Chapter 12

Trauma, Memory, and Dissociation: An Integrative Formulation

J. Douglas Bremner, M.D., Eric Vermetten, M.D., Steven M. Southwick, M.D., John H. Krystal, M.D., and Dennis S. Charney, M.D.

Several theoretical papers published in 1989 introduced a new phase in trauma research. Nemiah (1989), Putnam (1989), and van der Kolk and van der Hart (1989) (see also Nemiah, Chapter 1; Putnam and Carlson, Chapter 2; van der Hart et al., Chapter 9, in this volume) published reviews that highlighted the contribution Pierre Janet (1889, 1920) has made to the field of psychological trauma. Janet gave the first description of symptoms associated with extreme stress, including both what is known today as symptoms of posttraumatic stress disorder (PTSD) and symptoms of dissociation. He also pointed out that individuals who dissociated in response to a single trauma were at increased risk to dissociate with subsequent stressors. Whether this subsequent dissociation is due to a constitutional predisposition to dissociate or whether psychological trauma has an effect on the individual that results in long-term susceptibility to dissociation is an open question. Janet himself felt that there was in fact a contribution of both factors. The renewed focus on the theoretical contributions of Janet stimulated empirical research in this area, which is represented by the chapters in this volume.

Recently, alterations in memory (which are an important component of dissociation) have been the subject of considerable interest. In addition to the obvious role that alterations in memory play in amnesia, they also are involved in other dissociative symptoms. For example, amnestic episodes often involve symptoms of

derealization and depersonalization, and traumatic memories are typically recalled in a state of derealization or depersonalization. Identity alterations can involve gaps in memory and are intimately related to alterations in autobiographical memory, which recently have received appropriately increased attention in the field of traumatic stress (see van der Hart et al., Chapter 9, in this volume). It is clear that understanding the relationship between stress-induced alterations in memory and dissociation will contribute to our understanding of the effects of traumatic stress on the individual. In this chapter, we review the relationship between dissociation and alterations in memory in traumatized individuals, using both theoretical and biological frameworks (Bremner et al. 1993c, 1995a; Charney et al. 1993) in an attempt to develop a more comprehensive understanding of the effects of stress on the individual.

Is There Specificity for the Dissociative Disorders, Posttraumatic Stress Disorder, and Other Trauma-Spectrum Psychiatric Disorders?

Some authors have questioned whether there is specificity to diagnoses such as PTSD. Implicit in this criticism is the idea that if PTSD cannot be shown to have specificity in relation to other disorders, such as depression, that are commonly held to have validity as constructs, then this indicates that PTSD does not have validity as a diagnosis. Taking the other side of this argument, clinicians who commonly treat patients with PTSD have concluded, based on their clinical experience, that PTSD does indeed have validity as a diagnosis. They therefore attempt to argue for the specificity of the disorder, which they feel is needed to support their viewpoint that PTSD has validity as a diagnosis. Both lines of reasoning, of course, are based on the assumption that PTSD should have specificity in relation to other diagnoses to be valid as a diagnosis.

The current diagnostic nomenclature has contributed to this issue of specificity, in addition to in many ways inhibiting a broader understanding of the relationship between traumatic stress and psychiatric symptomatology. The editions of the *Diagnostic and*

Statistical Manual of Mental Disorders written since the original recognition of PTSD as a diagnostic entity in 1980 have separated PTSD from dissociative disorders and other disorders that are believed to be related to traumatic stress, such as depression or borderline personality disorder. Most people focus on a particular disorder, such as PTSD or the dissociative disorders, to the exclusion of the rest. Clinicians tend to ask about the symptoms with which they are most familiar. If patients have a wide range of trauma-related symptoms in a PTSD-dissociative-anxiety spectrum, they will therefore be diagnosed as having PTSD by a PTSD-focused clinician, a dissociative disorder by a dissociation-focused clinician, and so on. What is missing is an appreciation for the central role that traumatic stress plays in these psychiatric disorders.

PTSD is the only disorder that has the requirement for diagnosis of exposure to a traumatic stressor. Clinicians and investigators therefore were focused from the outset on the primacy of the traumatic experience in the symptoms of patients with PTSD. Investigators working in other trauma-related disorders, such as the dissociative disorders, discovered that psychological trauma also was an important part of the presenting symptoms of their patients (Putnam et al. 1986; Spiegel and Cardena 1991). As clinicians and researchers began communicating with each other more, and as studies were conducted addressing these issues of traumatic exposure, they realized that there was considerable overlap between their patient populations. Converging evidence now suggests that traumatic exposure results in a range of symptom outcomes that go beyond DSM-IV (American Psychiatric Association 1994) criteria for PTSD. For instance, there are a number of studies that have examined the relationship between the onset of depression and exposure to stressful life events (Mazure 1994). More than 80% of patients with the diagnosis of borderline personality disorder have been found to have a history of exposure to extreme childhood abuse (Herman et al. 1989); 90% of patients with the diagnosis of multiple personality disorder (what is termed today by DSM-IV as dissociative identity disorder) have been found to have a history of exposure to extreme childhood abuse (Putnam et al.

1986). Childhood abuse also has been associated with alcoholism, substance abuse, panic disorder, and eating disorders (Finkelhor 1986).

There is considerable overlap between the symptoms listed in the diagnostic criteria for psychiatric disorders associated with traumatic stress. For instance, many symptoms of depression are equivalent to symptoms of PTSD. Psychomotor agitation can be rephrased as hyperarousal, and hopelessness as a sense of foreshortened future. Other symptoms that are identical in the criteria for depression and PTSD include decreased sleep, decreased concentration, and feeling cut off from others. In fact, the only symptom of depression that is not included in the criteria for PTSD is depressed mood, and most clinicians who work with PTSD patients feel that these patients are frequently anhedonic. The only symptoms of PTSD that are not a part of depression are increased startle, feeling on guard, flashbacks, and amnesia.

