

# Dissociative Detachment and Memory Impairment: Reversible Amnesia or Encoding Failure?

Jon G. Allen, David A. Console, and Lisa Lewis

The authors propose that clinicians endeavor to differentiate between reversible and irreversible memory failures in patients with dissociative symptoms who report "memory gaps" and "lost time." The classic dissociative disorders, such as dissociative amnesia and dissociative identity disorder, entail reversible memory failures associated with encoding experience in altered states. The authors propose another realm of memory failures associated with severe dissocia-

tive detachment that may preclude the level of encoding of ongoing experience needed to support durable autobiographical memories. They describe how dissociative detachment may be intertwined with neurobiological factors that impair memory, and they spell out the significance of distinguishing reversible and irreversible memory impairment for diagnosis, patient education, psychotherapy, and research.  
Copyright © 1999 by W.B. Saunders Company

**M**ANY PATIENTS with a severe childhood trauma history and dissociative disorders complain of gaps in their memory. These gaps include an inability to remember specific traumatic events in the past and a lack of memory for substantial periods of childhood. Many such patients also report ongoing memory lapses, for example, not remembering what they have said, what they have done, or where they have been. They complain that they have "lost time"—they cannot account for blocks of their day. These ongoing memory disturbances and the associated loss of continuity in experience profoundly disrupt their functioning. Clinicians treating these patients often conclude that the memory for the events transpiring during the "lost time" exists but is not currently accessible, and/or that complex activity during the time interval for which the patient is amnesic is evidence of dissociative identity disorder. Recent research on memory and the neurophysiology of trauma suggest that neither of these conclusions is invariably warranted.

In the context of conducting diagnostic evaluation and treatment of patients with severe and chronic trauma-related disorders, we have endeavored to understand the basis of memory gaps and lost time. We propose that it is useful in principle, although not easy in practice, to distinguish between potentially reversible and relatively irreversible memory gaps. The diagnostic classification of dissociation includes three disorders that entail

potentially reversible memory impairment: dissociative amnesia, fugue, and dissociative identity disorder. However, on the basis of routine clinical experience, we believe that another reason for irreversible memory impairment in patients with posttraumatic disorders is a failure to encode ongoing experience associated with severe dissociative detachment.

To set the stage for our argument that encoding failures associated with dissociative detachment may contribute to memory impairment, we begin by delineating crucial distinctions among the types and stages of memory. After briefly reviewing the three dissociative disorders associated with reversible amnesia, we describe different degrees of dissociative detachment and their potential relation to irreversible memory impairment. We also review evidence suggesting that memory impairment associated with dissociative detachment may be intertwined with neurophysiological concomitants of stress. We conclude by defining the implications for clinical practice and research.

To preview our argument, we propose that evidence of complex actions in dissociative states is not necessarily diagnostic of dissociative identity disorder, and that memory gaps are not necessarily reversible by various memory retrieval techniques. We do not advocate that clinicians aspire to answer definitively the question, "Reversible amnesia or encoding failure?" Instead, we propose that they ask the question.

## TYPES AND STAGES OF MEMORY

### *Declarative Versus Nondeclarative Memory*

The distinction between nondeclarative and declarative memory is one of the most robust in memory research.<sup>1,2</sup> Nondeclarative (implicit) memory is a kind of procedural or "habit memory,"

---

*From the Trauma Recovery Program (J.G.A.) and Neuropsychology Services (L.L.), The Menninger Clinic, Topeka, KS; D.A.C. is in private practice, Topeka, KS.*

*Address reprint requests to Jon G. Allen, Ph.D., Box 829, The Menninger Clinic, Topeka, KS 66601-0829.*

*Copyright © 1999 by W.B. Saunders Company  
0010-440X/99/4002-0005\$10.00/0*

perhaps best exemplified by motor skills such as riding a bicycle or playing the piano. **Nondeclarative memory is manifested in performance, and it is not available to conscious, volitional recall. Nondeclarative memory encompasses conditioned responses; hence, emotional responses to cues associated with stressful events can be included in the realm of implicit memory.**<sup>3</sup>

**In contrast, declarative (explicit) memory can become conscious and can be articulated. Within the domain of declarative memory, it is useful to distinguish between episodic and semantic memory.**<sup>4</sup> **Simply put, episodic memory is memory for the detail of an event, whereas semantic memory pertains to the meanings and abstractions derived from the event.** In this paper, we are concerned primarily with autobiographical memory, that is, memory for information significant to the self.<sup>5-6</sup> Although autobiographical memory includes semantic memory (e.g., knowledge of birthplace), the pervasive dissociative memory impairment of interest to us pertains to episodic autobiographical memories.

We focus on narrative autobiographical memory insofar as clinical practice rests on patients' verbal accounts of their history. We note, however, that the term "autobiographical memory" is inherently vague. We prefer to focus on autobiographical *narrative* as being part of the grist for psychotherapy. Autobiographical narrative is supported—to highly varying degrees—by memory. That is, across individuals or across epochs within an individual's life span, the episodic memory supporting autobiographical narrative may vary in extent (e.g., sparse v rich), modality (e.g., visual v auditory), coherence (e.g., fragmented v organized), and accuracy (e.g., true v false and everything in between). As already noted, autobiographical narratives may be supported not only by episodic memories, but also by semantic knowledge acquired in myriad ways (e.g., by history gathering). In addition, autobiographical narrative may be influenced to varying degrees by conjecture or confabulation—a source of influence psychotherapists should not abet.

#### *Encoding Versus Retrieval Failures*

Another set of distinctions crucial to understanding dissociative memory impairment pertain to the time course of memory. In sequence, these distinctions include encoding (or acquisition), consolidation, storage, and retrieval. Encoding and consolida-

tion are the processes leading to relatively durable storage that permits retrieval.

Encoding explicit episodic memories that will provide a foundation for autobiographical narrative entails attending to information or events in a fashion that meaningfully integrates new information with prior information. Schacter<sup>7</sup> explicated the relation between the extent of encoding and durability of memory in a fashion central to our thesis:

Only a certain kind of semantic encoding promotes high levels of memory performance—an elaborative encoding operation that allows you to integrate new information with what you already know. . . . Elaborative encoding is a critical and perhaps necessary ingredient of our ability to remember in rich and vivid detail what has happened to us in the past. But the dependence of explicit memory on elaboration has a downside, too: if we do not carry out elaborative encoding, we will be left with impoverished recollections (pp. 45-46).

