THE NATURE OF TRAUMATIC MEMORIES OF CHILDHOOD ABUSE

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ABSTRACT

In recent years, the explosion of reports of childhood abuse has raised questions about the nature of memory for traumatic events. In particular, there has been heated debate concerning amnesia for childhood abuse and the validity and accuracy of recovered memories. This discussion reviews the psychobiology, cognitive research, clinical research, and clinical practice concerning traumatic memory. From experimental investigations and clinical observations, there is ample evidence to support the existence of a variety of memory processes for traumatic events, both conscious and nonconscious. Experimental studies have also shown that memory content is dynamic and subject to suggestion and distortion. There is considerable evidence that traumatic memory may be associated with psychobiologic features and cognitive characteristics that are quite different from ordinary memory. The characteristics of traumatic memory are quite varied and are dependent on the nature of the traumatic events and the age when they were experienced. Clinical evidence and some studies suggest that brief or limited traumatization results in increased clarity or recall (hypermnesia), and a high level of accuracy concerning the central details of the experience. On the other hand, severe and chronic early traumatization may be correlated with denial, dissociation, and amnesia. Clinical studies have supported the existence of amnesia and recovered memories especially for severe and chronic childhood abuse. However, such memories also may be most vulnerable to distortion and errors in recall. Mental health professionals who treat survivors of childhood abuse should be well informed about the complexity of traumatic memory in order to provide a sound and balanced approach to their clinical work.

INTRODUCTION

In the United States in 1990, 2.7 million cases of suspected child maltreatment were reported to child protective agencies (National Research Council,1993). In nearly half of the cases, child maltreatment was substantiated. Our clinical experience suggests that these figures represent a substantial underestimate of prevalence; intrafamilial abuse often occurs in great secrecy and often goes unreported to child protective agencies. Leaving aside the question about the accuracy of reporting, the abuse of children is a very serious problem for our society. While not all, and perhaps even not most, of these abused children will develop serious psychiatric difficulties, a substantial number is likely to suffer lasting effects.

In the past decade, we have begun to understand some of the effects of childhood abuse. Clinical investigations have suggested that early abuse may be associated with numerous psychological and psychiatric difficulties including depression, anxiety, emotional lability, impaired self-esteem, social withdrawal, self-destructive behavior, impaired characterologic development, alcohol and drug abuse, eating disorders, somatization, and various physiologic changes (Barksy, Wool, Barnett, & Cleary, 1994; Bryer, Nelson, Miller, & Krol, 1987; Courtois,1979; Finkelhor, 1984; Gelinas, 1983; Goldman, D'Angelo, Demaso, & Mezzacappa, 1992; Hall, Tice, Beresford, Wooley, & Hall, 1986; Herman, 1981; Herman, Perry, & van der Kolk, 1989; Herman, Russell, & Trocki, 1986; Herman & van der Kolk, 1987; Loewenstein, 1990; Ludolph et al., 1990; Morrison, 1989; National Victim Center, 1992; Ogata et al., 1990; Pribor & Dinwiddie, 1992; Pribor, Yutzy, Dean, & Wetzel, 1993; Russell, 1986; Shapiro, 1987; Summit, 1983; Swanson & Biaggo, 1985; van der Kolk, 1987; van der Kolk, Perry, & Herman, 1991; Welch & Fairburn, 1994; Zanarini, Gunderson, & Marino, 1987). While many of these associated difficulties are based on clinical observations and preliminary findings, there is clearly considerable psychiatric morbidity associated with early traumatization.

In recent years, clinicians and investigators have focused on post-traumatic and dissociative symptoms that appear to be etiologically related to childhood traumatization (Bernstein & Putnam, 1986; Braun, 1990; Chu, 1991; Chu & Dill, 1990; Coons, Cole, Pellow, & Milstein, 1990; Donaldson & Gardner, 1985; Kirby, Chu, & Dill, 1993; Putnam, 1985; 1993; Putnam, Guroff, Silberman, Barban, & Post, 1986; Ross, Anderson, Fleisher, & Norton, 1991; Saxe et al., 1993; Ulman & Brothers, 1988; van der Kolk & Kadish,

1987). Dissociative symptoms including depersonalization, derealization, psychogenic amnesia, fragmentation of identity, and post-traumatic reexperiencing phenomena (e.g., "flashbacks" of traumatic events), are frequent clinical findings in disorders that derive from childhood abuse.

Dissociative amnesia is at the core of the controversy concerning traumatic memory of childhood abuse. The idea that overwhelming experiences can be forgotten and then later recalled has its origins in major schools of thinking concerning intrapsychic experience. Early in this century, Janet (1907) described psychogenic amnesia and theorized that traumatic events could be dissociated from conscious awareness, only to be remembered at some later point in time. Freud's psychoanalytic theory included the belief that events that were traumatic or that resulted in intense intrapsychic conflict could be repressed and become unconscious, and could later be repeated and reexperienced (Freud, 1955). Freud described repression as an active process, itself unconscious, that holds unacceptable or overwhelming thoughts and impulses outside of conscious awareness. These theoretical constructs laid the groundwork for modern investigators who were interested in the effects of traumatic events on memory.

Psychogenic amnesia has been observed for overwhelming and traumatic experience. For example, a recent media report (National Public Radio Morning Edition, 1996) documented the story of Ross Cheit, an associate professor of political science at Brown University. In 1992, Professor Cheit learned that his nephew was joining a boys' chorus. He became quite depressed, and could think of no reason for his depression. However, he soon had the abrupt recovery of memories of childhood sexual molestation by an administrator of a boys' chorus in a summer camp. Professor Cheit set out to corroborate his memory. He found other men who had been molested at the same camp as boys, and found two former employees of the camp had witnessed some episodes of sexual molestation. He subsequently found the perpetrator, who admitted to the acts. He filed civil suit against the organizers of the boys' chorus; the case was settled in 1994. Professor Cheit was not in psychotherapy at the time he recovered these memories.

Perhaps even more striking are the many adult psychiatric patients who report extensive amnesia for childhood events. To some patients, much of their childhood is foggy. Others can recount the events of their past, but feel as though these happened to someone else. Many patients describe a kind of "Swiss cheese" memory with gaps in their recall of their early experiences, or even complete amnesia for any events over months or years. In clinical settings, this extensive psychogenic amnesia may be highly significant, and many patients with this kind of amnesia eventually report recovered memories of extensive childhood abuse.