These symptoms also overlap with other psychiatric disorders. For instance, flashbacks and amnesia are part of PTSD but essentially represent dissociative phenomena (Spiegel 1984). Flashbacks in PTSD patients would potentially qualify diagnostically for panic attacks in 100% of cases (Mellman and Davis 1985), and dissociative symptoms such as derealization and depersonalization form part of the spectrum of symptoms that occur during panic attacks in patients with panic disorder. Disturbances of identity are captured by the borderline personality disorder symptom of feeling empty inside, are part of dissociative identity disorder, and are also commonly seen in PTSD patients (although not part of the diagnostic criteria for PTSD). Patients with borderline personality disorder are also commonly observed to have dissociative symptoms during self-mutilation or express the desire to use self-mutilation to break out of states of depersonalization or derealization. Figure 12–1 shows the range of overlap of symptomatology existing among psychiatric disorders that have been linked with trauma, including PTSD, depression, dissociative disorders, borderline personality disorder, alcoholism, substance abuse, and anxiety disorders. This overlap suggests that PTSD and dissociation are part of a continuum of trauma-spectrum psychiatric disor-

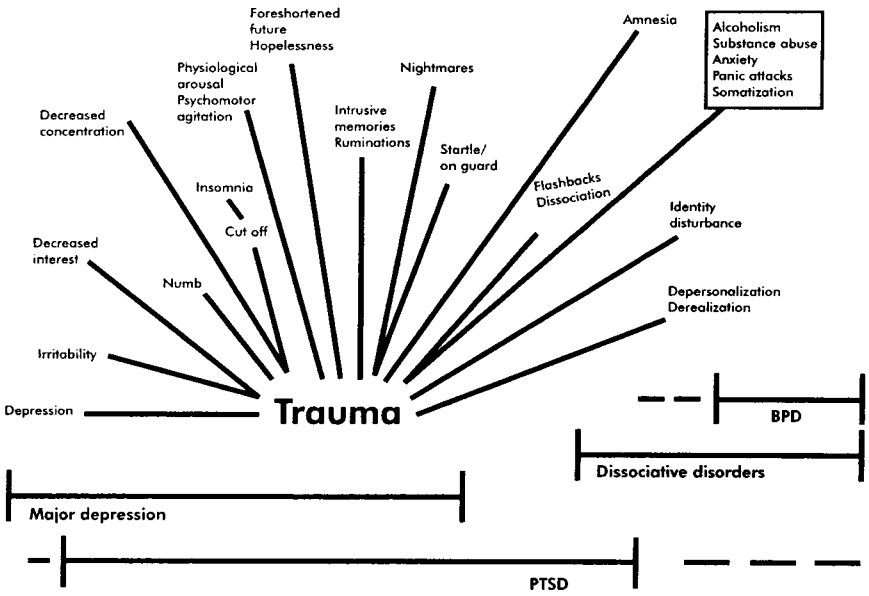

Figure 12–1. Range of symptomatology among psychiatric disorders linked with trauma. *Note.* BPD = borderline personality disorder. PTSD = posttraumatic stress disorder.

ders. According to this model, clinicians should not be focusing on whether PTSD has specificity as a diagnosis but should be thinking about the range of psychiatric symptoms that can be part of the outcome of exposure to traumatic stress.

Dissociative Responses to Trauma

Empirical findings are consistent with an overlap between PTSD and the dissociative disorder. In 1992, we studied Vietnam combat veterans with and without PTSD by using the Dissociative Experiences Scale (DES) of Bernstein and Putnam (1986). Vietnam combat veterans with the diagnosis of PTSD (N = 53) were compared with Vietnam combat veterans without PTSD (N = 32) who were recruited from a Veterans Administration outpatient medical clinic. PTSD patients in that study had a significantly higher score on the

DES (27.0 [18.0 SD]) than Vietnam veterans without PTSD (13.7 [SD = 16.0]). This difference persisted after statistically controlling for differences in level of traumatic exposure in the form of combat stress for combat veterans with and without PTSD. Dissociative symptoms as measured with the DES were correlated with PTSD symptoms measured with the Mississippi Scale for Combat-Related Posttraumatic Stress Disorder (Keane et al. 1988) ($r = 0.34$, $P < .05$). PTSD patients reported higher levels of dissociation at the time of combat-related traumatic events than did non-PTSD subjects. The magnitude of difference was substantial, suggesting an all-or-none type of phenomenon. On a 13-point scale for the measurement of dissociative states (a modification of the PDEQ by Marmar et al. [see Chapter 8 of this volume]) mean score for the PTSD patients was 11.5 (1.6 SD) compared with 1.8 (2.1 SD) for the non-PTSD subjects. In addition, dissociation at the time of combat trauma was strongly related to current PTSD symptomatology (as measured with the Mississippi Scale), after adjusting for level of combat exposure, participation in atrocities, and number of months in Vietnam, with multiple linear regressions (Bremner et al. 1992). We also have found an increase in baseline dissociative states as measured with the Clinician-Administered Dissociative States Scale (CADSS) in PTSD patients in comparison with patients with other psychiatric disorders and control subjects (Bremner et al., in press). The CADSS was found to have good interrater reliability, test-retest reliability, and convergent validity with the DES and SCID-D. [1]

In addition, consistent with Janet's formulation that dissociation due to extreme trauma increased the risk for dissociative responses to subsequent traumas, as well as for long-term psychopathology (Janet 1889, 1920), Vietnam combat veterans with PTSD who dissociated at the time of trauma had an increase in dissociative responses to subsequent postmilitary stressful events. We also found that PTSD patients had an increase in dissociative states

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[1]The CADSS is available on the Internet at the following address: http://info.med.yale.edu/psych/org/ypi/trauma/cadss.txt

during traumatic recall (reading scripts of their traumatic events) as measured with the CADSS (Bremner et al., in press).

Other studies have reported an association between trauma and dissociative symptomatology. These studies have found an increase in hypnotizability (Spiegel et al. 1988) and dissociative symptomatology (Bremner et al. 1992, 1993e; Carlson and Rosser-Hogan 1991; Loewenstein and Putnam 1988; Marmar et al. 1994) in patients with PTSD. In addition, individuals exposed to childhood abuse (Chu and Dill 1990; Putnam et al. 1986) and the acute stressors of natural disasters (Cardena and Spiegel 1989; Koopman et al. 1994) have been observed to have an increase in dissociative symptomatology. Other studies have found a relationship between dissociation at the time of trauma and long-term psychopathology (Koopman et al. 1994; Marmar et al. 1994).

Patients with trauma-related dissociation experience a wide range of dissociative symptomatology. We examined dissociative symptoms with the Structured Clinical Interview for DSM-IV Dissociative Disorders (SCID-D; Steinberg 1993) in patients with Vietnam combat-related PTSD ($N = 40$) in comparison with Vietnam veterans without PTSD ($N = 15$). Vietnam veterans with PTSD had markedly increased levels of dissociative symptomatology in comparison with non-PTSD subjects. For instance, mean score on the amnesia subscale of the SCID-D, which measures gaps in memory that are not due to ordinary forgetting, in PTSD patients was 3.7 (0.8 SD) on a scale of 1–4, compared with 1.1 (0.3 SD) in the non-PTSD patients. Other symptoms also were increased in PTSD patients compared with non-PTSD subjects as measured on the subscales of the SCID-D (Bremner et al. 1993d), including derealization (3.2 [1.2 SD] versus 1.1 [0.3 SD]), a measure of changes in the perception of one's environment; depersonalization (3.4 [1.1 SD] versus 1.1 [0.4 SD]), a measure of distortions in one's perception of one's body; and identity confusion and identity alteration. In summary, these studies are consistent with a high degree of overlap between PTSD and the dissociative disorders and suggest that dissociative responses to trauma are often seen in patients who later develop psychopathology related to traumatic stress.