Consolidation is the process by which, over time, encoded information becomes more durable. As Moscovitch<sup>8</sup> summarized, consolidation is complete "when explicit memory for an event can survive disruption by amnesic agents. With this as the marker, consolidation has been estimated to take up to three years in humans" (p. 296). The result of elaborative encoding and consolidation is storage that, in turn, makes retrieval possible. Retrieval depends on cues (e.g., percepts or thoughts) that constitute a fragment of the memory and—by virtue of associative processes—activate the memory network.

To summarize, the key distinction we are proposing in the context of stages of memory is as follows. Memory impairments based on disruption of encoding, consolidation, or storage are irreversible. If a durable memory network is not established, it cannot be reactivated for retrieval. In contrast, memory impairments that involve retrieval failures are, in principle, reversible. If a suitable cue or set of cues is found, the memory can be reactivated.

#### REVERSIBLE DISSOCIATIVE AMNESIA

Potentially reversible dissociative amnesia occurs in three DSM-IV<sup>9</sup> dissociative disorders: dissociative amnesia, fugue, and dissociative identity disorder. Predominantly, these disorders entail impairment of episodic autobiographical memory, although semantic autobiographical memory may

also be involved (e.g., when an individual in a fugue is unable to recall facts pertaining to identity). Steinberg<sup>10</sup> believes amnesia to be the central feature of dissociative disturbance, and there is extensive evidence that dissociative amnesia occurs in conjunction with traumatic events.<sup>11,12</sup> Amnesia is also a criterion for the DSM-IV diagnosis of acute stress disorder, which encompasses numerous dissociative symptoms.<sup>9</sup>

In describing “pure cases of dissociative amnesia,” Kihlstrom and Schacter<sup>13</sup> argued that, “In most cases the amnesia is reversible, and access to the autobiographical memories covered by the amnesia is eventually restored” (p. 341). Presumably, the potential reversibility of such amnesia is based on encoding in altered states. Loewenstein,<sup>12</sup> for example, described dissociative amnesia (DA) as follows:

Dissociation is conceptualized as a basic part of the psychobiology of the human trauma response: a protective activation of altered states of consciousness in reaction to overwhelming psychological trauma . . . *Memories and affects relating to the trauma are encoded during these altered states.* When the person returns to the baseline state, there is relatively less access to the dissociated information, leading, in many cases, to DA for at least some part of the traumatic events (p. 312, emphasis ours).

In theory, returning to the altered mental state in which a traumatic memory was encoded may enable the individual to remember the event insofar as the mental state itself becomes a retrieval cue. From the vantage point of state-dependent memory,<sup>13-16</sup> a mental state (e.g., a mood) can become a retrieval cue. Accordingly, while delineating the effects of dissociation on encoding and storage, Spiegel<sup>17</sup> emphasized reversibility of memory impairment as follows: “To the extent that individuals do enter a spontaneous dissociative state during trauma, the memories may be stored in a manner that reflects this state (e.g., narrower range of associations to context). . . . Furthermore, retrieval should be facilitated by being in a similar dissociative state (e.g., hypnosis)” (p. 137). These various views of dissociative amnesia are consistent with the recent conclusion by Krystal et al.<sup>18</sup> that “Posttraumatic amnesia appears to arise from the suppression of retrieval rather than from an ongoing memory encoding deficit” (p. 350).

A dissociative fugue goes beyond dissociative amnesia inasmuch as the individual is unable to recall not only prior events but also facts pertaining

to personal identity. However, as Kihlstrom and Schacter<sup>13</sup> pointed out, the “boundaries between amnesia and fugue are blurred,” and further, “it appears that most psychogenic losses of autobiographical memory are associated with some alteration of identity. Of course, this is an empirical question, and deserving of systematic research in the future” (p. 343).

Dissociative identity disorder is prototypical of a rapidly reversible impairment of autobiographical memory (associated with state-dependent memory). Specifically, dissociative identity disorder involves amnesia in conjunction with identity alteration, which Steinberg<sup>10</sup> defined as “a person’s shift in role or identity, which is observable by others through changes in the person’s behavior” (p. 13). She characterized severe identity alteration such as seen in dissociative identity disorder as involving “the patient’s shifting between distinct personality states that take control of his or her behavior and thought” (p. 234). The patient with dissociative identity disorder, for example, may alternate between calm and destructively aggressive states.

*When in a calm state, the patient may not remember actions and events that occurred in the aggressive state. When the patient is in the aggressive state (or “alter”), the memories of actions and events in prior aggressive states become more accessible.*

After reviewing this spectrum of dissociative disorders, Kihlstrom and Schacter<sup>13</sup> concluded: “As such, the classical functional disorders, whose underlying mechanism is described as dissociative, may be construed as disorders of memory retrieval” (p. 355). We do not take issue with this conclusion. Rather, we wish to draw attention to another realm of dissociative pathology that we believe may be associated with encoding failures.

### DISSOCIATIVE DETACHMENT

For a century, multiple personality disorder (now dissociative identity disorder) has occupied center stage in the field of dissociative disorders, with the only slightly less dramatic presentations of fugues and dissociative amnesia on its heels. Dissociative identity disorder is prominent in our work with patients with severe trauma-related disorders. Yet we have been increasingly impressed by the role of pervasive and severe dissociative detachment in patients’ impairment—whether or not the detachment is concomitant with dissociative identity

disorder. Indeed, we have found dissociative detachment per se to be associated with profound impairment, including psychotic symptoms and personality decompensation.<sup>19</sup>

### *Degrees of Dissociative Detachment*

As we have described previously,<sup>19,20</sup> detachment takes a variety of forms. In benign contexts, detachment can be seen as a concomitant of “absorption”<sup>21</sup> or “imaginative involvement.”<sup>22</sup> Thus being absorbed in a book, entranced by a ballet performance, deeply engaged in a task, or lost in a reverie entails a narrowed focus of attention and a corresponding degree of detachment from the inner and the outer worlds. Such benign, nondefensive detachment is relatively adaptive, flexible, and reversible.

Pathological dissociative detachment is defensive and driven by fear and anxiety. It escapes conscious control and is often experienced passively, as automatic or reflexive. Furthermore, defensive detachment may be subjectively distressing to the degree that it feels out of control and engenders a painful sense of alienation. Patients complain, for example, about feeling “spacey,” “foggy,” or “unreal.” They have the experience of “floating” or “drifting.” They may feel as if they are acting in a play or a movie, watching themselves from a distance, or dreaming. Some feel as if they are automata or robots—or on “autopilot.” They feel as if they are a “shell,” in a bubble, or behind glass.