Many clinicians and investigators accept recovered memories of childhood abuse as essentially valid reports of repressed or dissociated early experiences. For some patients, clinical work with recovered memories has been a vehicle for the resolution of psychiatric symptomatology, and has led to an enhanced understanding of the traumatic etiologies of their difficulties. Recently, however, a number of prominent investigators have questioned the validity of recovered memory and have argued that such memories can be false and that many clinicians may be colluding in the creation of pseudomemories. A heated debate has emerged regarding therapists' role in the retrieval of dissociated memories of childhood abuse. In order to provide an integrated perspective concerning memories of childhood abuse, this discussion reviews recent findings concerning the psychobiology of traumatic memory, recent progress in cognitive memory research, issues concerning memory distortion, clinical research, and clinical practice concerning traumatic memory. Despite the difficulties of trying to integrate the findings of several major schools of scientific endeavor, it is crucial to understand the complexity of traumatic memory in order to provide a balanced approach to clinical work with patients who have memories of childhood abuse.

THE PSYCHOBIOLOGY OF TRAUMATIC MEMORY

Clinical experience has suggested that traumatic experiences are often remembered differently than ordinary experience. Indeed, this observation is the basis for the symptom complex of post-traumatic stress disorder (PTSD). The central features of PTSD are hyperarousal, hyperactivity, and repeated reexperiencing of the traumatic events through vivid memories, flashbacks, affective flooding, or nightmares (American Psychiatric Association, 1994). These symptoms typically coexist or alternate with affective numbing, avoidance, and amnesias. Each of these phenomena can be usefully examined as reflections of altered or pathological memory function.

The psychobiology of PTSD has been reviewed extensively (Charney, Deutch, Krystal, Southwick, & Davis, 1993; Krystal et al., 1989; van der Kolk, 1994; van der Kolk & Saporta, 1991; van der Kolk & van der Hart, 1991). Many investigators propose that the symptoms of PTSD result from the multifaceted neurohormonal changes that occur in response to acute and chronic stress, and that memory for traumatic events may be processed and stored differently from ordinary memory. It should be noted, however, that although many physiologic changes have been observed in PTSD patients, many of the stress-responsive neurohormonal changes (especially in the brain) have been observed only in animal models. Thus, applications of these models to humans with PTSD remains speculative.

At least three stress-responsive neurohormonal systems have emerged as critical in the development of PTSD: 1) catecholamines, including adrenaline, noradrenaline, and dopamine, that modulate bodily activation and arousal par-

ticularly in stressful emergency situations; 2) hormones of the hypothalamic-pituitary-adrenal (HPA) axis, including corticotrophin-releasing factor (CRF), adrenal corticotrophic hormone (ACTH), and glucocorticoids, that have an essential role in maintaining physiological and psychological homeostasis; and 3) endogenous opioids, opiate-like substances produced by the body in response to stress. Persistent increases in catecholamine activity, alterations in hormonal functions in the HPA axis, and opioid responses have all been documented in patients with PTSD (Charney et al., 1993; McFall, Murburg, Roszell, & Veith, 1989; Pitman, van der Kolk, Orr, & Greenberg, 1990; Southwick et al., 1993; van der Kolk, 1994; Yehuda, Giller, Southwick, Lowy, & Mason, 1991). As discussed below, each of these systems also has complex effects on memory function that may be fundamental to the memory disturbances seen in post-traumatic syndromes.

Patients with PTSD exhibit multiple symptoms of heightened arousal, including an exaggerated startle response, increased response to stress-related stimuli, panic attacks, and hypervigilance (American Psychiatric Association, 1994; Charney et al., 1993; van der Kolk, 1994). The early recognition of these persistent physiologic changes led Kardiner to characterize PTSD (then called a "war neurosis") as a "physioneurosis" (Kardiner, 1941). Autonomic reactivity has been quantitatively studied in combat veterans with PTSD (Orr, 1994; Pitman, Orr, Forgue, Altman, de Jong, & Herz, 1990; Pitman et al., 1987). When patients with PTSD are exposed to trauma-related stimuli in a controlled laboratory setting, they exhibit marked psychophysiological arousal, such as elevated heart rate, increased galvanic skin response, and increased tone in facial musculature. Recently these observations have been extended to victims of traumatic events not related to combat trauma (Shalev, Orr, & Pitman, 1993; Blanchard, Hickling, & Taylor, 1991).

Many investigators have suggested that the psychophysiological arousal found in PTSD is mediated by chronic elevations in both central and peripheral catecholamine function (see Charney et al., 1993 and Southwick et al., 1993, for reviews). In support of this idea, it has been shown that acute stress in animals is associated with increased activity of neurons in the locus ceruleus, the midbrain nucleus which is the source of widespread adrenergic projections (Simson & Weiss, 1988), and is also associated with increased turnover of noradrenalin in the locus ceruleus, hypothalamus, amygdala, hippocampus, and neocortex (Tsuda & Tanaka, 1985). Heightened central adrenergic reactivity may make patients with PTSD more vulnerable to extreme anxiety reactions and flashbacks. For example, compared to control subjects, PTSD patients have greater occurrence of both panic attacks and flashbacks in response to lactate challenge (Rainey et al., 1987), or yohimbine (Southwick, et al., 1993), an alpha-adrenergic antagonist that stimulates central adrenergic release.

Acute stress is associated with similar increases in activity of midbrain dopaminergic neurons, and increased release and metabolism of dopamine, particularly in the prefrontal cortex (see Charney, et al., 1993). The prefrontal dopaminergic system may be significant for focused attention and vigilance, and the increased dopaminergic function may be a factor in the hypervigilance of PTSD. The prefrontal cortex is also postulated to mediate memory search and retrieval via its extensive connections to the hippocampal system (Moscovitch, 1992). Abnormality in this system may contribute to experiences of intrusive remembering such as flashbacks, failures of remembering such as amnesia, or misattribution of the source of the memory.

