly state-dependent and cannot be evoked at will. They also are not condensed in order to fit social expectations. In contrast, according to Janet, narrative (explicit) memory is semantic and symbolic, it is social, and adapted to the needs of both the narrator and the listener and can be expanded or contracted, according to social demands.

The irony is that, while the sensory perceptions reported in PTSD may well reflect the actual imprints of sensations that were recorded at the time of the trauma, all narratives that weave sensory imprints into a socially communicable story are subject to condensation, embellishment and contamination. While trauma may leave an indelible imprint, when people start talking about these sensations, and try to make meaning of them, it is transcribed into ordinary memory, and, like all ordinary memory, it is prone to become distorted. People seem to be unable to accept experiences that have no meaning; they will try to make sense of what they are feeling. Once people become conscious of intrusive elements of the trauma they are liable to try to fill in the blanks, and complete the picture.

Like all stories that people construct, our autobiographies contain elements of truth, of things that we wish did happen, but that did not, and elements that are meant to please the audience. The stories that people tell about their traumas are as vulnerable to distortion as people's stories about anything else. However, whether the brain is able to take pictures, and whether some smells, images, sounds, or physical sensations may be etched into the mind and remain unaltered by subsequent experience and by the passage of time, remains to be answered.

REFERENCES

1. Janet P. *Psychological Healing*, vols 1–2. Macmillan, New York 1925 (originally published 1919).
2. Seyle H. *The Stress of Life*. McGraw-Hill, New York, 1956.
3. Pitman RK, Orr SP, Forgue DF, de Jong J, Clairborn JM. Psychophysiologic assessment of posttraumatic stress disorder imagery in Vietnam combat veterans. *Arch. General Psychiatry* 1987; **17**: 970–975.
4. Rauch S, van der Kolk BA, Fisler R *et al.* Pet imagery: Positron immision scans of traumatic imagery in PTSD patients. Paper presented at the annual conference of ISTSS, Chicago, 1994.
5. Rainey JM, Aleem A, Ortiz Yaragani V, Pohl R, Berchow R. Laboratory procedure for the inducement of flashbacks. *Am. J. Psychiatry* 1987; **144**: 1317–1319.
6. Southwick SM, Krystal JH, Morgan A. Abnormal noradrenergic function in posttraumatic stress disorder. *Arch. Gen. Psychiatry* 1993; **50**: 266–274.
7. Janet P. *L'automatisme Psychologique*. Alcan, Paris, 1889.
8. van der Kolk BA, van der Hart O. The intrusive past: The flexibility of memory and the engraving of trauma. *Am. Imago* 1991; **48**: 425–454.
9. Schachtel EG. On memory and childhood amnesia. *Psychiatry* 1947; **10**: 1–26.
10. Bohannon JN. Arousal and memory: Quantity and consistency over the years. Paper presented at the Conference on Affect and Flashbulb Memories, Emory University, Georgia, 1990.
11. Christianson SA. Emotional stress and eyewitness memory: A critical review. *Psychol. Bull.* 1992; **112**: 284–309.
12. Pillemer DB. Flashbulb memories of the assassination attempt on President Reagan. *Cognition* 1984; **16**: 63–80.
13. Yuille JC, Cutshall JL. A case study of eyewitness memory of a crime. *J. Appl. Psychol.* 1986; **71**: 318–323.
14. Yuille JC, Cutshall JL. Analysis of the statements of victims, witnesses and suspects. In: Yuille JC (ed.), *Credibility Assessment*. Kluwer Academic Publishers, Dordrecht, 1989.
15. Brown R, Kulik J. Flashbulb memories. *Cognition* 1977; **5**: 73–99.
16. Neisser U, Harsch N. Phantom flashbulbs: False recollections of hearing the news about Challenger. Paper presented at the Emory Cognition Conference on Affect and Flashbulb Memories, Atlanta, Georgia, 1990.
17. Squire LR, Zola Morgan S. The medial temporal lobe memory system. *Science* 1991; **153**: 2380–2386.
18. van der Kolk BA. The body keeps the score: Memory and the evolving psychobiology of posttraumatic stress. *Harvard Rev. Psychiatry* 1994; **1**: 253–265.
19. Squire LR. Declarative and nondeclarative memory: Multiple brain systems supporting learning and memory. In: Schacter DL, Tulving E (eds), *Memory Systems*. MIT Press, Cambridge, USA, 1994.