We have raised a number of questions about the current diag-

nostic separation of PTSD from other psychiatric disorders, including dissociative disorders, depression, and anxiety disorders. One therefore might ask, Should the diagnostic schema be changed and PTSD included as one of the dissociative disorders, or should all of these disorders be collapsed into a single "disorder of stress"? The answer is that currently there are not sufficient data to justify any particular reformulation of the psychiatric consequences of traumatic stress.

Is Dissociation a Sign of Pathology or Part of a Normal Continuum?

If PTSD and dissociative disorders are part of a spectrum of disorders related to psychological trauma, dissociation should be a pathological outcome of trauma. There is currently debate, however, about whether dissociation is part of a normal spectrum of personality traits or a marker of psychopathology. Dissociation could be an adaptive response to trauma, and increases in dissociation in traumatized populations could be a by-product of the defenses of those individuals. A second possibility is that individuals who are born with a capacity for dissociation are more likely to develop psychiatric disorders. In this situation, dissociation would represent a risk factor for the development of psychiatric disorders in response to traumatic stress. A third possibility is that dissociation during trauma and dissociative responses to subsequent stressors are markers of psychopathology related to traumatic stress. A fourth possibility is that dissociation is a normal personality trait that varies in the general population, is nonspecifically associated with psychiatric disorders, but is not related to the development of psychopathology.

One strategy in approaching these four possibilities is to examine the relationship between dissociation and constructs that are a part of normal personality. Constructs that appear to be phenomenologically similar to dissociation include hypnotizability, absorption, fantasy proneness, and openness to experience (Kihlstrom 1992; Kihlstrom et al. 1994). Because these constructs occur nor-

mally in the general population, a relationship between them and dissociation would suggest that dissociation may be a healthy personality trait rather than a marker of psychopathology. Absorption occurs in healthy individuals and involves the ability to become completely involved in something to the point where everything else is excluded. The Tellegen Absorption Scale (TAS; Tellegen and Atkinson 1974), which is used to measure absorption, includes items such as "sometimes when I am reading poetry I feel a chill of excitement" and "I enjoy concentrating on a fantasy or daydream and exploring all its possibilities, letting it grow and develop." Absorption, as measured by the TAS, is strongly related to other constructs that are healthy personality traits, such as openness to experience (r = .62–.85 [Glisky et al. 1991; Radtke and Stam 1991]) and daydreaming (r = .57 [Hoyt et al. 1989]). Absorption has been shown to be modestly correlated with hypnotizability (Tellegen and Atkinson 1974), which is normally distributed in the population (r = .3 [Frischholz et al. 1987]; r = .15 [Glisky and Kihlstrom 1993]; r = .22 [Glisky et al. 1991; Radtke and Stam 1991]; r = .24 [Hoyt et al. 1989]). Absorption also has been shown to be modestly correlated with dissociation as measured by the DES (Nadon et al. 1991). However, the relationship between the TAS and the DES weakens significantly when absorption-like items are removed from the DES (Kihlstrom et al. 1994). This finding raises the question of whether absorption is really related to the "core" symptoms of dissociation. Dissociation and hypnotizability, as reviewed by Putnam and Carlson (Chapter 2, in this volume), are only weakly correlated (r = .1–.2) in healthy populations, although Spiegel et al. (1988) have found increases in hypnotizability in traumatized psychiatric populations, who also have been found to have increases in dissociative symptomatology. Hypnotizability also has been shown to be weakly correlated with openness to experience and fantasy proneness (daydreaming) (r = .1–.2 [Glisky and Kihlstrom 1993]). In summary, these findings do not provide convincing evidence that dissociation is related to nonpathological personality traits, although they do not necessarily provide evidence for the opposite view that dissociation is a construct of pathology.

The determination of pathology is based on the presence of symptoms that result in disability and distress. To judge whether dissociative symptoms are disabling, it is important to have an appreciation for how the symptoms manifest themselves in patients. In the following discussion, we describe some clinical cases of Vietnam veterans with PTSD and high levels of dissociative symptomatology.

Clinical Presentations of Dissociation in Patients With Posttraumatic Stress Disorder

Case Example 1

D is a 44-year-old white male who came from a working-class neighborhood in Connecticut. His childhood was without incident, and he described himself as an "average American kid." He was drafted out of high school and served in the army in Vietnam from 1967 to 1968.

D drove a "track," a mobile artillery unit that fired rounds at the enemy, during his frequent combat missions into enemy territory. He became good friends during his tour with a man named Bobby, and they would help each other out during times of need. One day D's unit was on a patrol, and Bobby was driving his track just in front of D, when the unit was suddenly ambushed by the Vietcong. An artillery round hit Bobby's track, and Bobby started screaming for help. D jumped out of his track and ran to help Bobby. As D pulled him out of the track, Bobby emitted a chilling moan, and he realized that the lower part of Bobby's body was missing. Suddenly, it seemed to D as if everything around him was slowing down. Sounds were disappearing, even though the battle continued, and D's perception of the world around him changed. Everything seemed foggy, as if it were far away, and it seemed as if he were looking through a tunnel, with the periphery of his vision gone. The world seemed strange or foreign, and D could not recognize his friends or his surroundings (symptoms of derealization). He felt like he had no idea who he was (depersonalization). After the battle, D sat immobile and was unresponsive to others. Some of his friends protected him by hiding him in a Vietnamese hooch (straw house) for 2 days. He sat on the floor, not knowing who he was or

being able to recognize his surroundings, only looking at the glint coming off a knife that he held in his lap.

After his discharge from the army, D returned to his hometown where he married, started working at the local factory, and later had a son. D continued to have discrete episodes, usually during times of stress, of suddenly being unable to recognize familiar surroundings or people close to him, such as his wife and son. D said that during these episodes, he would look at his wife and be able to recognize her facial features but be completely without the sensation of familiarity. During one episode, D was on a family vacation in Florida, and he, his wife, and son were walking through crowded Disney World. He suddenly had the sensation that all of the people around him were strange or foreign. Looking at his family, he felt as if he did not know who they were. Colors in the environment seemed blurred, and his surroundings seemed foreign or strange. He said that his wife had to lead him by the hand out of Disney World, and they could not spend any more time there during their vacation.

Case Example 2

B is a 44-year-old white male who served in the army from 1963 to 1966. He was a jokester who was everybody's friend in high school. After graduation from high school, a group of his friends went down to the army recruiter to get information about enlisting, and so he went along. He enlisted to have the opportunity of being with his friends.