Hormonal activity in the HPA axis is highly responsive to acute stress, producing an immediate increase in CRF, ACTH, and glucocorticoids (Sapolsky, Krey, & McEwen, 1986). CRF not only stimulates release of ACTH from the pituitary, but also acts as a neurotransmitter itself, enhancing activity in locus ceruleus adrenergic neurons (Axelrod & Reisine, 1984; Yehuda et al., 1991). Glucocorticoid receptors are particularly abundant in the hippocampus, a brain structure thought to be central in memory function. Glucocorticoids receptor density in the hippocampus is decreased by both acute stress and circulating glucocorticoid hormones (Sapolsky, Krey, & McEwen, 1986). Finally, there is considerable evidence for glucocorticoid toxicity to the hippocampus, resulting in eventual neuronal cell death in a dose and time dependent manner. Hippocampal cell death occurs in aging, and is accelerated by stress. Hippocampal neuronal degeneration has been demonstrated in monkeys after either prolonged stress or prolonged administration of glucocorticoids (Sapolsky, 1986; Sapolsky, Uno, Rebert, & Finch, 1990), suggesting a possible mechanism for some of the changes in memory function seen in PTSD. Reduced hippocampal volume (as measured by magnetic resonance imaging) and memory deficits have been demonstrated in veterans with combat-related PTSD (Bremner et al., 1995) and in patients with PTSD associated with childhood physical and sexual abuse (Bremner et al., in press).

Uncontrollable stress results in stress-induced analgesia in both animal model systems and in humans that is attributed to the release of endogenous opioids (Hemingway & Reigle, 1987; Willer, Dehen, & Cambier, 1981). Furthermore, opioid release can become a conditioned response (Fanselow, 1986) in a "fear conditioning" paradigm as described below. For example, Pitman et al. (1990) have shown that Vietnam veterans with PTSD develop significant analgesia when exposed to combat-related stimuli, and that this effect is blocked by the opiate antagonist, naloxone. Endogenous opioids are also known to have inhibitory action in the amygdala (McGaugh, 1989 & 1992), a brain area believed to be central in the emotional evaluation of information (Davis, 1992a). In addition, opiates interfere with early memory consolidation via the inhibition of the amyg-

daloid projections to the hippocampus (McGaugh, 1989 & 1992).

The long-term physiologic changes associated with severe stress may be understood broadly as a form of non-conscious or conditioned somatic memory, and may be the basis of certain forms of non-conscious memory of traumatic events. A common paradigm for this is the concept of fear conditioning (Charney, et al., 1993; Davis, 1992a & b, 1992; Kolb, 1987; LeDoux, Cicchetti, Xagoraris, & Romanski, 1990), a classical conditioned response in which a fear response is coupled to an otherwise neutral auditory or visual stimulus. Fear conditioning may be a model to understand the clinical phenomena of affective "flashbacks," panic, and physiologic arousal that PTSD patients experience when exposed to a stimulus reminiscent of the trauma. The neurobiologic basis of fear conditioning has been studied in several species including primates. Conditioning is mediated by subcortical thalamoamygdaloid projections (Davis 1992a; LeDoux et al., 1990), and seems to involve noradrenergic, dopaminergic, opiate, and CRF systems (Davis 1992a & b).

At a cellular level it is believed that all learning, including fear conditioning, may involve the phenomenon of long-term potentiation. Neurophysiologists have demonstrated that short bursts of intense electrical stimulation can induce long-term potentiation of neuronal synaptic function (i.e., an enduring increase in the post-synaptic response to stimulus) particularly in the hippocampus and amygdala (Bliss & Lomo, 1973; Chapman, Kairiss, Kiemanm, & Brown, 1990; Swanson, Teyler, & Thompson, 1982). NMDA glutamate receptors, which are abundant in both the hippocampus and amygdala, have been postulated as the neurochemical mediators for long-term potentiation. The development of fear conditioning is inhibited by blockade of the NMDA glutamate receptors in the amygdala (Miserendino, Sananes, Melia, & Davis, 1990), suggesting that fear conditioning and other strong affective association in traumatic memory may depend on long-term potentiation in the amygdala.

Conditioned responses are subject to gradual extinction if they are not reinforced by continued presentation of the aversive stimulus. Charney et al. (1993) have suggested that trauma-induced conditioned responses may persist due to abnormal extinction. Furthermore, it is believed that extinction involves suppression or masking of the conditioned association rather than "erasure." Extinguished responses are easily recovered to full strength by a single exposure to the aversive stimulus even after long delays (Charney et al., 1993). This phenomenon may explain why PTSD symptoms can be activated by a specific stimulus reminiscent of the trauma even after years of quiescence.

Traumatic experiences result in profound alterations of memory, ranging from the enhanced and repetitive memory seen in flashbacks to amnesia for the traumatic events. How does the traumatic experience alter memory function? In theory, traumatic experiences could alter memory function at any of a number of stages: attention and registering of the events, formation of the initial memory trace in short-term memory, association to other memories leading to interpretation of the experience, conversion to stable memory, long-term memory consolidation, integration of the traumatic experience with other personal history, or memory access or retrieval. Traumatic experience may be associated with moderate emotional arousal that may heighten and focus attention, or may involve more overwhelming stimulation that may induce cognitive disorganization and impaired attention. Fragmented perception and attention may ultimately contribute to incomplete or incoherent memory. In addition, the mechanisms by which individuals maintain attentional focus involve seeking salience in the perceptual world (Crick & Koch, 1990). As a result, perceptual input may be biased towards details which are emotionally significant, threatening, novel, or otherwise central to the individual. Thus, the raw material of memory is intrinsically selective, and highly emotional states may further alter attention, perception, and subsequent memory.

MacGaugh and others have performed extensive studies in animals, examining the effects of neuromodulators including catecholamines, acetylcholine, GABA, and opioids, on retention of newly learned material (McGaugh, 1989 & 1992). Exposure to these endogenous hormones immediately post-learning affects learning retention. These effects are both dose- and time-dependent for all hormones studied. For example, low to moderate levels of epinephrine enhance retention of newly learned material, while higher doses interfere with retention. In contrast, low doses of opioids suppress retention and attenuate the enhancement effects of epinephrine (McGaugh, Introini, & Castellano, 1993). These effects are localized to the amygdaloid complex (McGaugh et al., 1990; McGaugh, Introini-Collison, Cahill et al., 1993). Output from the amygdala to the hippocampus and the neocortex contributes affective significance and personal meaning to experience and memory, and modulates the encoding of stable memory from short-term memory (McGaugh, Introini-Collison, Nagahara, & Cahill, 1990). Stress-related mechanisms may account for "over-consolidation" of memory contributing to the formation of deeply ingrained traumatic memory with subsequent intrusive remembering and conditioned recall (McGaugh, Introini-Collison, Cahill et al., 1993; Pitman, 1989). Conversely, extreme stress may interfere with memory consolidation in the hippocampus, resulting in amnesia or dissociation of perceptual experience from explicit memory formation. These findings in animals suggest that traumatic memory may be quite different from ordinary memory.