Some patients indicate that they go beyond feeling “spacey” and the like to being utterly “blank” or “gone,” as if they are in “the blackness” or “a void.” At the extreme, they sit and stare, almost as if in a catatonic or comatose state. Some patients describe being in such profoundly detached states for hours at a time. When patients are in such states, others find it extremely difficult to engage their attention and to help them resume a normal state of consciousness. Many patients report that they are able to regain full consciousness only after a period of sleep.

Our concept of dissociative detachment encompasses depersonalization and derealization. We believe, however, that the term, “detachment,” is preferable for capturing the common thread among the amalgams of depersonalization and derealization, as well as myriad related states that patients experience. We also find that the term, “dissocia-

tive detachment,” is one that patients immediately grasp.

### *Dissociation of Context Versus Dissociation of Contents*

It is relatively straightforward to conceptualize what is dissociated in dissociative amnesia (traumatic events), fugue (ordinary identity), and dissociative identity disorder (altered identity). But what is dissociated in conjunction with dissociative detachment?

In conceptualizing hypnotic absorption, Butler et al.<sup>23</sup> articulated a concept of “dissociation of context” that we find helpful in understanding dissociative detachment: “By dissociation of context we mean that the narrowed attentional focus of absorption effectively suspends higher order reflective cognitive structures and processes as well as distal environmental information that under normal circumstances would control or constrain thoughts and actions” (p. 44). Dissociation of context captures what Kihlstrom<sup>24</sup> construed as the “essential feature of the dissociative disorders,” namely, “a disruption of the monitoring and controlling functions of consciousness” (p. 384). Butler et al.<sup>23</sup> noted that both environmental and personal context may be dissociated. These two realms of dissociated context reflect derealization and depersonalization, respectively.

The meaning of “dissociation” in the phenomenon of dissociative detachment (including depersonalization and derealization) is radically different from the import of “dissociation” in dissociative amnesia, fugue, and dissociative identity disorder. In these latter disorders, dissociated mental contents are compartmentalized and—even when out of explicit awareness—may continue to influence mental states and behavior (implicitly). In contrast to dissociated contents, dissociated contexts are shut out rather than, in effect, taken in and cordoned off in the mind.

### *Alteration of Consciousness Versus Narrowing of Attention*

Butler et al.<sup>23</sup> emphasized the narrowing of attention in conjunction with dissociation of context. We propose that dissociative detachment may also entail a pervasive alteration of consciousness in addition to a narrowing of attention. Thus, we find it helpful to distinguish dissociative detach-

ment from related states that entail a narrowing of attention.

Butler et al.<sup>23</sup> articulated the concept of dissociation of context in conjunction with hypnotic absorption. In effect, a narrow range of stimuli absorb attention. Spiegel<sup>25</sup> subsequently reiterated this point in conjunction with traumatic dissociation: "To the extent that individuals do enter a spontaneous dissociated state during trauma, memories may be stored in a manner that reflects this state (e.g., narrower range of associations to context)" (p. 226). The same may be said of states of fear. Extensive evidence shows that anxiety biases attention in the direction of a narrowed focus on potentially threatening stimuli.<sup>26,27</sup> In a state of extreme fear, attention may become completely focused on the threatening stimulus, to the exclusion of all contextual stimuli. For example, assault victims may focus all their attention on the weapon. In this fearful state, owing to the shift of attention, encoding of context is decreased. Accordingly, the assault victim may have a clear memory of the weapon, but little memory for the characteristics of the assailant.<sup>28</sup>

Individuals in a state of fear could be considered "detached" insofar as they are unaware of much situational context. Yet here we would emphasize the converse of detachment. In a state of fearful hypervigilance, attention is narrowed but heightened. Indeed, the frightened individual whose attention is riveted on a weapon is highly engaged—not detached. The same might be said of the individual who is absorbed in a fantasy, memory, or flashback—to the exclusion of a broad awareness of environmental events.

In dissociatively detached states, patients are not only narrowing their attentional focus but also coping by disengaging attention more pervasively, from both the outer and the inner worlds. Consciousness in this dissociatively detached state is perhaps better characterized as diffuse or vacuous than as narrowly focused, consistent with patients' accounts of feeling "spacey," "foggy," "fuzzy," "gone," and the like. Patients report that they are "tuned out," and they may not be aware of anything to which they are "tuned in."

#### ENCODING FAILURES ASSOCIATED WITH DISSOCIATIVE DETACHMENT

Steinberg<sup>10</sup> recognized a connection between dissociative detachment and memory impairment.

She observed that depersonalization "may be related to amnesia in that patients may be amnesic for depersonalization, and this amnesia may occur because the symptom itself is initially frightening to many people who experience it" (p. 10). She made a similar argument for derealization. In the context of acute trauma, Spiegel<sup>25</sup> also delineated evidence for the co-occurrence of amnesia with depersonalization, derealization, and detachment. But the specific relation between detachment and amnesia merits further exploration.

Our clinical observations indicate that states of profound detachment are inevitably associated with memory gaps or lost time. After periods of being in the dissociative "void," patients report little or no memory for the experience. They may have been sitting and staring, oblivious to the passage of time. They recall no mentation, only aware of having been "gone" for a period of time, the duration of which they can discern only by external cues (e.g., a clock). In these extreme cases, limited memory is not surprising insofar as there has been relatively little coherent experience to forget or remember. Plainly, the extent of encoding will vary with the extent of consciousness, and the richness of consciousness is a function of novelty and complexity of cognitive processing.<sup>29</sup> The more simple, routine, and practiced the activity, the less consciousness needs to be devoted to it, and the less encoding will occur. Sitting and staring requires minimal consciousness.

Of most interest to us, are memory gaps in cases in which an individual has been somewhat detached—as in depersonalization and derealization—but engaging in behavior that requires a greater degree of conscious attention. Kihlstrom<sup>30</sup> has pointed out, however, that conscious awareness is not always necessary for fairly complex psychological functioning. Many dissociative patients report no memory, for example, of putting on clothes, holding a conversation, writing a list, driving around town, or buying articles at a store. They may be chagrined when they find evidence of having engaged in such unremembered actions. They have "lost time."

Of course, such memory gaps may be indicative of dissociative identity disorder—behavior associated with identity alteration (e.g., an adult woman who, in a dissociated childlike state, buys a toy for herself). Often, however, we hear such reports from patients for whom there is no indication of dissocia-



tive identity disorder. Their actions—albeit not remembered—are relatively consistent with their ordinary behavior and sense of identity.

#### *Dissociation of Context and Impaired Encoding*

We propose that, to the extent that the individual is detached, the dissociation of context interferes with the process of elaborative encoding that Schacter<sup>7</sup> considers requisite for constructing declarative memories. In a state of absorption or hypervigilance, a narrow range of stimuli may be sharply encoded. In contrast, we propose that, in a state of dissociative detachment, there is liable to be a more diffuse and pervasive impairment of the elaborative encoding of episodic memory that would support autobiographical narrative.