Soon after his enlistment, the United States became involved in the Vietnam War. B was sent over as a combat infantryman. One time, B was killing wounded enemies with a bayonet instead of a gun to prevent the gunfire from revealing their position to enemy reinforcements. B described the experience in this way:

> As I was bayoneting the wounded, I had the sensation of separating from myself and looking down from a distance at the "killer" who was bayoneting the enemy soldiers. I first had this feeling when I noticed some pleasure within myself at killing the enemy, which filled me with horror and disgust and which seemed unconnected with my conception of myself and of what I felt I was capable of doing. From that time

> on, in combat situations I would separate from myself and watch while the killer would carry out combat missions. The killer was better able to do the job, as he was without concern for others or fear, unlike myself.

After his discharge from the army in 1966, B reported continued episodes of separating from his body in times of stress, usually when he became angry or felt physically threatened. These episodes occurred during situations such as barroom brawls, which recreated the experience of being in combat. B came for treatment with the chief complaint of "blackouts," which were increasing in frequency and which he felt he had no control over (amnesia). A seizure disorder had been ruled out with electroencephalogram and computed tomography of the head. After a careful history, it was discovered that these blackouts were actually episodes in which he would separate from his body and watch while the killer would do something harmful or destructive that would get him into trouble. During the most recent episode, B had become angry at someone during the course of an argument and ran home to get a knife. While running back to the site of the dispute, B separated from his body (depersonalization) and watched with consternation the activity of the killer (identity alteration). B ran up to the other person and put the knife to his throat, while B tried to urge the killer not to do anything that would get him into trouble. B eventually was able to get control of himself (or the other side of himself) and the other person was not hurt.

Case Example 3

P was born and raised in a small town in Connecticut. At the age of 18, he joined the Marines and was sent to Vietnam for active combat duty. He was a member of the 101st cavalry, General Custer's division.

In 1969, while on patrol in Vietnam, P's unit was ambushed and his best friends, Johny, Willy, and Mack, were killed. Willy walked into a claymore mine and it blew his legs off and blew his body into P, knocking him down. Then the Vietcong opened fire. They shot Mack in the face. P saw it happen as if in slow motion (derealization). He saw his arm moving and the glitter of his watch. Then he saw Johny with a bullet in his head. He had a strange look, with his eye blown out of the socket. P related the event in the following way:

> Johny was screaming. I held him in my arms, rocking him. He told me to tell his mother he was sorry and that it hurt real bad. Then he told me to shoot him, and I did.
>
> I felt like the experience was drawn out over a long period of time. I had moments of losing track of what was happening, and things happened during the event that I later couldn't account for. At first, things became extremely bright, like under the floodlights at night in a baseball field, and then suddenly, colors became very dull as if I were looking at the world through a fog. It seemed like I was looking at things through a tunnel or as if there were a pair of binoculars turned backwards. I could hear the moaning of my dying friends, but it sounded long and drawn out. Things seemed unreal, as if I were in a dream or as if I were watching the situation as a spectator. I felt disconnected from my own body, like I could look back at myself and see my own reaction. My own body felt much larger than normal, like a giant, and I thought that I would get hit by a bullet. I felt like I was "in shock."

P started having episodes where he saw his dead buddies soon after returning from the war. The first time he saw them it was dusk, and he was sitting on his porch. There was a field behind the house. They were out there, on patrol, walking in full combat gear. He started calling them. His sister said, "Who are you calling?" He said, "Call, you can get them back." He remembered her calling for them there on the porch, even though she could not see them.

P would sit at the bar in his basement and drink with his dead buddies. He walked into the house one day, and they were sitting there at the bar. They drank beer together. They told him to go back to Vietnam with them and that they would be together again. They would fight together again, but this time they would win the war. They had the smell of the Mekong delta, a damp smell.

P was at his sister's house walking through a field in the backyard. It looked like a landing zone. He had the sensation of walking through a bubble. He heard a fleet of helicopters. His daughter came and hugged him and asked what was wrong—did he hear helicopters? He said, "Yeah, can't you hear them?" She said, "Let them land; don't go with them." He went out and got in his car. Then he saw his dead buddies. Johny was sitting next to him. He was young and had freckles, he was only 18. He started driving.

> Willy and Mack were in the back seat. They did not say anything; they were like zombies. They were wearing combat fatigues, and their faces were real white. You could see their freckles like they were glowing. P said to himself, "It's not real, it's in my mind." They were not talking, but he felt he could read their minds. He felt scared. They had been with him a long time, and he was getting scared of them. Then Johny grabbed the wheel and yanked it. The car drove off the road onto someone's lawn. He felt light-headed and was having a hard time breathing. He drove home, parked the car, and ran up to see his girlfriend. He said, "Get them out of my car." She went down and looked and said that there was no one there.

These case examples illustrate the wide range of dissociative symptoms that can be associated with exposure to psychological trauma. They also show how dissociative symptoms occur at the time of the original trauma and then recur, often in a similar form, during exposure to subsequent stressors. These stressors, as seen in the case examples, often involve something as trivial as a trip to Disney World and have an important impact on patient functioning. Dissociation is a part of the daily lives of these patients, which makes it difficult for them to interact socially with family and friends and to cope with challenges in their environment.

There are important interrelationships between individual symptoms of dissociation. For example, amnesia can be associated with depersonalization or identity alteration, and depersonalization and derealization can occur simultaneously. Consistent with these interrelationships, we have found a high degree of correlation between individual symptoms of dissociation as measured with the CADSS (Bremner et al., in press). These data suggest that the individual symptoms of dissociation do not have validity as separate constructs. As shown in the previous case examples, patients typically experience dissociative symptoms in combination with one another.

Dissociation and Memory

Dissociation can be conceptualized as being related to alterations in memory function. Psychogenic amnesia is characterized by gaps in

memory lasting from minutes to days. Pathological recall of traumatic memories typically occurs in a dissociated state, with symptoms of derealization and depersonalization. Dissociative identity disorder essentially involves the transition between different collections of autobiographical memories that have coherence as separate entities. Patients with disorders such as PTSD have a variety of other manifestations of alterations in memory, including deficits in explicit and implicit memory function. In addition, hypnosis, which is phenomenologically similar to dissociation, involves many features that resemble alterations in memory function. Individuals undergoing hypnosis are subject to posthypnotic amnesia, in which the hypnotist can suggest to them that they will have no memory for what occurred while they were under hypnosis. Hypnosis has been described, in fact, as "an interaction in which one person experiences subjectively compelling responses to suggestions offered by another person, the hypnotist, of imaginative alterations in *"perception and memory"* (Kihlstrom 1992, p. 307, italics added).

Mechanisms of Normal Memory Function

To fully understand how stress-related alteration in memory function relates to dissociation and PTSD, it is useful to review mechanisms of healthy memory function. Memory formation involves encoding, storage (or consolidation), and retrieval. Encoding is the initial laying down of the memory trace. Storage involves the keeping of the memory trace over time. A related concept is consolidation, which refers to the process, occurring over several weeks or more, of establishing the permanence of a memory trace, during which time the memory trace is theoretically susceptible to modification. Retrieval is the process of bringing out a memory from storage into consciousness.