A considerable body of research points to state-dependence in learning, memory, and recall (van der Kolk, 1994; Eich & Metcalfe, 1989; Tobias, Kihlstrom, & Schacter, 1992). Findings from research in state-dependent learning suggest that when in one emotional and physiologic state, it is more

difficult to access memories and experiences of a different state. Since the traumatic experience induces a marked physiological arousal and altered neurohormonal state, it is possible that both the encoding and recall of memory is specific to this state. This may contribute to the phenomena of amnesia and dissociated memory in PTSD, since when not physiologically aroused, it may be more difficult to access the traumatic state. Conversely, when presented with triggers reminiscent of the trauma, access to the traumatic state may be facilitated, leading to flashbacks and other reexperiencing phenomena (Rainey et al., 1987; Southwick et al., 1993; van der Kolk & van der Hart, 1991).

Finally, based on clinical observation, some investigators (Crabtree, 1992; Kolb, 1987; van der Kolk & Ducey, 1989; van der Kolk & van der Hart, 1991) have suggested that traumatic memories are stored segregated from ordinary narrative memory, and are less subject to ongoing modification in response to new experiences. In contrast to narrative memories that are integrative, malleable, and fitted into the individual's personal cognitive schemas, traumatic memories are inflexible, non-narrative, automatic, triggered, and disconnected from ordinary experience. They suggest that this non-integration is the basis for dissociated remembering through behavioral reenactment, somatic sensation, or intrusive images which are disconnected from explicit memory of events. Because the memories are unassimilated, they retain their original force – "unremembered and therefore unforgettable" (van der Kolk & Ducey, 1989). Ordinary narrative memory is dynamic and both changes and degrades over time. In contrast, traumatic memory may be less changeable and has been described as "indelible" (LeDoux, 1992).

THE CONTRIBUTIONS OF MEMORY RESEARCH

Research in the field of cognitive psychology has paralleled psychobiologic investigations of memory. In particular, research concerning the types of memory and the malleability of memory has been useful in elucidating the particular nature of traumatic memory. Much of the experimental research and the more recent debate about traumatic memory has concerned concepts of unconscious memory. It should be noted in comparing experimental findings to theories about trauma, that the term "unconscious" has been used to describe both nonconscious memory processes demonstrated by laboratory investigations and the psychodynamic unconscious as described by Freud. Both uses of the term imply nonconscious mental processes, outside of conscious awareness, that may exert influence on behavior, thought, or experience. The psychoanalytic use of the term unconscious has additional implications, i.e., memory that is suppressed by the active process of repression.

Types of Memory

The study of memory has produced descriptions of several forms of memory. As described by Polster, Nadel, & Schacter (1991), psychological and medical researchers of the 19th century agreed that memory was not a unitary entity, but more likely a multidimensional function consisting of various domains and various representations of recollections. For example, in the late 19th and early 20th centuries, French philosopher Maine de Biran suggested three memory types: 1) the accumulation of motor habits or mechanical memory, 2) memory that represented facts and events, and 3) memory for emotions and feelings (Polster et al., 1991).

Much of the study of different memory processes has derived from the study of patients with amnesia. In 1911, Claparède (in Rapaport, 1951) reported his observations of a woman with Korsakoff's syndrome who consistently had no memory of him within minutes of numerous introductions. Finally, upon one introduction, he shook her hand with a pin hidden in his own and pricked her. Later, she still had no memory of him, but refused to shake his hand eventually admitting that pins are sometimes hidden in the hands of others. She did not consciously remember Claparède, but had some form of memory of the pain caused by the pin-handshake. In the modern era of memory research, neuropsychological studies on patients with amnesia have repeatedly shown that these patients could acquire various skills (e.g., in cognitive and perceptual tasks) in spite of having no memory of the episodes during which the skills were learned (Cohen & Squire, 1980; Daum, Channon, & Canavar, 1989; Jacoby, Lindsay, & Toth, 1992; Jacoby & Witherspoon, 1982; Sartori, Masterson, & Job, 1987; Warrington & Weiskrantz, 1982). Subsequent research demonstrated similar nonconscious learning and memory in normal subjects (Graf, Mandler, & Haden, 1982; Jacoby, Lindsay, & Toth, 1992; Jacoby & Witherspoon, 1982; Tulving, Schacter, & Stark, 1982).

A diverse terminology has been used to describe what appears to be two separate and distinct memory systems. Tulving (1972 & 1983) proposed that memory occurs in two primary modes: episodic and semantic. Episodic memory included autobiographical information about past experiences. Semantic memory referred to a general fund of knowledge not necessarily associated with specific past experiences. Subsequent investigators described two memory systems labeled declarative and procedural (Kihlstrom & Hoyt, 1990). Declarative memory, similar to episodic, referred to memory for factual knowledge of one's material world and social experiences. Procedural memory, like semantic, referred to the memory of abilities, rules, and schematic approaches to dealing with the world. Schacter ultimately coined the currently accepted descriptive distinctions of two primary memory systems: explicit and implicit (Graf & Schacter, 1985; Schacter, 1985, 1987, 1990 & 1992a). Explicit

memory consists of the collection of prior experiences within "intentional or conscious" recall and includes declarative and episodic memories. Implicit memory refers to changes in performance or behavior based on prior experiences of which one has no conscious recall. Implicit memory subsumes the previous concepts of semantic and procedural memories, as well as conditioned responses and cognitive priming (see below). Implicit memory may also be the active process in affective and somatic memory (Erdelyi, 1990), which supports the clinical observation in PTSD patients that these kinds of memory may be experienced without conscious awareness of their origins (Chu, 1991; Braun, 1988).