20. Schacter DL. Implicit memory: History and current status. *J. Exp. Psychol. Learning, Memory, Cognition* 1987; **13**: 510–518.
21. van der Kolk BA, Blitz R, Burr WA, Hartmann E. Nightmares and trauma: Life-long and traumatic nightmares in Veterans. *Am. J. Psychiatry* 1984; **141**: 187–190.
22. Grinker RR, Spiegel JP. *Men Under Stress*. Blakiston, Philadelphia, 1945.
23. Kardiner A. *The Traumatic Neuroses of War*. Hoeber, New York, 1941.
24. Terr L. *Unchained Memories*. Basic Books, New York, 1993.
25. Heuer F, Rausberg D. Emotion, arousal and memory for detail. In: Christianson SA (ed.), *Handbook of Emotion and Memory*. Lawrence Erlbaum, Hillsdale, NJ, 1992; 151–506.
26. LeDoux JE. Emotion as memory: Anatomical systems underlying indelible neural traces. In: Christianson SA (ed.), *Handbook of Emotion and Memory*. Lawrence Erlbaum, Hillsdale, NJ, 1992; 269–288.
27. McGaugh JL. Affect, neuromodulatory systems, and memory storage. In: Christianson SA (ed.), *Handbook of Emotion and Memory*. Lawrence Erlbaum, Hillsdale, NJ, 1992; 245–268.
28. Nilsson LG, Archer T. Biological aspects of memory and emotion: Affect and cognition. In: Christianson SA (ed.), *Handbook of Emotion and Memory*. Lawrence Erlbaum, Hillsdale, NJ, 1992; 289–306.
29. Pitman R, Orr S, Shalev A. Once bitten twice shy: Beyond the conditioning model of PTSD. *Biol. Psychiatry* 1993; **33**: 145–146.
30. van der Kolk BA. The complexity of adaptation to trauma. In: McFarlane A, van der Kolk BA (eds), *The Black Hole of Trauma. Human Adaptation to Overwhelming Experience*. Guilford Press, New York, 1995.
31. Charcot JM. *Leçons sur les maladies du système nerveux faites... la Salpêtrière* [Lessons on the illnesses of the nervous system held at the Salpêtrière], tome III. Progrès Medical en A. Delahaye and E. Lecrosne, Paris, 1887.
32. van der Hart O, Steele K, Boon S, Brown P. The treatment of traumatic memories: Synthesis, realization, integration. *Dissociation* 1993; **6**: 162–180.
33. Janet P. L'Amnésie continue. *Revue Generale Des Science* 1893; **4**: 167–179.
34. van der Kolk BA, Kadish W. Amnesia, dissociation, and the return of the repressed. In: van der Kolk BA (ed.), *Psychological Trauma*. American Psychiatric Press, Washington, DC, 1987.
35. van der Kolk BA. The compulsion to repeat trauma. Revictimization, attachment and masochism. *Psychiatric Clin. North Am.* 1989; **12**: 389–411.
36. Collins AM, Loftus EF. A spreading activation theory of semantic processing. *Sychol Bull.* 1975; **82**: 407–428.
37. Leichtman MD, Ceci S, Ornstein PA. The influence of affect on memory: Mechanism and development. In: Christianson SA (ed.), *Handbook of Emotion and Memory*. Lawrence Erlbaum, Hillsdale NJ, 1992.
38. Madakasira S, O'Brian K. Acute posttraumatic stress disorder in victims of a natural disaster. *J. Nervous Mental Dis.* 1987; **175**: 286–290.
39. Wilkinson CB. Aftermath of a disaster: The collapse of the Hyatt Regency Hotel skywalks. *Am. J. Psychiatry* 1983; **140**: 1134–1139.
40. Archibald HC, Tuddenham RD. Persistent stress reaction after combat. *Arch. Gen. Psychiatry* 1956; **12**: 475–481.
41. Hendin H, Haas AP, Singer P. The reliving experience in Vietnam veterans with posttraumatic stress disorder. *Comp. Psychiatry* 1984; **25**: 165–173.
42. Kubie LS. Manual of emergency treatment for acute war neuroses. *War Med.* 1943; **4**: 582–599.
43. Myers CS. A contribution to the study of shell-shock. *Lancet* 1915; January: 316–320.
44. Sargant W, Slater E. Amnesic syndromes in war. *Proc. Royal Soc. Med.* 1941; **34**: 757–764.
45. Sonnenberg SM, Blank AS, Talbott JA. *The Trauma of War: Stress and Recovery in Vietnam Veterans*. American Psychiatric Press, Washington, DC, 1985.
46. Southard EE. Shell-shock and other neuropsychiatric problems. WW Leonard, Boston, 1919.
47. Thom DA, Fenton N. Amnesias in war cases. *Am. J. Insanity* 1920; **76**: 437–448.
48. Goldfeld AE, Mollica RF, Pesavento BH, Faraone SV. The physical and psychological sequelae of torture. *Symptomol. Diagnosis. J. Am. Med. Assoc.* 1988; **259**: 2725–2729.
49. Kinzie JD. Posttraumatic effects and their treatment among Southeast Asian refugees. In: Wilson JP, Raphael B (eds), *International Handbook of Traumatic Stress Syndromes*. Plenum, New York, 1993; 311–319.
50. Niederland WG. Clinical observations on the 'survivor syndrome'. *Int. J. Psychoanalysis* 1968; **49**: 313–315.
51. Briere J, Conte J. Self-reported amnesia for abuse in adults molested as children. *J. Traumatic Stress* 1993; **6**: 21–31.
52. Burkett E, Bruno F. *A Gospel of Shame*. Viking, New York, 1993.
53. Loftus EF, Polensky S, Fullilove MT. Memories of childhood sexual abuse. Remembering repressing. *Psychol. Women Q.* 1994; **18**: 67–84.
54. Williams LM. Adult memories of childhood abuse: Preliminary findings from a longitudinal study. *The Advisor* 1992; **5**: 19–20.
55. Schacter DL. Amnesia and crime: How much do we really know? *Am. Psychologist* 1986; **41**: 286–295.
56. Elliott DM, Briere J. Epidemiology of memory and trauma. Paper presented at the annual meeting of the International Society on Traumatic Stress Studies, Chicago, 1995.
57. Herman JE, Shatzow E. Recovery and verification of memories of childhood sexual trauma. *Psychoanalytic Psychol.* 1987; **4**: 1–14.
58. van der Kolk BA, Pelcovitz D, Roth S, Mandel FS, McFarlane A, Herman JL. Dissociation, somatization, and affect dysregulation: The complexity of adaptation of trauma. *Am. J. Psychiatry* 1996; **153**: 83–93.