As stated earlier, dissociatively detached individuals are not only detached from the environment, but also from the self—their body, their own actions, and their sense of identity. We believe that dissociative detachment from the self (depersonalization) is especially pertinent to problems with conscious encoding of autobiographical memory. As Kihlstrom<sup>30</sup> described, “in order for ongoing experience, thought, and action to become conscious, a link must be made between its mental representation and some mental representation of the self as agent or experiencer. . . . Without such linkages certain aspects of mental life are dissociated from awareness, and are not accompanied by the experience of consciousness” (p. 1451). Thus autobiographical memory is organized around a sense of self.<sup>31</sup> As MacLean<sup>32</sup> tersely stated, “Without an integrated sense of self, there is, so to speak, no place to deposit a memory of ongoing experience” (p. 578).

We have argued that the diminished awareness of context associated with detachment interferes with immediate encoding of experience. In addition, detachment is liable to interfere with reflective thinking about experience in a manner that is conducive to storage. As Johnson and Chalfonte<sup>33</sup> stated, “Mental rehearsal or reactivation of unique, one-time autobiographical events may be the single most important determinant of which personal memories survive to become part of our autobiographical narrative” (p. 334).

Although we developed our understanding of irreversible memory impairment associated with dissociative detachment on the basis of patients’ reports about ongoing memory gaps in their daily

functioning, we speculate that detachment may also account for some memory gaps with respect to past trauma. That is, to the extent that an individual is significantly detached during a traumatic event, elaborative encoding will not occur, and the event will not be remembered in a form that supports autobiographical narrative. We speculate further that, to the extent an individual cannot sustain a state of detachment during a traumatic event, some aspects of the experience might be encoded (i.e., during periods of greater focal attention). Fluctuations in level of detachment during trauma may, therefore, be one factor that contributes to the patchy or fragmented quality of traumatic memories.

#### NEUROPHYSIOLOGICAL CONTRIBUTIONS

Neurobiological changes may coincide with dissociative detachment in contributing to trauma-related memory impairment. The recent discovery of decreased hippocampal volume in relation to trauma and posttraumatic stress disorder<sup>34-37</sup> has underscored the significance of neurobiological contributions to memory impairment, inasmuch as the hippocampal complex plays a pivotal role in the encoding of context in autobiographical memory.<sup>38</sup> Both acute and chronic impairment of brain functioning are likely to be intertwined with dissociative detachment in contributing to encoding failures. We also speculate that neurophysiological changes may contribute to the experience of dissociative detachment. As always, psychology and neurobiology are two sides of the same coin, and dissociative detachment does not occur in a neurobiological vacuum.

#### *Neurobiological Contributions to Stress-Related Memory Impairment*

There is extensive evidence that—at moderate levels—emotional arousal and associated adrenergic activation may enhance memory, likely in concert with heightened attention.<sup>39-40</sup> Yet neurophysiological changes associated with traumatic levels of affect may impair autobiographical memory.<sup>41</sup> Extreme fear can trigger large volleys from the amygdala and thalamus that block hippocampal activity, resulting in failure of consolidation and memory impairment for immediately preceding and subsequent events<sup>41-43</sup>—a mechanism akin to amnesia associated with electroconvulsive therapy or partial complex seizures. Parallel to our

thesis regarding dissociative detachment and encoding failures, LeDoux<sup>43</sup> argued the following:

If the hippocampus was completely shut down by the stress to the point where it had no capacity to form a memory during the event, then it will be impossible through any means to dredge up a conscious memory of the event. If no such memory was formed, then no such memory can be retrieved or recovered. On the other hand, if the hippocampus was only partially affected by the trauma, it may have participated in the formation of a weak and fragmented memory. In such a situation, it may be possible to mentally reconstruct aspects of the experience. Such memories will by necessity involve 'filling in the blanks,' and the accuracy of the memory will be a function of how much filling in was done and how critical the filled-in parts were to the essence of the memory (p. 244).

Endogenous opioids may also contribute to memory impairment.<sup>44</sup> Acute, extreme fear—especially, but not exclusively, when accompanied by physical pain—can trigger the release of opioid peptides that exert an inhibitory and analgesic effect, enabling the individual to carry out whatever behavioral response will be most associated with survival.<sup>45</sup> While facilitating adaptive behavior, however, high levels of opioids may also impair encoding of experience in declarative memory.<sup>39</sup> Concomitantly, as Spiegel<sup>25</sup> pointed out, numbing (dissociative detachment) may constitute an overmodulation of affect that inhibits the level of noradrenergic arousal needed to promote memory.

Extensive evidence indicates that stress is associated with changes in neuronal connectivity in the hippocampus,<sup>46</sup> and that these neurophysiological changes are associated with impairment of learning and cognitive functioning. High levels of circulating glucocorticoids associated with stress-related activation of the hypothalamic-pituitary-adrenal axis can lead to adaptive atrophy of hippocampal neurons, although the neurophysiological mechanisms underlying these atrophic changes are anything but clear.<sup>47</sup> Indeed, the finding of decreased hippocampal volume in patients with posttraumatic stress disorder appears paradoxical, because posttraumatic stress disorder is typically associated with decreased basal levels of cortisol, albeit in conjunction with hyperresponsiveness of the hypothalamic-pituitary-adrenal axis.<sup>48,49</sup> Hence, Yehuda<sup>48</sup> speculates that hippocampal atrophy in posttraumatic stress disorder may relate to heightened glucocorticoid receptor responsiveness.

Whatever the pathophysiology, this period of

atrophy is associated with a reduction in the efficiency with which the hippocampus can lay down new declarative memories and effectively access recent declarative memories. If stress is diminished, neuronal atrophy is reversed, and hippocampal activity normalizes. However, continued stress may also eventuate in irreversible cell loss in the hippocampus, with the possible consequence of permanent compromise of the capacity to lay down and retrieve declarative memories. Furthermore, as a result of decreased hippocampal function and intact procedural (implicit) memory substrates, the impaired individual remains able to behave normally (e.g., to carry on a conversation, write, drive a car, or make purchases) with relatively little capacity to retrieve a verbally encoded record of such behavior.<sup>50</sup>

Hippocampal cell death may result in a relatively generalized and chronic impairment of cognitive processing and memory function—even when the individual is not in a stressful or traumatic situation.<sup>46,47</sup> These findings are consistent with recent reports of generalized memory impairment (especially in verbal memory) in patients with posttraumatic stress disorder.<sup>51</sup> However, much remains to be clarified about the relation between memory impairment and posttraumatic stress disorder,<sup>52</sup> and there are inconsistent findings regarding the correlation between hippocampal volume and memory impairment.<sup>34-37</sup>