Memory function can be divided into explicit (declarative) and implicit (nondeclarative or "procedural") (Squire and Zola-Morgan 1991). Explicit memory includes free recall of facts and lists, as well as working memory, which is the ability to store information in a visual or verbal buffer while performing a particular

operation using that information. In contrast, implicit memory is demonstrated only through tasks or skills in which the knowledge is embedded. Forms of implicit memory include priming, conditioning, and tasks or skills. Priming involves providing the first few letters of a word and asking the subject to say the first word that comes to mind. Conditioning refers to the development of consistent physiological and emotional responses to a previously neutral stimulus. The most common animal model of conditioning (reviewed in the following discussion) involves pairing a tone or a light (the conditioned stimulus) with an electric shock (the unconditioned stimulus). With repeated trials, the conditioned stimulus alone will be evoke responses, such as an increase in startle amplitude, that were previously only elicited by the unconditioned stimulus. This paradigm implies that a form of learning has occurred that is embedded in the conditioned response and is not available for conscious recall.

Neuroanatomical Basis of Memory

Several brain structures are involved in a mutually interrelated network in the mediation of memory function. The hippocampus and adjacent cortex (entorhinal, perirhinal, parahippocampal cortex) and dorsomedial nucleus of the thalamus mediate explicit memory function (Mishkin 1978; Murray and Mishkin 1986; Squire and Zola-Morgan 1991; Zola-Morgan et al. 1989). The dorsolateral prefrontal cortex (also known as the principle sulcus or middle frontal gyrus) is involved in explicit recall as measured by working memory tasks (Goldman 1971; Goldman-Rakic 1988). Parietal cortex plays an important role in spatial memory and attention (Posner et al. 1988; Save et al. 1992). Memories are stored in the primary neocortical sensory and motor areas and later evoked in those same cortical areas (Damasio 1990; Zola-Morgan and Squire 1990). Visual information is stored in the occipital cortex, tactile information in the sensory cortex, auditory information in the middle temporal gyrus, and olfactory information in the orbitofrontal cortex. It has been hypothesized (Zola-Morgan and Squire 1990) that the role of the hippocampus is to bring together memory elements from di-

verse neocortical areas at the time of retrieval of explicit memory.

Implicit memory function is mediated by the amygdala, neocortex, and hippocampus. Conditioned fear responses are a type of implicit memory mediated by the amygdala, and these responses are measured with the acoustic startle response. The acoustic startle response is a primitive reflex that is part of an animal's response to threat. The startle response can be potentiated by the addition of something aversive, such as electric shock. The neuroanatomy and neurophysiology of emotional memory (measured by the conditioned fear response in animals) have been well characterized (Davis 1992). Lesions of the central nucleus of the amygdala have been shown to completely block fear-potentiated startle (Hitchcock and Davis 1986; Hitchcock et al. 1989), whereas electrical stimulation of the central nucleus increases acoustic startle (Rosen and Davis 1988). The amygdala integrates information that is necessary for the proper execution of the stress response, including (internal) emotion and information from the external environment (Turner and Herkenham 1991; Turner et al. 1980). The central nucleus of the amygdala projects to a variety of brain structures that are involved in effecting the stress response (Rosen et al. 1991). The hippocampus is involved in emotional memory for the context of a fear-inducing situation. Reintroduction to the context of the shock or the environment where the shock took place (i.e., the testing box), even in the absence of the shock, will result in conditioned fear responses. Lesions of the hippocampus interfere with acquisition of conditioned emotional responses to the environment where the shock was received (reviewed in Bremner et al. 1995a).

Effects of Stress on Memory Function

Evidence from a variety of studies shows a relationship between exposure to traumatic stress and deficits in explicit memory function. Concentration camp survivors from World War II have been found to have high rates of impairment in explicit memory function (Helweg-Larsen et al. 1952; Thygesen et al. 1970). Korean prisoners of war have been found to have an impairment on explicit

memory tasks of free verbal recall measured with the Logical component of the Wechsler Memory Scale—Revised (WMS-R; Russell 1975) in comparison with Korean veterans without a history of containment (Sutker et al. 1990, 1991). We have measured explicit memory function with the WMS-R Logical (verbal memory) and Figural (visual memory) components in Vietnam combat veterans with PTSD ($N = 26$) and control subjects matched for factors that could affect memory function ($N = 15$). PTSD patients had a significant decrease in free verbal recall (explicit memory) (Bremner et al. 1993b) as measured by the WMS-R Logical component, without deficits in IQ as measured by the Wechsler Adult Intelligence Scale—Revised (WAIS-R; Wechsler 1981). PTSD patients also had deficits in explicit recall as measured with the Selective Reminding Test (SRT; Hannay and Levin 1985for both verbal and visual components. We have subsequently found deficits in explicit memory tasks of free verbal recall measured by the WMS-R Logical component in adult survivors of childhood abuse seeking treatment for psychiatric disorders (Bremner et al 1995b). Studies have found deficits in explicit short-term memory as assessed with other measures in Vietnam combat veterans with PTSD in comparison with National Guard veterans without PTSD (Uddo et al. 1993) and in Vietnam combat veterans with PTSD in comparison with control subjects (Yehuda et al. 1995). Studies are currently in progress in female Vietnam combat nurses with PTSD (J. Wolfe, personal communication, October 1994). Deficits in academic performance also have been shown in Beirut adolescents with PTSD in comparison with Beirut adolescents without PTSD (P. Saigh, personal communication, August 1994). These studies suggest deficits in encoding on explicit memory tasks. Other studies in patients with PTSD have shown enhanced recall on explicit memory tasks for trauma-related words relative to neutral words in comparison with control subjects (Zeitlin and McNally 1991). In summary, the findings are consistent with deficits in encoding on explicit memory tasks and deficits in retrieval, as well as enhanced encoding or retrieval for specific trauma-related material.

Studies are needed to examine the effects of unconscious cognitive material (Kihlstrom 1987), also referred to as implicit memory

function (Kihlstrom 1987; Schacter 1995), on mental life in patients with PTSD. Alterations in implicit memory in PTSD patients include conditioned fear responding and alterations in priming effects in comparison with control subjects. PTSD patients have been shown in preliminary studies to have an enhancement of implicit recall (i.e., recall following priming) for trauma-related words relative to neutral words in comparison with control subjects (Zeitlin and McNally 1991). The Stroop Test (cited in Foa et al. 1991) is a measure of nonexplicit (i.e., not available for conscious recall) cognitive processes that has been used in the study of PTSD. This test involves presenting words in different colors and asking subjects to name the color. Delays in color naming can be interpreted as a measure of interference from unconscious cognitive processes. Vietnam combat veterans with PTSD have been found to take longer to color-name "PTSD" words, such as "body bag," than obsessive words, positive words, and neutral words (McNally et al. 1990, 1993), and this delay was correlated with severity of PTSD symptomatology as measured by the Mississippi Scale for Combat-Related Posttraumatic Stress Disorder (Keane et al. 1988). Stroop interference also has been shown in patients with PTSD related to the trauma of rape (Cassiday et al. 1992; Foa et al. 1991). The cognitive processes that are part of the performance of the Stroop Test are associated with activation of the cingulate cortex. These studies therefore make Stroop interference one of the more replicated findings in PTSD.