Considerable research has provided evidence that these explicit and implicit forms of memory can be dissociated from each other (Bower, 1990; Kihlstrom & Hoyt, 1990). Schacter (1987 & 1990) has cautioned that the distinction between explicit and implicit memories is descriptive rather than proof of anatomically separate systems. However, current work on the anatomy of memory supports the speculation that separate anatomical systems may be involved in explicit and implicit memory. Studies of animals with precise lesions have confirmed that the hippocampus and closely related medial temporal lobe structures are of central importance in the formation and consolidation of explicit memory (Schacter, 1992b; Squire, 1986; Squire, 1992; Squire & Zola-Morgan, 1991; Thompson, 1986). In contrast to explicit memory, implicit memory is unaffected by lesions of the medial temporal lobe. Implicit memories seem to be stored in diverse neocortical and subcortical systems. For example, motor conditioning depends on modification of neuronal connections in the cerebellum (Thompson, 1986), and learning of skills and habits may be reflected in change in the neostriatum (Heindel, Salmon, Shults, Walicke, & Butters, 1989). Emotional conditioning appears to be localized in the amygdala (Davis, 1992; LeDoux et al., 1990).

Research on "priming," a type of implicit memory, has shed further light on the nature of conscious and nonconscious mental processes. Priming refers to facilitation of a simple cognitive task (e.g., object recognition or word completion) as a result of a prior encounter with the cue, and independent of conscious recollection of that encounter (Schacter, 1992a). Research conducted on priming effects has produced evidence of a group of modality-specific memory subsystems that function presemantically and outside of conscious awareness (Schacter, 1992a & b). For example, some of these subsystems are involved in the acquisition of the form and structure of words and objects, but not their meaning and associative qualities. That is, they operate independently of both the need to access meanings of words or objects, and of conscious memory of prior experiences. As with other types of implicit memory, priming subsystems are expressed by neurocognitive systems that are distinct both functionally and anatomically from the neurocognitive structures that support explicit memory (Frackowiak, 1994; Sartori, Masterson, & Job, 1987; Schwartz, Saffran, & Marin, 1980; Squire, 1992). For example, neuroimaging studies with positron emission tomography (PET) scans suggest that the visual perceptual representation subsystem is located primarily in an area of the extrastriate occipital cortex (Zeti et al., 1991), whereas semantic language processing localizes to the temporal and parietal cortex (Frackowiak, 1994).

The implications of research concerning the psychobiology of memory and types of memory are indeed significant, demonstrating the existence of nonconscious mental processes, and contributing to an understanding of the nature of memory. Memory and memory processing is highly complex, with multiple memory systems involved in the acquisition, retention, and recall of information, and these systems are represented in various locations in the brain and function through a variety of neurocognitive networks.

The Malleability of Memory

In 1977, Brown and Kulick described so-called "flashbulb" memory as a process in which precise details of a traumatic scene are permanently etched in memory in a potentially retrievable manner. In contrast, more recent studies of memory suggest that such memories can be remarkably inaccurate, particularly concerning peripheral details of the experience. Despite the difficulties of simulating traumatic experiences in the laboratory, investigations have studied the nature of memory in stressful experiences. Experimental procedures have included testing college students under demanding conditions (Eriksen, 1952 & 1953; Tudor & Holmes, 1973), and exposing subjects to shocking photographic material (Christianson & Loftus, 1987; Kramer, Buckout, Fox, Widman, & Tusche, 1991). Data from these investigations show that participants are often remarkably inaccurate in recounting details of their experience (e.g., who made critical statements to whom, where they were at the time, what other people looked like, what time of day it was, etc.) (Christianson & Loftus, 1987 & 1991; Holmes, 1990; Kramer et al., 1991). However, details of the experiences that are central to the individual seem to be accurately retained. A number of investigators (e.g., Chistianson and Loftus [1991], and Holmes [1990]) suggest that an individual's recall of central detail is retained as a direct function of the intensity of the affect associated with the experience: the more emotional the experience, the better selected central details are remembered.

The role of suggestion in the malleability of memory has also been well established in laboratory studies (Loftus, 1979; Loftus, Korf, & Schooler, 1989; Schooler, Gerhard, & Loftus, 1986; Schumaker, 1991). In some protocols, participants are shown pictures, slides, or videotape of an event and are then asked to recall the event. When participants are given cues or suggestions regarding the event, they often make errors concerning peripheral details of the events. For example, in one experiment (Schooler, Gerhard, & Loftus, 1986), par-

ticipants were shown slides of an automobile accident. For one set of participants, the slides contained a yield sign. For another set of participants, the slides did not contain a yield sign, but its existence was suggested in a questionnaire about the slides. When asked, many participants in both groups reported seeing the sign and could provide a verbal description of the sign. These studies are important because they demonstrate that memory is not a static phenomenon, but can be influenced by a variety of factors including the retrieval context.

Actual traumatic events can be misremembered. Studies of memory of the space shuttle *Challenger* explosion showed that many participants had many incorrect recollections of the events (Neisser & Harsch, 1992; Warren & Swartwood, 1992). Moreover, participants' certainty that they were accurate in recall was not related to actual accuracy (Warren & Swartwood, 1992). For example, Neisser and Harsch (1992) describe college students who were very confident about their recall of the *Challenger* explosion, but who made many errors about when the event occurred, where they were at the time, and how they heard about it. Loftus describes other situations where serious life-and-death situations such as war and sports accidents were misremembered (Loftus, 1993). However, once again, the central features of the events (e.g., the fact of the *Challenger* explosion, the bombing of Pearl Harbor, and sports accidents) were accurately retained.

Despite evidence that memory content can be influenced by suggestion, emotional arousal, and personal meaning, the bulk of memory research actually supports the accuracy of remembered events that are known to have occurred. However, there is also evidence that persons can have memory for events that did not occur. One well-known personal pseudo-memory was described by the Swiss psychologist, Piaget (1962), the well-known theorist of cognitive development in childhood. For many years during his childhood, Piaget had clear visual memory of someone trying to kidnap him from his pram when he was two. The memory also involved his nanny chasing away the potential kidnapper and then going home and telling the family. Years later, when Piaget was 15, the nanny returned to the Piaget family and confessed that the incident had never occurred. Her motive had been to enhance her position in the household, but she subsequently had suffered guilt about the fabrication and about the watch she had received as a reward.

Piaget's experience suggests that after being told about certain past events by a trusted individual or someone in a position of authority, persons may create pseudo-memories of events that never actually occurred. The "memories" may seem valid, and persons may not recall the true source of the information (so-called "source amnesia"). An experiment by Hyman, Husband, and Billings (1995) attempted to implant false memories of childhood events in college students. Participants were asked to describe both real events (from information supplied by parents) and false events. Over three interviews, approximately 6% of participants developed vivid pseudo-memories of false events as in the following description (p. 191):

First Interview

I: The next one is attending a wedding. At age six you attended a wedding reception and while you were running around with some other kids you bumped into a table and turned over a punch bowl on a parent of the bride.