#### *A Biological Counterpart to Dissociative Detachment?*

Krystal and colleagues<sup>53</sup> pointed out that, beyond the narrowing of attention in the face of danger, “At extremely high levels of arousal, coherent integration of sensory information breaks down and dissociative symptoms emerge” (p. 245). Degraded hippocampal functioning, with impaired encoding of context, may play a part in the experience of detachment. But Krystal et al.<sup>18,53</sup> proposed far more broadly that dissociation is associated with disrupted cortical integration—“cortical disconnectivity”—including frontal regions that, along with limbic structures, are critical for encoding.<sup>38,54,55</sup> We speculate that dissociative detachment may be a phenomenological counterpart to this cortical disconnectivity that disrupts encoding of internal and external contexts in the course of ongoing experience. Furthermore, sensi-

zation of the opioid response may contribute to the numbing aspect of dissociation, as well as to the dreamlike state of detachment.

In discussing their cortical disconnectivity hypothesis, Krystal et al.<sup>18</sup> note “shifts in interhemispheric processing in hypnotic and dissociative experience” (p. 342), and they point specifically to impaired functioning of the left frontal cortex. These observations of interhemispheric differences are consistent with the functional neuroimaging studies of regional cerebral blood flow in patients with posttraumatic stress disorder by Rauch et al.<sup>56,57</sup> These patients were studied in the midst of reexperiencing trauma, a state evoked by scripted reminders of their trauma history. Albeit with relatively small samples, findings consistently show increased activation in the right-sided paralimbic cortex (including amygdala), concomitant with decreased activation in left frontal cortex (Broca’s area), as well as in the left middle temporal cortex.

Although these neuroimaging studies focus on posttraumatic stress disorder rather than on dissociation, posttraumatic stress disorder and dissociative disorders are highly comorbid, and as Bremner et al.<sup>58</sup> recently observed, “pathological recall of traumatic memories typically occurs in a dissociated state, with symptoms of derealization and depersonalization” (p. 379). Accordingly, alongside the research on compromised hippocampal function, the neuroimaging studies provide convergent evidence of impairment of higher cognitive function in dissociative states. The deactivation of higher cortical structures involved in language production is especially significant. In discussing this neuroimaging research, van der Kolk et al.<sup>59</sup> refer to the reexperiencing of trauma as “speechless terror” and conclude that “during activation of the traumatic memory, the brain is ‘having’ its experience. The person may feel, see, or hear the sensory elements of the traumatic experience, but he or she may be physiologically prevented from translating this experience into communicable language” (p. 109). These dissociative states are not conducive to encoding experience in a form that supports autobiographical narrative. Of course, the extent to which these neuroimaging findings generalize to states of dissociative detachment that do not entail reexperiencing trauma is an open question.

## CLINICAL IMPLICATIONS

### *Diagnosis*

Failure to consider a distinction between dissociative amnesia and memory impairment secondary to dissociative detachment may result in overdiagnosis of dissociative identity disorder. Dissociative identity disorder entails dissociative amnesia coupled with identity alteration.<sup>10</sup> In evaluating the diagnostic significance of memory gaps with patients with dissociative disorders, we regard the extent of out-of-character behavior associated with memory impairment as a useful guide. For example, patients may learn that they have gone to a bar when they would not ordinarily do so; they may discover that they have purchased toys when they have no children; or they may find clothes in their closet that are inconsistent with their usual style of dressing.

Many patients who report various forms of “lost time,” as well as evidence of somewhat elaborate actions in dissociatively detached states, show no such indications of identity alteration. Instead, they are profoundly detached and oblivious to their surroundings, not elaboratively encoding or reflecting on either the stream of outer events or on their own thoughts, feelings, and actions. In light of the unabated controversy over the diagnosis of dissociative identity disorder,<sup>60,61</sup> it is prudent in these instances to consider the possibility of memory impairment secondary to dissociative detachment, rather than simply assuming reversible dissociative amnesia and identity alteration and then endeavoring to confirm a diagnosis of dissociative identity disorder.

Confronted with a mélange of depersonalization and derealization, along with ongoing memory impairment that does not correspond clearly to classic dissociative amnesia, we make a DSM-IV<sup>9</sup> diagnosis of dissociative disorder NOS (DDNOS). This diagnosis is consistent with the multiplicity of overlapping dissociative symptoms, inasmuch as the general criteria for DDNOS in DSM-IV include “disorders in which the predominant feature is a dissociative symptom . . . that does not meet the criteria for any specific Dissociative Disorder” (p. 490). The DDNOS diagnosis in such cases is also consistent with Steinberg’s findings of pervasively elevated subscales in the Structured Clinical Interview for DSM-IV Dissociative Disorders,<sup>62</sup> not



only for patients with dissociative identity disorder but also for those with DDNOS.

### *Patient Education*

Just as it is important for clinicians to not overdiagnose dissociative identity disorder, so is it also important for patients to not overdiagnose themselves. Many patients tend to equate “dissociation” with “multiple personality disorder.” Some infer from the fact that they have memory gaps or lost time that they have dissociative identity disorder. Clinicians must educate themselves about the wide spectrum of dissociation, as well as the complex relations between dissociative disturbances and memory impairments, to be in a position to help patients understand their disorders and treatment needs. In conducting patient education groups,<sup>63,64</sup> we find it helpful to start with a global distinction between dissociative detachment and dissociative identity disorder. Then we point out that dissociative detachment, by drawing attention away from the self and the outer world, may account for some memory failures. Many patients who have engaged in a frustrating and seemingly futile struggle to remember traumatic events feel relieved when they learn about the possibility of dissociatively based irreversible memory impairment.

### *Psychotherapy*

To the extent that it impairs encoding, dissociative detachment will be the bane of the psychotherapist. Many patients who complain of persistent detachment also remember little from one therapy session to the next. Such patients may benefit from written notes as a compensatory device for fostering some continuity in the therapeutic process.

Many traumatized patients face the daunting therapeutic task of constructing some coherent autobiographical narrative from rather fragmentary memory. Some of those with an amalgam of too much reactivity and too little autobiographical memory<sup>41,65</sup> embark on a memory retrieval quest. Some therapists are willing to go to considerable lengths to help patients search for memories. Poole et al.<sup>66</sup> surveyed a large sample of licensed US and British psychotherapists and found that 71% reported employing at least one of a list of potentially suggestive memory recovery techniques to help clients remember childhood sexual abuse. Moreover, 25% of these psychotherapists gave a constel-

lation of responses suggesting a significant focus on memory recovery in the context of having rapidly drawn conclusions about childhood sexual abuse in the face of their clients' denials. This study was conducted in 1993, and such treatment strategies may have abated with the recent alarm about false memories. Yet we would emphasize that such practices take for granted not only that there is memory failure but that also this failure is reversible.