59. Christianson SA. The relationship between induced emotional arousal and amnesia. *Scand. J. Psychol.* 1984; **25**: 147–160.
60. Janet P. *Les Nervoses*. Flammarion, Paris, 1909.
61. Horowitz MJ. Stress-response syndromes: A review of posttraumatic and adjustment disorders. *Hosp. Commun. Psychiatry* 1986; **37**: 241–249.
62. Holen A. The North Sea Oil Rig disaster. In: Wilson JP, Raphael B (eds), *International Handbook of Traumatic Stress Syndromes*. Plenum, New York, 1993; 471–478.
63. Marmar CR, Weiss DS, Schlenger WE *et al.* Peritraumatic dissociation and post-traumatic stress in male Vietnam theater veterans. *Am. J. Psychiatry* 1994; **151**: 902–907.
64. Spiegel D. Dissociation and trauma. In: Tasman A, Goldfinger SM (eds), *American Psychiatric Press Annual Review of Psychiatry* (Vol. 10), American Psychiatric Press, Washington, DC, 1991.
65. Carlson EB, Rosser-Hogan R. Trauma experiences, post-traumatic stress, dissociation, and depression in Cambodian refugees. *Am. J. Psychiatry* 1991; **148**: 1548–1551.
66. Bremner JD, Southwick SM, Brett E, Fontana A, Rosenheck R, Charney DS. Dissociation and posttraumatic stress disorder in Vietnam combat veterans. *Am. J. Psychiatry* 1992; **149**: 328–332.
67. Koopman C, Classen C, Spiegel D. Predictors of post-traumatic stress symptoms among survivors of the Oakland/Berkeley, California firestorm. *Am. J. Psychiatry* 1994; **151**: 888–894.
68. Shalev AY, Orr SP, Pitman RK. Psychophysiologic assessment of traumatic imagery in Israeli civilian patients with posttraumatic stress disorder. *Am. J. Psychiatry* 1993; **150**: 620–624.
69. Nemiah J. Early concepts of trauma, dissociation, and the unconscious: their history and current implications. In: Bremner D, Marmar CR (eds), *Trauma, Memory, and Dissociation*. American Psychiatric Press, Washington, DC, 1995.
70. van der Kolk BA, van der Hart O. Pierre Janet and the breakdown of adaptation in psychological trauma. *Am. J. Psychiatry* 1989; **146**: 1530–1540.
71. Piaget J. Play, dreams, and imitation in childhood. Longmans, Green, New York, 1962.
72. Rauch S, van der Kolk BA, Fisler R *et al.* A symptom provocation study using position emission tomography and script driven imagery. *Arch. Gen. Psychiatry* 1996; **53**: 380–387.
73. Bernstein EM, Putnam F. Development, reliability, and validity of a dissociation scale. *J. Nervous Mental Dis.* 1986; **174**: 727–735.
74. Saxe GN, Chinman G, Berkowitz R *et al.* Somatization in patients with dissociative disorders. *Am. J. Psychiatry* 1994; **151**: 1329–1335.
75. Demitrack MA, Putnam FW, Brewerton TD *et al.* Relation of clinical variables to dissociative phenomena in eating disorders. *Am. J. Psychiatry* 1990; **147**: 1184–1188.
76. van der Kolk BA, Perry JC, Herman JL. Childhood origins of self-destructive behavior. *Am. J. Psychiatry* 1991; **148**: 1665–1671.
77. Herman JL, Perry JC, van der Kolk BA. Childhood trauma in borderline personality disorder. *Am. J. Psychiatry* 1989; **146**: 490–495.
78. Putnam FW. *Diagnosis and Treatment of Multiple Personality Disorder*. Guilford, New York, 1989.
79. van der Kolk BA, Fisler R. Dissociation and the perceptual nature of traumatic memories: Review and experimental confirmation. *J. Traumatic Stress* 1995; **8**: 505–525.