S: I have no clue. I have never heard that one before. Age six?

I: Uh-huh.

S: No clue.

I: Can you think of any details?

S: Six years old, we would have been in Spokane, um, not at all.

Second Interview

I: The next one was when you were six years old and you were attending a wedding.

S: The wedding was my best friend in Spokane, T___. Her brother, older brother was getting married, and it was over here in P___, Washington, because that's where her family was from and it was in the summer or spring because it was really hot outside and it was right on the water. It was an outdoor wedding and I think we were running around and knocked over like the punch bowl or something and um made a big mess and of course got yelled at for it. But uh.

I: Do you remember anything else?

S: No.

I: OK.

In a similar subsequent experiment, Hyman and Pentland (1996) found that vivid pseudo-memories increased from 9% to 25% if participants were asked to imagine the false events in detail. These studies support the contention that pseudo-memories can be induced, particularly with repeated suggestion, rehearsal, and the use of imagery. It should be noted, however, that only a minority of partici-

pants responded to cues to remember false events, suggesting that certain individuals may have more vulnerability than others to creating pseudo-memories.

The now well-known case of Paul Ingram has added evidence to the notion of the creation of pseudo-memory. As described by Ofshe (1992) and Loftus (1993), Ingram, the chair of his county Republican party committee, was accused of the sexual abuse of his daughter and other children, and participation in Satanic cult activities including ritual sexual abuse and murder. At first, he denied everything. However, after interrogation and pressure from a psychologist and other advisors, Ingram began to have vivid memories of his involvement in the alleged abuse. Ofshe, an expert witness, attempted to test Ingram's suggestibility. He told Ingram that he had been accused of forcing his son and daughter to have sex together, an event that his children had agreed had not occurred. Ingram initially had no memory of this "event," but after being urged to think about the scene, he began to vividly recall it, and eventually confessed to the alleged activities. This apparent pseudo-memory does not necessarily invalidate Ingram's other recollections. In fact, Peterson (1994), a psychologist involved with the case, reports that Ingram was convicted of six counts of child molestation after "care was taken to eliminate those allegations that could have been contaminated by the investigator's questioning or by contact with the daughters and Mr. Ingram." However, the case does raise issues concerning the effect on memory of questioning, suggestion, and interrogation in both legal and clinical settings.

Psychobiological and cognitive memory research has demonstrated the existence of a variety of different and complex memory systems. The elucidation of these memory systems, particularly nonconscious (implicit) memory, may provide an explanation of how traumatic experiences are stored and recalled. There is evidence that the central theme of stressful events is preserved at least in some experimental situations. However, depending on factors such as the circumstances and severity of stressful events, the accuracy of memory encoding and conscious recall may be affected. It is also clear that the mechanisms and context of memory retrieval may have a profound effect in influencing the content of memory, particularly in susceptible individuals.

The contributions of memory research raise many thorny questions, some which challenge clinical observations. For example, cognitive psychology research does not empirically substantiate the existence of the psychoanalytic theory of repression, an active ego-defensive process. However, it is problematic to extrapolate experimental findings to clinical situations. As acknowledged by a number of investigators, there are ecological differences between experimental conditions and actual clinical situations (Erdelyi, 1985; Holmes, 1974 & 1990; Schacter, 1990). Learning and recall that result from actual involvement in traumatic events may be substantially different than memory for neutral events, or even memory for hearing about or witnessing trauma. Experimental investigations of traumatic memory are limited by the ethical constraints of exposing subjects to truly overwhelming or very prolonged traumatic events. Thus, results from experimental psychology may be limited in understanding the actual impact of traumatic events. Nonetheless, a clear understanding of contributions of memory research is important in evaluating clinical studies of traumatic memory and clinical situations. As a whole, both experimental and clinical observations should be part of an integrated understanding of traumatic memory.

CLINICAL STUDIES OF TRAUMATIC MEMORY

Compared to laboratory memory research, there has been relatively little clinical research concerning traumatic memories originating in childhood. Until relatively recently, the only published study concerning the nature of amnesia for childhood abuse and validity of recovered memories was reported by Herman and Schatzow in 1987. Their study involved 53 women who had sought treatment in time-limited incest survivors' groups. A majority of these women reported that they had experienced complete or partial amnesia for their sexual abuse at some time in the past: 26% reported having had no memory for the abuse and 36% had remembered only some or parts of the events. More amnesia was related to early age of onset of the abuse, chronic abuse, and severity of abuse such as violent or sadistic abuse. Perhaps even more notable was that the overwhelming majority of these women were able to find some corroborating evidence of the abuse. Seventy-four percent (74%) were able to find convincing evidence that the incest had occurred, such as a family member who confirmed it, or, in one case, diaries and other evidence of a deceased brother who had been the abuse perpetrator. Another 9% found family members who indicated that they thought the abuse had likely occurred, but who could not confirm it. Eleven percent (11%) made no attempt to corroborate their abuse, leaving only 6% who could find no validating evidence despite efforts to do so. Critiques of this study note that a proportion of the amnesiac subjects were very young and perhaps had normal childhood amnesia, and that clear, independent corroboration of abuse was not obtained (Ofshe & Singer, 1994; Pope & Hudson, 1995).

A study by Briere and Conte (1993) asked therapists to administer a questionnaire to their patients who reported sexual abuse memories. The questionnaire consisted of a variety of scales about current symptomatology and past life experiences. Of the 450 subjects who reported childhood sexual abuse, approximately 54% reported having had some amnesia for the abuse between the time of occurrence and age eighteen. Greater levels of amnesia were correlated with greater current levels of psychiatric symptoms, early age of onset and severity (e.g., multiple perpetrators, physical

injury, fear of death if they revealed the abuse). The researchers also asked about factors which were likely to produce a greater level of psychological conflict such as enjoyment of the abuse, acceptance of bribes, feelings of guilt or shame. These factors were not correlated with amnesia, suggesting that it may be the noxiousness of the experience itself which produces amnesia, and not intrapsychic conflict. Critiques of this study note methodological problems concerning recruitment and various interpretations of "remembering" (Loftus, 1993; Ofshe & Singer, 1994; Pope & Hudson, 1995). For example, Loftus (1993) notes that the question concerning "forgetting" could be interpreted as volitional suppression of the memory, (e.g., "There were times when I could not remember because I could not remember without feeling terrible").