We believe that not all dissociative memory failure is reversible. In that case, such aggressive techniques run the risk of abetting confabulation. As Moscovitch<sup>67</sup> succinctly stated, “A damaged system is more likely to produce faulty output when it is queried” (p. 235). Moreover, when patients do succeed in constructing a narrative, they may only confirm the therapist's conviction that a memory subjected to dissociative amnesia has been “recovered.” The videotape metaphor for memory, though misguided, dies hard.<sup>68</sup> We do not argue that patients and their therapists should not engage in autobiography construction. On the contrary, narrative is essential for healing.<sup>69,70</sup> Rather, clinicians and their patients must be mindful that autobiography—supported to varying degrees by different facets of memory—is a construction. Because of memory impairment, patients who are so inclined may find it helpful to attempt to corroborate their clouded or fragmented memories. In the case of encoding failures secondary to dissociative detachment and the functional brain impairment associated with traumatic states, patients are in the same position as other biographers in that they may need to do considerable historical research into their own past to piece together their own history.

As we strive to put the construction of autobiographical narrative on more solid ground, we should not view the elucidation of memories per se as a therapeutic goal. Fonagy and Target<sup>71</sup> argued cogently that explicating episodic autobiographical memories does not lead to therapeutic change. Episodic memories become schematized in procedural (implicit) memories that comprise working models of relationships. The memories have therapeutic value only insofar as their explication may contribute to illuminating and changing these working models. As Fonagy and Target state,

Change can be thought to occur as a function of a shift in emphasis between different mental models of interpersonal relationships—a change of procedures of ways of living

with oneself and others, not of autobiographical memory (p. 213). . . . Change will occur through the re-evaluation of mental models, or the understanding of self-other representations implicitly encoded as procedures in the human mind. Change is a change of form more than of content: therapy modifies mental procedures, ways of thinking, not thoughts. Insight or new ideas, by themselves, cannot sustain change (pp. 215-216).

### RESEARCH IMPLICATIONS

In the face of burgeoning research,<sup>72</sup> the field of dissociation continues to need theoretical refinement.<sup>73</sup> As Kihlstrom<sup>24</sup> and Frankel<sup>74</sup> pointed out, the term "dissociation" has been loosely applied to such a wide range of phenomena that it is at risk of becoming virtually meaningless. Given this state of the profession, it is little wonder that patients diagnosed with dissociative disorders are often confused about their condition. We believe that theoretical clarification has considerable pragmatic value for sharpening our thinking about different facets of dissociation and their relationships to help prevent clinicians and their patients from drawing erroneous diagnostic conclusions and engaging in therapy techniques that abet confabulation.

Although we have tied our theorizing to research where possible, our thesis that dissociative detachment may contribute to irreversible memory impairment should be considered a working hypothesis. We have had extensive experience using the Dissociative Experiences Scale<sup>75</sup> as a structured clinical interview,<sup>76</sup> and we have routinely observed the impairment of ongoing memory in the context of severe dissociative detachment without evidence of identity alteration and dissociative identity disorder. We are now conducting research interviews with the Structured Clinical Interview for DSM-IV Dissociative Disorders,<sup>62</sup> which will enable us not

only to document these cases more systematically but also to determine their prevalence. Yet we are working with a highly traumatized population with severe psychopathology and extensive comorbidity,<sup>77</sup> so the extent to which our observations pertain to other clinical populations must also be studied.

To sharpen our hypothesis, it is worth considering what an experimental test might entail. One would need to use a symptom-provocation paradigm (as in studies by Rauch et al.<sup>56,57</sup> of posttraumatic stress disorder) that would deliberately evoke dissociative states in suitable patients. First, to test for dissociatively based impairment, the patients' memory for events and actions during evoked dissociative states could be contrasted with their memory for events and actions during baseline (alert) states. Second, to test the proposition—contrary to our hypothesis—that these events and actions were encoded in the altered state (and can be retrieved with the aid of the mental-state retrieval cue), the memory testing would also need to include both evoked dissociative and baseline states. To confirm our hypothesis, one would need to demonstrate that memory is more impaired for the dissociative state, and that recreating that state does not improve memory. Until such research has been conducted, we are inclined to be cautious when making inferences about the potential availability of elaborate episodic memories in patients with severe dissociative detachment.

### ACKNOWLEDGMENT

The authors thank Kathy Ferguson, Peter Fonagy, Janis Huntoon, Lee Smithson, and Helen Stein for their critical reading of the manuscript.

### REFERENCES

1. Schacter DL, Tulving E (eds). *Memory Systems* 1994. Cambridge, MA: MIT Press, 1994.
2. Squire LR. Biological foundations of accuracy and inaccuracy in memory. In: Schacter DL (ed): *Memory Distortion: How Minds, Brains, and Societies Reconstruct the Past*. Cambridge, MA: Harvard University Press, 1995:197-225.
3. Tobias BA, Kihlstrom JF, Schacter DL. Emotion and implicit memory. In: Christianson SA (ed): *The Handbook of Emotion and Memory: Research and Theory*. Hillsdale, NJ: Erlbaum, 1992:67-92.
4. Tulving E. Episodic and semantic memory. In: Tulving E, Donaldson W (eds): *Organization of Memory*. New York, NY: Academic, 1972:381-403.
5. Brewer WF. What is autobiographical memory? In: Rubin DC (ed): *Autobiographical Memory*. New York, NY: Cambridge University Press, 1986:25-49.
6. Nelson K. The psychological and social origins of autobiographical memory. *Psychol Sci* 1993;4:7-14.
7. Schacter DL. *Searching for Memory: The Brain, the Mind, and the Past*. New York, NY: Basic Books, 1996.
8. Moscovitch M. Memory and working with memory: evaluation of a component process model and comparisons with other models. In: Schacter DL, Tulving E (eds): *Memory Systems* 1994. Cambridge, MA: MIT Press, 1994:269-310.
9. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. Ed. 4. Washington, DC: American Psychiatric Association, 1994.
10. Steinberg M. *Handbook for the Assessment of Dissocia-*

tion: Clinical Guide. Washington, DC: American Psychiatric Press, 1995.