80. McGaugh JL. Involvement of hormonal and neuro-modulatory systems in the regulation of memory storage. *Ann. Rev. Neurosci.* 1989; **2**: 255–287.
81. McGaugh JL, Weinberger NM, Lynch G, Granger RH. Neural mechanisms of learning and memory. *Cells, Systems Computations. Navel Res. Rev.* 1985; **37**: 15–29.
82. Ademas RE. Normal and abnormal limbic system mechanisms of emotive biasing. In: Livingston KE, Hornykiewicz O (eds), *Limbic Mechanisms*. Plenum Press, New York, 1978.
83. LeDoux JE. Information flow from sensation to emotion: Plasticity of the neural computation of stimulus value. In: Gabriel M, Moore J (eds), *Learning Computational Neuroscience: Foundations of Adaptive Networks*. MIT press, Cambridge, MA, 1990.
84. van der Kolk BA, Greenberg MS, Boyd H, Krystal JH. Inescapable shock, neurotransmitters and addiction to trauma: Towards a psychobiology of post traumatic stress. *Biol. Psychiatry* 1985; **20**: 314–325.
85. Kraemer GW. Effects of differences in early social experiences on primate neurobiological behavioral development. In: Reite *et al.* (eds), *The Psychology of Attachment and Separation*. Academic Press, Orlando, FL, 1985.
86. Mitchell D, Osbourne EW, O'Boyle MW. Habituation under stress: Shocked mice show non-associative learning in a T-maze. *Behav. Neuro. Biol.* 1985; **43**: 212–217.
87. Solomon A, Garb R, Bleich A, Grupper D. Reactivation of combat-related post-traumatic stress disorder. *Am. J. Psychiatry* 1985; **144**: 51–55.
88. Phillips AG, LePiane FG. Disruption of conditioned taste aversion in the rat by stimulation of amygdala: A conditioned effect, not amnesia. *J. Comp. Physiol. Psychol.* 1980; **94**: 664–674.
89. Rawlins JNP. Associative and non-associative mechanisms in the development of tolerance for stress: The problem of state dependent learning. In: Levine S, Ursin H (eds), *Coping and Health*. Plenum Press, New York, 1980.
90. Bremner JD, Randall P, Scott TM *et al.* MRI-based measured of hippocampal volume in patients with PTSD. *Am. J. Psychiat.* 1995; **152**: 973–981.
91. Stein MB, Hannah C, Koverola C, Yehuda R, Torchia M, McClarty B. Neuroanatomical and neuroendocrine correlates in adulthood of severe sexual abuse in childhood. Paper presented at the 33rd annual meeting of the American College of Neuropsychopharmacology, San Juan, PR.
92. Gurvitz TV, Shenton ME, Pitman RK. Reduced hippocampal volume on magnetic resonance imaging in chronic posttraumatic stress disorder. Paper presented at the

- annual meeting of the International Society on Traumatic Stress Studies, Miami, 1995.
93. Ademas RE. Partial kindling of the ventral hippocampus: Identification of changes in limbic physiology which accompany changes in feline aggression and defense. *Physiol. Behav.* 1991; **49**: 443–454.
 94. LeDoux JE, Romanski L, Xagoraris A. Indelibility of subcortical emotional memories. *J. Cog. Neurosci.* 1991; **1**: 238–243.
 95. Ademas RE, Stark-Ademas C, Livingston KE. The development of predatory aggression and defense in the domestic cat. *Neurol. Biol.* 1989; **30**: 389–447.

Copyright of *Psychiatry & Clinical Neurosciences* is the property of Blackwell Publishing Limited and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.