Patients with PTSD commonly report an increase in startle responsiveness (used in the measurement of conditioned fear responding). Increased startle magnitude has been found in Vietnam combat veterans with PTSD in comparison with control subjects for 90–100 dB noise (Butler et al. 1990; see also Paige et al. 1990). Other studies have shown no difference in trials to habituation of startle response (Ross et al. 1989). An increase in heart rate and skin conductance during the startle paradigm has been reported in patients with civilian PTSD in comparison with control subjects (Shalev et al. 1992). Conditioned fear responding also is measured clinically in PTSD patients with the psychophysiology paradigm. As reviewed in Prins et al. (1995), PTSD patients have

shown an increase in heart rate and blood pressure responsiveness to traumatic cues in the form of both trauma-related slides and scripts. Clinically, PTSD patients commonly become physiologically aroused when they encounter trauma-related cues in their environment, such as hearing a car backfire or smelling cut grass. These findings are consistent with increased conditioned responding in patients with PTSD.

Effects of Stress on Brain Regions Involved in Memory

Stress has long-term effects on brain regions involved in memory. Stress-induced alterations in these brain regions may underlie many symptoms of PTSD, such as pathological recall of traumatic memories and deficits in free recall (Bremner et al. 1995a). In addition, brain regions involved in memory include many regions described as part of the "limbic brain," which includes the hypothalamus, thalamus, hippocampus, amygdala, orbitofrontal cortex, and cingulate. Limbic brain regions have long been believed to be involved in emotionality, fear responses, and effecting the stress response. These brain regions are in turn affected by exposure to traumatic stress.

Studies of monkeys who died spontaneously following exposure to severe stress because of improper caging and overcrowding were found on autopsy to have damage to the CA2 and CA3 subfields of the hippocampus (Uno et al. 1989). Follow-up studies showed that hippocampal damage was associated with direct exposure of glucocorticoids to the hippocampus (Sapolsky et al. 1990). Studies in a variety of animal species have shown that direct glucocorticoid exposure results in a loss of pyramidal neurons and dendritic branching (McEwen et al. 1992; Packan and Sapolsky 1990; Sapolsky et al. 1985; Uno et al. 1989; Watanabe et al. 1992; Wooley et al. 1990) that is associated with deficits in memory (Luine et al. 1994).

We compared hippocampal volume measured with magnetic resonance imaging (MRI) in Vietnam combat veterans with PTSD

(N = 26) and healthy subjects (N = 22) matched for factors that could affect hippocampal volume, including age, sex, race, years of education, height, weight, handedness, and years of alcohol abuse. Patients with combat-related PTSD had an 8% decrease in right hippocampal volume in comparison with control subjects ($P < .05$) but no significant decrease in volume of comparison structures, including temporal lobe and caudate. Deficits in free verbal recall (explicit memory) as measured by the WMS-R (Wechsler 1987) Logical component, percent retention, were associated with decreased right hippocampal volume in the PTSD patients (r = 0.64; $P < .05$) but not in the control subjects. There was not a significant difference between PTSD patients and control subjects in left hippocampal volume or in volume of the comparison regions measured in this study, the left or right caudate and temporal lobe volume (minus hippocampus) (Bremner et al. 1995b). Recently, we found a statistically significant 12% reduction in left hippocampal volume in 17 adult survivors of childhood physical and sexual abuse in comparison with 17 control subjects who were matched on a case-by-case basis for age, sex, race, handedness, years of education and years of alcohol abuse (Bremner et al. 1997b).

The amygdala plays an important role in conditioned fear responses and exaggerated startle, which are prominent features of the clinical presentation of PTSD. We present here previously unpublished data from measurements of the amygdala in patients with combat-related PTSD (N = 19) and matched control subjects (N = 15). PTSD patients were Vietnam combat veterans in inpatient and outpatient treatment programs for PTSD. Healthy control subjects were free of psychiatric or medical disorders and were selected to be similar to the patients in several demographic factors. All of the subjects were part of a previously reported study of hippocampal volume in combat-related PTSD patients and control subjects (Bremner et al. 1995b). There was no difference between patients and control subjects for age (47.8 [3.6 SD] versus 46.3 [8.5 SD]), sex (all male), race (95% white and 5% black versus 80% white, 13% black, 7% Hispanic), years of education (12.9 [1.9 SD] versus 13.9 [3.2 SD]), handedness (79% right-handed versus 87% right-handed), years of alcohol abuse (9.9 [8.5 SD] versus 6.9 [10.3

SD]), height, and weight. Coronal MRI scans were obtained on a 1.5 Tesla scanner with TR = 25, TE = 5, and slice thickness 3 mm. Volume of the amygdala was determined by measuring the cross-sectional area of the amygdala in all coronal slices, including and anterior to the bifurcation of the basilar artery; summing the cross-sectional areas; and multiplying by the slice thickness. Interrater reliability for the amygdala was determined using the intraclass correlation coefficient with one-way analysis of variance (ANOVA) (with values of the coefficient approaching one, representing a high level of agreement between two raters) for volumetric assessments of amygdala with two raters (JDB and EV). Intraclass correlation coefficients (ICC) for interrater reliability (R) were as follows: left amygdala ICC = 0.56 (F = 3.56; df = 33,34; $P < .01$) and right amygdala ICC = 0.56 (F = 3.55; df = 33,34; $P < .01$). These results demonstrate excellent interrater reliability for the amygdala. We did not find a difference between patients and control subjects for the left amygdala (2,047 [470 SD] versus 2,018 [332 SD] mm^3) or right amygdala volume (1,992 [392 SD] versus 1,950 [425 SD] mm^3). Our findings are not consistent with a difference in amygdala volume in PTSD patients compared with control subjects. Of course, alterations in amygdala function in PTSD will not necessarily be reflected in a change in volume of the amygdala.