11. Loewenstein RJ. Psychogenic amnesia and psychogenic fugue: comprehensive review. In: Tasman A, Goldfinger SM (eds): *American Psychiatric Press Review of Psychiatry*. Vol. 10. Washington, DC: American Psychiatric Press, 1991:189-221.

12. Loewenstein RJ. Dissociative amnesia and dissociative fugue. In: Michelson LK, Ray WJ (eds): *Handbook of Dissociation: Theoretical, Empirical, and Clinical Perspectives*. New York, NY: Plenum, 1996:307-336.

13. Kihlstrom JF, Schacter DL. Functional disorders of autobiographical memory. In: Baddeley AD, Wilson BA, Watts FN (eds): *Handbook of Memory Disorders*. New York, NY: Wiley, 1995:337-364.

14. Bower GH. Mood and memory. *Am Psychol* 1981;36:129-148.

15. Bower GH. Commentary on mood and memory. *Behav Res Ther* 1987;25:443-455.

16. Fuster JM. Memory in the cerebral cortex: an empirical approach to neuronal networks in the human and nonhuman primate. Cambridge, MA: MIT Press, 1995.

17. Spiegel D. Hypnosis and suggestion. In: Schacter DL (ed): *Memory Distortion: How Minds, Brains, and Societies Reconstruct the Past*. Cambridge, MA: Harvard University Press, 1995:129-149.

18. Krystal JH, Bremner JD, Southwick SM, Charney DS. The emerging neurobiology of dissociation: implications for treatment of posttraumatic stress disorder. In: Bremner JD, Marmar CR (eds): *Trauma, Memory, and Dissociation*. Washington, DC: American Psychiatric Press, 1998:321-363.

19. Allen JG, Coyne L, Console DA. Dissociative detachment relates to psychotic symptoms and personality decompensation. *Compr Psychiatry* 1997;38:327-334.

20. Allen JG, Coyne L. Dissociation and vulnerability to psychotic experience: the Dissociative Experiences Scale and the MMPI-2. *J Nerv Ment Dis* 1995;183:615-622.

21. Tellegen A, Atkinson G. Openness to absorbing and self-altering experiences ("absorption"), a trait related to hypnotic susceptibility. *J Abnorm Psychol* 1974;83:268-277.

22. Hilgard JR. *Personality and Hypnosis: A Study of Imaginative Involvement*. Chicago, IL: University of Chicago Press, 1970.

23. Butler LD, Duran REF, Jasiukaitis P, Koopman C, Spiegel D. Hypnotizability and traumatic experience: a diathesis-stress model of dissociative symptomatology. *Am J Psychiatry* 1996;153:42-63.

24. Kihlstrom JF. One hundred years of hysteria. In: Lynn SJ, Rhue JW (eds): *Dissociation: Clinical and Theoretical Perspectives*. New York, NY: Guilford, 1994:365-394.

25. Spiegel D. Trauma, dissociation, and memory. *Ann NY Acad Sci* 1997;821:225-237.

26. Easterbrook JA. The effect of emotion on cue utilization and the organization of behavior. *Psychol Rev* 1959;66:183-201.

27. Mineka S, Nugent K. Mood-congruent memory biases in anxiety and depression. In: Schacter DL (ed): *Memory Distortion: How Minds, Brains, and Societies Reconstruct the Past*. Cambridge, MA: Harvard University Press, 1995:173-193.

28. Heuer F, Reisberg D. Emotion, arousal, and memory for detail. In: Christianson SA (ed): *The Handbook of Emotion and Memory: Research and Theory*. Hillsdale, NJ: Erlbaum, 1992:151-180.

29. Baars BJ. *A Cognitive Theory of Consciousness*. New York, NY: Cambridge University Press, 1988.

30. Kihlstrom JF. The cognitive unconscious. *Science* 1987;237:1445-1452.

31. Howe ML, Courage ML. On resolving the enigma of infantile amnesia. *Psychol Bull* 1993;113:305-326.

32. MacLean PD. *The Triune Brain in Evolution: Role in Paleocerebral Functions*. New York, NY: Plenum, 1990.

33. Johnson MK, Chalfonte BL. Binding complex memories: the role of reactivation and the hippocampus. In: Schacter DL, Tulving E (eds): *Memory Systems* 1994. Cambridge, MA: MIT Press, 1994:311-350.

34. Bremner JD, Randall P, Scott TM, Bronen RA, Seibyl JP, Southwick SM, et al. MRI-based measurement of hippocampal volume in patients with combat-related posttraumatic stress disorder. *Am J Psychiatry* 1995;152:973-981.

35. Bremner JD, Randall P, Vermetten E, Staib L, Bronen RA, Mazure C, et al. Magnetic resonance imaging-based measurement of hippocampal volume in posttraumatic stress disorder related to childhood physical and sexual abuse—a preliminary report. *Biol Psychiatry* 1997;41:23-32.

36. Gurvitz TV, Shenton ME, Hokama H, Ohta H, Lasko NB, Gilbertson MW, et al. Magnetic resonance imaging study of hippocampal volume in chronic, combat-related posttraumatic stress disorder. *Biol Psychiatry* 1996;40:1091-1099.

37. Stein MB, Koverola C, Hanna C, Torchia MG, McClarty B. Hippocampal volume in women victimized by child abuse. *Psychol Med* 1997;27:951-959.

38. Schacter DL, Tulving E. What are the memory systems of 1994? In: Schacter DL, Tulving E (eds): *Memory Systems* 1994. Cambridge, MA: MIT Press, 1994:1-38.

39. McGaugh JL. Emotional activation, neuromodulatory systems, and memory. In: Schacter DL (ed): *Memory Distortion: How Minds, Brains, and Societies Reconstruct the Past*. Cambridge, MA: Harvard University Press, 1995:255-273.

40. Thompson RF. *The Brain: A Neuroscience Primer*. Ed. 2. New York, NY: Freeman, 1993.

41. van der Kolk BA. The body keeps the score: memory and the evolving psychobiology of posttraumatic stress. *Harv Rev Psychiatry* 1994;1:253-265.

42. Feindel W. Response patterns elicited from the amygdala and deep temporoinular cortex. In: Sheer DE (ed): *Electrical Stimulation of the Brain: An Interdisciplinary Survey of Neurobehavioral Integrative Systems*. Austin, TX: University of Texas Press, 1961:519-532.

43. LeDoux J. *The Emotional Brain*. New York, NY: Simon & Schuster, 1996.

44. Jessell TM, Kelly DD. Pain and analgesia. In: Kandel ER, Schwartz JH, Jessell TM (eds): *Principles of Neural Science*. Ed. 3. New York, NY: Elsevier, 1991:385-399.