Williams (1994) has recently studied a non-clinical population. She contacted adult women who had been treated for sexual abuse seventeen years earlier in a city hospital, and asked them to participate in a study about hospital services. One hundred twenty-nine (129) women were included in the study. Their age at the time of abuse ranged from infancy to twelve years old. Thirty-eight percent (38%) of the subjects were amnestic for the experiences or chose not to report them. Because many of the women who did not report the abuse did disclose other intimate details about their lives including subsequent sexual victimization, there is a strong implication that many were actually amnestic for the experiences. There was correlation between amnesia and age at time of abuse, which would be expected since verbal recall of events experienced prior to age three is limited. However, this factor alone could not explain the degree of amnesia in the subject population. Few of the women had been under three years of age, and there was actually greater amnesia in the group that was aged four to six at the time of abuse versus the under three group. More amnesia was associated with perpetrators being family members. Amnesia was not correlated with any particular kind of abuse or severity of abuse (e.g., fondling versus penetration). Critical reviews of this study warn against interpreting the data as representing amnesia since the subjects were not asked directly whether or not they remembered the abuse (Ofshe & Singer, 1994; Pope & Hudson, 1995). Thus, the absence of responses about early abuse could be based on avoidance or normal forgetting.

A recent study by Loftus, Polonsky, and Fullilove (1994) reports more conservative estimates of amnesia for sexual abuse in a clinical population. A substantial proportion (54%) of 105 women in outpatient treatment for substance abuse reported childhood sexual abuse. Most (81%) reported remembering part or all of the abuse, and 19% reported that they forgot the abuse for a period of time. In this study, amnesia was not correlated with either severity of abuse or intrafamilial abuse. Critiques of this study note the absence of independent corroboration and the methodological problems of defining the nature of the "forgetfulness" (Pope & Hudson, 1995).

All of the above studies have the disadvantage of being based on patients' self-report. Three of the studies (Herman & Schatzhow, 1987; Briere & Conte, 1993; Loftus et al., 1994) are clearly retrospective in nature and rely on a clinical population seeking treatment for sexual abuse which may also limit the applicability of the findings. Williams' study (1994) is perhaps the most persuasive in that it involves a non-clinical population and prospectively follows a traumatized population. All of these studies and clinical observations when taken as a whole support the idea that traumatic experiences may lead to profound alterations of mental processes including amnesia. However, as noted by Pope and Hudson (1995), good clinical studies are still needed that independently corroborate the validity of abuse and that victims of abuse develop clear amnesia.

Some potentially relevant and coherent evidence comes from Terr's (1979, 1983, 1985, 1988, 1991) pioneering work with children subjected to psychic trauma. Although Terr's work has been faulted for the absence of clear empirical data, her observations of hundreds of children over decades has face validity. In an overview of the effects of trauma on children (Terr, 1981), she notes that distinct differences are seen in children who have experienced limited, circumscribed trauma (Type I), versus those who have been subjected to chronic traumatization (Type II). Terr's description of memory in children who have experienced limited trauma certainly suggests that traumatic memory has a quality which is different from normal memory:

> Verbal recollections of single shocks in an otherwise trauma-free childhood are delivered in an amazingly clear and detailed fashion . . A few details from a traumatic event of childhood may be factually wrong because the child initially misperceived or mistimed the sequence of what happened. But children with Type I disorders seem to remember the event and to give impressively clear, detailed accounts of their experiences. This remarkable retrieval of full, precise, verbal memories of almost all single-blow traumas makes one conclude that these memories stay alive in a very special way...(Terr, 1991, p. 14)

In contrast, Terr has observed that chronically traumatized children often experience extensive amnesia:

> Children who experience Type II traumas often forget. They may forget whole segments of childhood – from birth to age nine, for instance. Where one sees the difference between these "forgetful" children and ordinary youngsters is in the multiply traumatized child's relative indifference to pain, lack

> of empathy, failure to define or acknowledge feelings, and absolute avoidance of psychological intimacy. Repeatedly brutalized, benumbed children employ massive denial... (Terr, 1991, p. 16)

Terr's clinical observations demonstrate the existence of various kinds of memory in response to traumatic events in childhood. In one study of preschool children who had been subjected to known traumatic events, verbal recall of the events depended on the child's age when traumatized, and on the chronicity of the trauma (Terr, 1988). Children under 28 to 36 months of age tended to have less verbal (explicit) memory of their experiences, a finding that is consistent with the development of verbal abilities around this period (Terr, 1985; Miller, 1979). However, even some of these very young children appeared to have implicit memory of the traumatic events, engaging in behavioral reenactments (e.g., traumatic play, fears, and dreams) of the events despite having no conscious recall. Terr also noted that of thirteen children over the age of 28 months at the time of being traumatized, seven had experienced single, brief events and generally had good verbal recall. The other six children, who had been subjected to more chronic traumatization, generally had impaired verbal memory of the events, but engaged in behavioral reenactments.

There are important implications from Terr's work which begins to explain that both a heightened clarity of memory (hypermnesia) and impairment of memory (amnesia) can result from traumatization. She postulates a kind of hypermnesia for traumatic events in situations that involve limited trauma, and she observes amnesia as a result of chronic traumatization. She also offers evidence that there may be nonconscious memory of traumatic events even when there is no conscious recall. (Little is known about the malleability of implicit memory.) However, it should be emphasized that the accuracy of explicit memory, including recovered memory, following chronic traumatization is not well established. Given that chronically traumatized children use massive denial and dissociative defenses, these children may not encode traumatic memories with the hypermnestic clarity that is characteristic of single event traumas. Thus, chronically traumatized patients are most likely to suffer amnesia for their abuse, and, given the level of denial and dissociative defenses they use, the accuracy of recovered memory in these patients may be most vulnerable to distortions and errors in recall.

There have been recent studies concerning the nature and validity of memory of childhood abuse. Kluft (1995) reviewed records of 34 dissociative identity disorder patients in his practice for evidence of confirmation or disconfirmation of recovered memory. A majority (19) had obtained clear confirmation of childhood abuse. Three (3) recovered "memories" that were later disconfirmed. Of the nineteen patients who obtained confirmation, ten (53%) confirmed events they had always remembered, and thirteen (68%) confirmed events first remembered in therapy (recovered memory). Both valid memories and pseudo-memories occurred two patients. Interestingly, many valid recovered memories (85%) were accessed by hypnosis, challenging the widely held belief that the use of hypnosis strongly contributes to the creation of pseudo-memories.