We also have used positron-emission tomography (PET) and [^{18}F]2-fluoro-2-deoxyglucose (FDG) in the measurement of cerebral glucose metabolic rate following administration of yohimbine and placebo in Vietnam combat veterans with PTSD (N = 10) and healthy control subjects (N = 10). We previously have found evidence for alterations in noradrenergic function—as demonstrated with increased PTSD symptoms, intrusive memories, flashbacks, and anxiety—following administration of the α_2 antagonist, yohimbine, which stimulates brain norepinephrine release, in PTSD patients in comparison with control subjects (Southwick et al. 1993). Animal studies have shown a decrease in metabolism in cerebral cortex following high levels of norepinephrine release. We found that administration of yohimbine resulted in a differential effect on brain metabolism in PTSD patients in comparison with control subjects in orbitofrontal, temporal, parietal, and pre-

frontal cortex, with PTSD patients showing a tendency to decrease brain metabolism, whereas control subjects showed a tendency to increase brain metabolism with yohimbine in comparison with placebo. PTSD patients also had a decrease in hippocampal metabolism, which was not seen in the control subjects (Bremner et al. 1997c). These findings are consistent with an increased release of norepinephrine in the brain following yohimbine in PTSD. Considering the role of norepinephrine in the hippocampus as a neuromodulator that affects memory encoding and retrieval, enhanced norepinephrine release in the hippocampus with stressors may be associated with the pathological recall that is typical of traumatic memories in patients with PTSD.

The studies previously summarized are consistent with alterations in brain regions involved in memory with associated deficits in memory function. It is also important to consider the effects on memory function of neurotransmitters and neuropeptides released during stress.

Modulation of Memory by Neurotransmitters and Neuropeptides Released During Stress

Brain chemicals released during stress, which are highly concentrated in brain regions involved in memory such as the hippocampus, amygdala, and prefrontal cortex, play a role in the modulation of memory function. These chemicals include norepinephrine, epinephrine, adrenocorticotropic hormone (ACTH), glucocorticoids, dopamine, acetylcholine, endogenous opiates, vasopressin, oxytocin, and gamma-aminobutyric acid (GABA) (for a review, see De Wied and Croiset 1991; McGaugh 1989, 1990). For instance, removal of the adrenal medulla, site of most of the body's epinephrine, results in an impairment in new learning, which is restored by administration of adequate amounts of epinephrine (Borrell et al. 1983). Posttraining administration of epinephrine after a learning task influences retention with an inverted U-shaped curve: retention is enhanced at moderate doses and impaired at high doses (Gold and van Buskirk 1975; Liang et al. 1986; McGaugh 1990). In

one recent study (Cahill et al. 1994), the α-adrenergic antagonist, propranolol, or placebo was administered to healthy human subjects 1 hour before a neutral or an emotionally arousing (stress-related) story. Propranolol, but not placebo, interfered with recall of the emotionally arousing story but not the neutral story, suggesting that activation of α-adrenergic receptors in the brain enhances the encoding of emotionally arousing memories (Cahill et al. 1994). We have found an increase in corticotropin-releasing factor (CRF), a neuropeptide that modulates memory function and plays an important role in the stress response in patients with PTSD (Bremner et al. 1997a). Vasopressin has been shown to facilitate traumatic recall in patients with PTSD (Pitman et al. 1993). It is hoped that extending preclinical findings about the effects of stress-related neuromodulators on memory function to clinical populations will enhance our understanding of memory alterations in PTSD.

Neural Mechanisms Mediating the Effects of Stress on Memory

The neural mechanisms of stress sensitization, fear conditioning, and extinction are useful in understanding the effects of stress on brain systems involved in memory. Stress sensitization refers to the phenomenon wherein repeated exposure to a stressor results in an amplification of responsiveness to subsequent stressors. For example, acute stress results in an increased release of norepinephrine in the hippocampus as well as other brain regions. In comparison with animals that have no history of exposure to stress, animals with a history of exposure to prior stressors become sensitized to exposure to subsequent stressors so that there is an accentuation of norepinephrine release in the hippocampus with a subsequent stressor. Norepinephrine (in addition to other neurotransmitters and neuropeptides) modulates memory formation and retrieval. This fact raises the possibility that stress sensitization, acting through neuromodulators, such as norepinephrine, may be associated with alterations in memory encoding and retrieval, which may have implications for understanding the mechanisms of trau-

matic recall in PTSD. Stress sensitization is clinically applicable to PTSD. Patients with PTSD have a difficult time with ordinary stressful events that healthy persons can tolerate without too much trouble. For instance, the stress of having a fender bender or having an argument with one's spouse can lead to a total decompensation in these patients. We have found that exposure to the stressor of childhood physical abuse increases the risk for the development of combat-related PTSD. Bremner et al. (1993a) suggest that sensitization resulting from early childhood stress may increase the vulnerability for the development of psychopathology in response to a subsequent stressor (i.e., combat stress in Vietnam).

We have reviewed the mechanism of fear conditioning, in which a pairing of a normally neutral stimulus with an aversive stimulus, such as electric shock, eventually results in fear responding to the light alone. Conditioned responding to cues in the environment that are reminiscent of the original trauma is a major source of disability for PTSD patients. Patients with PTSD have a heightened physiological responsiveness (increased heart rate and blood pressure) to reminders of the original trauma (e.g., combat films and sounds, scripts of traumatic events) relative to control subjects as measured with the psychophysiology paradigm. This increase in responsiveness resembles conditioned fear responding in animals.

Extinction refers to an inhibition of conditioned responses to cues associated with a fearful stimulus that takes place gradually over time following the removal of the original fearful stimulus. Extinction involves neocortical (Jarrell et al. 1987; LeDoux 1993) and orbitofrontal (Morgan and LeDoux 1994) inhibition of amygdala function. A failure of extinction is a prominent aspect of PTSD, which may explain why these patients continue to have conditioned responses to trauma-related cues for many years after exposure to the original trauma.

We have reviewed healthy memory function and how stress affects brain regions involved in memory and healthy memory function. In the following discussion, we review possible brain mechanisms for dissociation from the standpoint of the neurobiology of memory.

Brain Mechanisms in Dissociation

The neural basis for dissociative states may be found in the neurobiology of memory. The intimate relationship between dissociation and alterations in memory logically suggests that the biological basis for dissociative states may be found in brain structures that mediate memory function. Consistent with this idea, electrical stimulation of the hippocampus and adjacent cortex results in symptoms that are similar to dissociation (Halgren et al. 1978). As reviewed by Krystal et al. (Chapter 11, in this volume), administration of ketamine hydrochloride, a noncompetitive antagonist of the *N*-methyl-D-aspartate (NMDA) receptor, results in an increase in dissociative symptomatology, as measured with the CADSS (Bremner et al., in press), and in a disruption of delayed word recall in healthy subjects. The NMDA receptor, which is highly concentrated in the hippocampus, is involved in memory function at the molecular level through long-term potentiation (LTP). Subjects who took ketamine in the Krystal et al. (1994) study had a wide range of dissociative symptoms, including out-of-body experiences, feeling as if their arms were toothpicks, having gaps in time, feeling that time stood still, disturbances in the sense of self-identity, and derealization. Considering the role that the hippocampus plays in memory, dysfunction of the hippocampus may result in a breakdown of healthy integration of memory and consciousness. This breakdown may entail abnormalities of memory encoding, consolidation, or storage or some combination of the three. Dissociative states at the time of psychological trauma may represent a marker of pathological processes affecting brain structures involved in memory, such as the hippocampus. Traumatic events that are encoded when an individual is in a deficient state would be expected to be retrieved when the individual is in a similar deficient state. This theory may explain the phenomenon of how traumatic recall often occurs when the individual is in a dissociative state that is similar to the dissociative state that was experienced at the time of the original trauma.