45. McCabe PM, Schneiderman N. Physiological reactions to stress. In: Schneiderman N, Tapp JT (eds): *Behavioral Medicine: The Biopsychosocial Approach*. Hillsdale, NJ: Erlbaum, 1985:99-131.

46. McEwen BS. Adrenal steroid actions on brain: dissecting the fine line between protection and damage. In: Friedman MJ, Charney DS, Deutch AY (eds): *Neurobiological and Clinical Consequences of Stress: From Normal Adaptation to Posttraumatic Stress Disorder*. New York, NY: Lippincott-Raven, 1995:135-147.

47. Sapolsky RM. Stress, glucocorticoids, and damage to the nervous system: current state of confusion. *Stress* 1996;1:1-19.
48. Yehuda R. Sensitization of the hypothalamic-pituitary-adrenal axis in posttraumatic stress disorder. *Ann NY Acad Sci* 1997;821:57-75.
49. Yehuda R. Recent developments in the neuroendocrinology of posttraumatic stress disorder. *Int J Neuropsychiatr Med* 1998;3(2 Suppl):23-29.
50. Cohen NJ, Eichenbaum H. *Memory, Amnesia and the Hippocampal System*. Cambridge, MA: MIT Press, 1993.
51. Bremner JD, Scott TM, Delaney RC, Southwick SM, Mason JW, Johnson DR, et al. Deficits in short-term memory in posttraumatic stress disorder. *Am J Psychiatry* 1993;150:1015-1019.
52. Wolfe J, Schlesinger LK. Performance of PTSD patients on standard tests of memory: implications for trauma. *Ann NY Acad Sci* 1997;821:208-218.
53. Krystal JH, Bennett A, Bremner JD, Southwick SM, Charney DS. Toward a cognitive neuroscience of dissociation and altered memory functions in post-traumatic stress disorder. In: Friedman MJ, Charney DS, Deutch AY (eds): *Neurobiological and Clinical Consequences of Stress: From Normal Adaptation to Posttraumatic Stress Disorder*. New York, NY: Lippincott-Raven, 1995:239-269.
54. Schacter DL. Priming and multiple memory systems: perceptual mechanisms of implicit memory. In: Schacter DL, Tulving E (eds): *Memory Systems* 1994. Cambridge, MA: MIT Press, 1994:233-268.
55. Schacter DL. Memory distortion: history and current status. In: Schacter DL (ed): *Memory Distortion: How Minds, Brains, and Societies Reconstruct the Past*. Cambridge, MA: Harvard University Press, 1995:1-43.
56. Rauch SL, van der Kolk BA, Fisler RE, Alpert NM, Orr SP, Savage CR, et al. A symptom provocation study of posttraumatic stress disorder using positron emission tomography and script-driven imagery. *Arch Gen Psychiatry* 1996;53:380-387.
57. Rauch SL, Shin LM, Whalen PJ, Pitman RK. Neuroimaging and the neuroanatomy of posttraumatic stress disorder. *Int J Neuropsychiatr Med* 1998;3(2 Suppl):31-41.
58. Bremner JD, Vermetten E, Southwick SM, Krystal JH, Charney DS. Trauma, memory, and dissociation: an integrative formulation. In: Bremner JD, Marmar CR (eds): *Trauma, Memory, and Dissociation*. Washington, DC: American Psychiatric Press, 1998:365-402.
59. van der Kolk BA, Burbridge JA, Suzuki J. The psychobiology of traumatic memory: clinical implications of neuroimaging studies. *Ann NY Acad Sci* 1997;821:99-113.
60. Ross CA. History, phenomenology, and epidemiology of dissociation. In: Michelson LK, Ray WJ (eds): *Handbook of Dissociation: Theoretical, Empirical, and Clinical Perspectives*. New York, NY: Plenum, 1996:3-24.
61. Spanos NP. *Multiple Identities and False Memories: A Sociocognitive Perspective*. Washington, DC: American Psychological Association, 1996.
62. Steinberg, M. *Interviewer's Guide to the Structured Clinical Interview for DSM-IV Dissociative Disorders*. Washington, DC: American Psychiatric Press, 1993.
63. Allen JG. *Coping With Trauma: A Guide to Self-Understanding*. Washington, DC: American Psychiatric Press, 1995.
64. Allen JG, Kelly KA, Glodich A. A psychoeducational program for patients with trauma-related disorders. *Bull Menninger Clin* 1997;61:222-239.
65. Allen JG. The spectrum of accuracy in memories of childhood trauma. *Harv Rev Psychiatry* 1995;3:84-95.
66. Poole DA, Lindsay DS, Memon A, Bull R. Psychotherapy and the recovery of memories of childhood sexual abuse: U.S. and British practitioners' opinions, practices, and experiences. *J Consult Clin Psychol* 1995;63:426-437.
67. Moscovitch M. Confabulation. In: Schacter DL (ed): *Memory Distortion: How Minds, Brains, and Societies Reconstruct the Past*. Cambridge, MA: Harvard University Press, 1995:226-251.
68. Loftus EF, Loftus GR. On the permanence of stored information in the human brain. *Am Psychol* 1980;35:409-420.
69. Meichenbaum D. *A Clinical Handbook/Practical Therapist Manual for Assessing and Treating Adults With Post-Traumatic Stress Disorder (PTSD)*. Waterloo, Ontario, Canada: Institute Press, 1994.
70. Foa EB. Psychological processes related to recovery from a trauma and an effective treatment for PTSD. *Ann NY Acad Sci* 1997;821:410-424.
71. Fonagy P, Target M. Perspectives on the recovered memories debate. In: Sandler J, Fonagy P (eds): *Recovered Memories of Abuse: True or False?* Madison, CT: International Universities Press, 1997:183-237.
72. Michelson LK, Ray WJ. *Handbook of Dissociation: Theoretical, Empirical, and Clinical Perspectives*. New York, NY: Plenum, 1996.
73. Allen JG. The return of the dissociated. *Contemp Psychol* 1998;43:74-75.
74. Frankel FH. Dissociation in hysteria and hypnosis: a concept aggrandized. In: Lynn SJ, Rhue JW (eds): *Dissociation: Clinical and Theoretical Perspectives*. New York, NY: Guilford, 1994:80-93.
75. Bernstein EM, Putnam FW. Development, reliability, and validity of a dissociation scale. *J Nerv Ment Dis* 1986;174:727-735.
76. Allen JG, Smith WH. Diagnosing dissociative disorders. *Bull Menninger Clin* 1993;57:328-343.
77. Allen JG, Coyne L, Huntoon J. Complex posttraumatic stress disorder in women from a psychometric perspective. *J Pers Assess* 1998;70:277-298.