Our recent data also support the validity for some recovered memory of childhood abuse (Chu, Frey, Ganzel, & Matthews, unpublished data). In a study of 90 inpatients in a trauma program, there was a substantial rate of episodes of self-reported complete amnesia for childhood physical (20%) and sexual abuse (24%). The mean dissociative symptoms for all participants reporting abuse was elevated and consistent with post-traumatic stress disorder. More amnesia was correlated with earlier age of onset and greater chronicity of abuse. Most of the participants who reported a period of complete amnesia for their abuse had attempted to obtain corroboration of the events – 14 of 20 for physical abuse and 19 of 25 for sexual abuse. The overwhelming majority of those who attempted to corroborate their abuse – 13 of 14 for physical abuse and 17 of 19 for sexual abuse – found clear validation for their recovered memories. Of particular note is the descriptions of a subset of severely and chronically abused participants with high rates of amnesia. For the most part, these participants lost memory for whole periods of their lives, recollecting neither traumatic events nor neutral or positive experiences. These descriptions are strikingly similar to Terr's observation of pervasive amnesia for chronically traumatized children, and suggest that the underlying mechanism for this kind of amnesia may not be repression of overwhelming experiences or selective inattention to noxious events. Instead, the massive failure to integrate entire periods of childhood strongly suggests that intensely traumatic experiences may result in a different way of processing and storing information, supporting the notion that traumatic memory is different from ordinary memory. This model is also consistent with the concept of dissociation in which various mental contents exist in different states held separately from each other.

CONCLUSIONS AND IMPLICATIONS FOR CLINICAL PRACTICE

From both experimental investigations and clinical research, there appears to be substantial evidence to support the existence of both conscious and nonconscious memory processes. Under stressful or traumatic conditions, there is also ample evidence that the memory can be affected with both hypermnesia and amnesia. Psychobiologic investigations and studies of both clinical and non-clinical populations suggest that limited trauma can result in a kind memory enhancement, and that overwhelming or repetitive trauma may result in psychogenic amnesia for the experiences. This

latter observation concerning chronic traumatization supports the concept that overwhelming trauma may be dissociated from consciousness. There is also evidence that correlates amnesia for traumatic events with early age of onset, chronicity, severity, and family involvement. Thus, children who experience abuse at very young ages, who suffer multiple kinds of abuse, whose abuse continues over years, and who are abused by family members are most likely to develop serious dissociative symptoms, perhaps including psychogenic amnesia. Unfortunately, at least in many acutely ill clinical populations, this kind of abuse is more the rule than the exception.

It appears that horribly abused children do forget. However, this forgetting does not necessarily support the concepts of repression as opposed to simple dissociation of traumatic experience. When individuals begin to recover memories of past traumatic events, it remains unclear as to what extent these memories reflect the actual events. Recall of single events of childhood trauma have been observed to have an unusual clarity. However, until there is further research, we cannot assume that there is similar clarity for recovered memories of chronic traumatization. In fact, the defenses used to cope with chronic traumatization – denial, depersonalization, and derealization – may make encoding of memory substantially less accurate. After all, even single events of trauma are often recalled with misperceptions and distortions. It is reasonable to assume that chronic traumatization is likely to result in an even greater degree of inaccuracy.

Despite the uncertainties concerning the processes of traumatic memory encoding and recall, recovered memories of severe childhood abuse cannot be dismissed out-of-hand. Although chronic traumatization may negatively affect the accuracy of memory, several clinical studies do support the validity of some recovery memories of childhood abuse. However, more investigation is required to bridge the rift between clinical observations of traumatic events, and research and the empirical observations of the laboratory. Corroboration of childhood abuse, while not necessarily useful in clinical situations, may be essential in demonstrating the effects of abuse. Detailed questions concerning memory may also be important to distinguish between true amnesia, normal forgetting, and selective inattention.

Nonetheless, therapists must understand the findings from memory research that memory content can be highly influenced by the mechanisms of memory retrieval. Although experimental research is not necessarily applicable to clinical situations, therapists must use caution in inquiring about histories of childhood abuse. There is little evidence that direct questioning about abuse, per se, results in false memories of abuse. However, research concerning the use of suggestion and certain kinds of interrogation has shown that memory content can be affected by interactions with others and that pseudo-memory can be induced. Hence, therapists must be careful not to inquire about possible abuse in a way that even subtly encourages a particular kind of response.

Therapists, particularly those who specialize in the treatment of survivors of abuse, must not impose any particular idiosyncratic model of understanding or treatment on patients, and must be open to understanding patients' difficulties in a variety of ways. Fantasies about abuse, suspicions or partly formed ideas about abuse, and dreams about abuse are not the same as the actuality of abuse. Especially when memories are fragmentary, therapists must support the psychological validity of the memories but avoid coming to premature conclusions when there is insufficient evidence to support the occurrence of abuse. Similarly, when recovered memory begins to replace amnesia, therapists must remain open to the possibility of real abuse, but must also encourage patients to reconstruct their personal history in a way that is thoughtful and rational, and is consistent with what is known about the past and current symptomatology.

Therapists who work with patients who have recovered memories of abuse must also scrupulously avoid regressive clinical practices. Most accepted models of treating abuse survivors emphasize building solid ego-functioning, containing and stabilizing symptomatology, and establishing personal safety prior to abreactive work (Brown & Fromm, 1986; Chu, 1992; Herman, 1992; Lebowitz, Harvey, & Herman, 1993). Premature attempts at abreaction of traumatic material often results in profound regression in which current reality, past realities, fantasies, dreams, and fears become inextricably entangled. Such regressive practices may make it difficult or even impossible to establish a coherent and believable personal history.

Understanding the role of childhood trauma and abuse in the development of psychiatric illness has had a profound effect on the lives of many psychiatric patients. Acknowledging and working through traumatic events has helped many patients to deal with the unspeakable horrors of past abuse, and to reclaim a sense of normal human worth. However, work with traumatic memory has not only the potential for enormous benefits, but for serious detrimental effects as well. In order to be most helpful, therapists must combine an understanding of trauma, knowledge concerning memory processes, cautious inquiry, validation and support, and sound psychotherapeutic practices. Work of this kind may be maximally helpful to patients who suffer from sequelae of childhood abuse, and who struggle to remember and make sense of their painful and chaotic lives. ■

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