Validity of Childhood Memories of Abuse: Relevance of Stress-Induced Changes in Brain Regions Involved in Memory

Recently, there has been considerable controversy concerning the validity of memories related to childhood abuse. The controversy is in part related to the vast increase in reported incidents of childhood abuse. Some authors have claimed that psychotherapists are suggesting to their patients abuse incidents that never occurred, using the term *false memory syndrome* to describe the alleged development of memories for abuse in suggestible individuals (Loftus et al. 1994a). As many as 38% of individuals who had been abused to an extent severe enough to require a visit to the emergency room (with full documentation that the abuse had indeed taken place) have been found to have no recall of the abuse up to 20 years later (Loftus et al. 1994b; Williams 1994a). A central issue in this debate is whether the lack of recall is because of "normal forgetting" (Loftus et al. 1994b) or memory-related mechanisms specific to psychological trauma, such as "repression" or, its synonymous appellation, dissociative amnesia (Williams 1994b). A mechanism such as amnesia could explain why individuals have a delayed recall of abuse for up to 20 years after the event.

Some authors have pointed to studies from cognitive psychology to support the claim that memory is subject to distortions over time and that this mechanism is operative in individuals who claim delayed recall of abuse. For example, in one of these studies, subjects viewed a series of slides that included a slide with a stop sign. After the slide presentation, the subjects received misleading verbal information that included reference to a yield sign. When given a test of recall of material from the slides, which involved a forced choice between a stop sign and a yield sign, subjects who received misleading verbal information performed more poorly than subjects who did not receive misleading information. The authors concluded from these results that misleading information can result in a modification, or "rewriting," of the original memory trace (Loftus and Loftus 1980). Other studies, however, have provided evidence that is not consistent with the idea that misleading

information can result in a modification of the original memory trace. In one study, slides that included a hammer were followed by misleading verbal information that made reference to a wrench. Subjects were then tested for recall of the slide material, but the forced choice test involved a hammer and a screwdriver (i.e., not the item from the misleading verbal information, which was a wrench). Subjects in this experiment who received misleading verbal information performed equally to subjects who did not receive misleading verbal information. The authors concluded that there is not convincing evidence for the idea that misleading information can result in a modification of the original memory trace (McCloskey and Zaragoza (1985a). McCloskey and Zaragoza (1985a, 1985b) suggested that other factors, such as forgetting the source of the original information, may be involved in the findings of E. F. Loftus and G. R. Loftus (1980).

These studies also involved memories of typical events and did not address whether memories of events such as abuse are subject to distortion. One then might wonder, Are memories for stress-related events similar to those experienced by victims of childhood abuse equally vulnerable? A study addressing this question focused on 5- and 7-year-old children's recall of the details of a doctor's examination. Half of the children received anal and genital examinations as part of a routine checkup, whereas the other half received a scoliosis examination (nongenital examination). There were no cases of free recall of genital touching in children who received the nongenital examination; with direct questioning, only one child incorrectly reported being genitally touched. Accuracy of recall was better for the genital examination than for the nongenital exam. Children were highly resistant to misleading questions regarding details of the examination (Saywitz et al. 1991). This study suggests that the types of events forming the basis of childhood abuse may not be as subject to distortions, insertions, and deletions as may be more "mundane" memories.

Alterations in neural mechanisms in memory with stress exposure may result in a difference in memory function in abused patients. Some authors have criticized the statement that memories of abuse could be unavailable to consciousness for many years,

only to return to active consciousness years after the fact. It should be considered that alterations in brain regions involved in memory could result in unusual manifestations of memory function, such as long-term amnesia. Certain emotional or physiological states, which are triggered in psychotherapy, also could lead to state-dependent recall, in effect cueing recall of abuse events for which there had been long-term amnesia. Future studies on the effects of stress on the neurobiology of memory should provide useful additional information to help clarify this issue.

Principles of Treatment for Alterations in Memory and Symptoms of Dissociation Related to Psychological Trauma

Understanding trauma, memory, and dissociation has the potential to be useful in the treatment of traumatized patients. Many authors have advocated a reintegration of splintered aspects of memory and experience in the treatment of traumatized patients (Kluft 1993). This type of therapy is often long term because many traumatized individuals have a weakening of the identity and personality structure, making it difficult for them to tolerate the intense affect that comes with the reintegration of traumatic memories (Fine 1991). Patients often have an increase in recall of traumatic events as they proceed through therapy. This recall of events may be upsetting, and sometimes, patients will become symptomatically worse. Clinical judgment is required to determine if the patients have a strong enough personality structure to tolerate the added anxiety associated with discussing traumatic memories. Sometimes, developing a long-term relationship with the therapist before discussing traumatic events in detail can be beneficial in these situations.

Considering the current controversy regarding issues such as false memory syndrome, how should the therapist treat patients who are experiencing a delayed recall of traumatic events? We have reviewed questions regarding the effects of misleading and suggestive information on memory recall. It is clear that a therapist should not push patients to remember abuse events that the thera-

pist feels may exist. On the other hand, studies in children have clearly shown that if one does not ask specific questions, chances are the patients will not volunteer information about abuse events, probably because of shame. Therefore, the psychiatric interview and history, with specific inquiries about abuse experiences, should not be abandoned. On the other hand, the therapist should exercise great caution not to suggest to patients events that may not have happened.

Conclusion

Traumatic stress results in a variety of symptom outcomes, as well as long-term effects on brain systems involved in memory. In this chapter, we have argued for a comprehensive approach to the effects of stress on the individual, emphasizing the fact that psychological trauma is associated with increased symptoms of PTSD, as well as dissociation, depression, and other outcomes such as alcohol and substance abuse. The variety of possible outcomes of psychological trauma may explain why there is considerable overlap in the symptomatology of these different disorders. Alterations in brain regions involved in memory may provide a link between dissociation and other symptoms related to traumatic stress. Stress results in long-term changes in these brain regions, and there is evidence that dysfunction of these brain regions may mediate symptoms of dissociation as well as symptoms of PTSD. A biological approach to stress and memory can provide some useful insights into the current controversy surrounding false memory syndrome. Understanding the effects of stress on memory, and the relationship between alterations in memory and dissociation, is also useful in planning treatment approaches for traumatized patients.

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