Implicit Memory in Posttraumatic Stress Disorder With Amnesia for the Traumatic Event

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This report documents a case of posttraumatic stress disorder (PTSD) following psychological trauma with cerebral insult and amnesia for the traumatic event. The case history demonstrates the role of implicit memory in PTSD and indicates that the mechanisms of psychopathology are one-trial sensitization and conditioned emotional responses.

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Recent clinical reports have suggested a role for implicit or nondeclarative memory in posttraumatic stress disorder (PTSD) associated with chronic abuse, as well as for PTSD following exposure to a single traumatic event.[1,2] Evidence for the latter is found in case reports of the co-occurrence of PTSD and brain injury where there is neurogenic amnesia for the trauma.[2–4] These cases presumably represent examples of one-trial, nondeclarative learning in the context of cerebral dysfunction producing amnesia, and they provide evidence contrary to the position that neurogenic amnesia precludes the development of PTSD. The latter position is based on the commonsense view that conscious recall of a traumatic experience is essential to the development of PTSD. In support of this claim, observational studies of head-injured samples indicating low rates of PTSD and mild or incomplete forms of the syndrome have been cited.[5,6]

Although the incidence of PTSD in cases involving neurologically caused amnesia is not high,[7,8] the existence of the clinical phenomenon requires explication. The unfortunate co-occurrence of emotional and cerebral trauma is, scientifically, a felicitous combination of events in which the cause of the emotional trauma also causes disruption of the nervous mechanism responsible for consolidation or retrieval of conscious memory. Therefore, the examination of cases where PTSD occurs in conjunction with neurogenic amnesia should enhance understanding of the functional implications of the operation of implicit memory, not only in PTSD, but in other forms of psychopathology as well.

This report documents the occurrence of PTSD following a discrete episode of psychological trauma associated with an event that resulted in amnesia caused by anoxic encephalopathy. To our knowledge, all previously reported cases of PTSD in the context of neurogenic amnesia have involved traumatic brain injuries. This case represents the initial documentation of PTSD associated with anoxic encephalopathy in which the duration of exposure to the trauma is more extended than that associated with traumatic brain injury and in which there is a corresponding increase in the severity of the psychopathology. The significance of this report lies not only in the documentation of an instance of PTSD with neurogenic amnesia, but also in the observation of the relationship between duration of emotional trauma and severity of symptoms.

CASE REPORT

A healthy 53-year-old man with no psychiatric or neurological history was buried completely under 5.5 m of sand in a construction accident. It required 15 minutes to uncover him. He was given artificial respiration at the scene and was unconscious when admitted to the university hospital. A CT scan of the brain on admission was normal. The anoxia caused a coma of 2 days' duration. After regaining consciousness, he was acutely confused and intermittently agitated for several days. He made a relatively good physical recovery with minor residual problems that included pulmonary contusions and left upper extremity weakness. An EEG study obtained 2 weeks after the anoxia was normal.

He was discharged from the hospital after a 3-week stay, although he continued to experience episodic confusion over the next month. He was followed by the neurology service on an outpatient basis and was subsequently referred for neuropsychological evaluation and psychiatric treatment.

Neurological Examination. Initial neurological examination about one month after discharge from the hospital was significant for headache in the left frontal region as well as

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pain in the left side of the neck and left arm. There was mildly diminished sensation and strength in the left arm. There was no diplopia, dizziness, vertigo, or incontinence. The patient described a variable retrograde memory disturbance of 15 years' duration and a complete anterograde memory disturbance for the initial 2 weeks after the injury. He was unable to remember the occurrence of the accident. With the exception of the headache and evidence of peripheral injury, mental changes comprised the major residual deficits. An electromyogram obtained 3 months after the accident confirmed a mild left brachial plexus injury. A second head CT scan, this time with contrast, was carried out 6 months after the accident. That scan also was normal.

At the time of the initial neurological examination, the patient's family indicated that he had exhibited forgetfulness and occasional episodes of confusion, the latter diminishing in frequency. In addition, they reported that he had shown a change in personality since discharge from the hospital 3 weeks after the accident. In particular, his wife said that he was not the man he had been previously, apparently referring to expressed fears and observed hyperarousal. The patient himself complained of continuing memory and concentration problems and of losing track of his train of thought. He indicated that he was frightened and felt that he had changed in some fundamental way. He was not able to return to work and was referred for neuropsychological studies to assess his cognitive functioning and apparent personality change.

Neuropsychological Evaluation. Neuropsychological examination was conducted 3 months after the accident. That evaluation involved assessment of overall intellectual ability and of verbal and nonverbal cognitive abilities in several domains, including executive function, attention, language, visuospatial function, memory, praxis, and emotional functioning. The results indicated high average–range intelligence and a number of specific cognitive impairments. With respect to executive function abilities, he showed a mild verbal reasoning impairment that was due largely to cognitive rigidity and perseveration. On the other hand, his ability to organize and effectively solve problems involving complex visuospatial information was unimpaired. Mild, global attentional impairments also were evident in terms of diminished immediate memory capacity and impaired concentration. Oral language functioning was characterized by slowed rate of speech, diminished language production, and mild difficulty with naming on confrontation. Visuospatial perceptual and constructional abilities were unimpaired. Ability to learn and remember new information, both verbal and nonverbal, was mildly impaired. This difficulty was thought to result, in part, from attentional and verbal organizational deficits, given that his performance on memory tasks involving rapidly presented and complex verbal information was more deficient than on tasks in which attentional and organizational demands were minimized. There was no evidence of dyspraxia for skilled movements of either hand, the tongue, or the lips. He did show diminished grip strength and motor speed of the left hand, presumably related to the left peripheral injury.

On examination of emotional status, the patient was found to be depressed, anxious, and preoccupied with his physical condition and changes in his functional abilities. He ruminated continually about sudden death, and these ruminations took the form of specific fears, particularly of the earth opening up and swallowing him. He had nightmares each night with the same content. It was felt that the depression, anxiety, and rumination exacerbated the attentional and memory problems produced by the neurological dysfunction.

The circumstances of the accident and the severe emotional and behavioral symptoms fulfilled diagnostic criteria for PTSD.[9] These included the following: being buried alive and suffocating for several minutes (exposure to an overwhelmingly stressful event); presumed terror while buried and still conscious (response involving great fear or horror); recurrent, intrusive thoughts about the trauma and fear of its imminent recurrence (re-experience of the traumatic event); avoidance of most activities, among them exposure to construction sites and driving and walking outside his home, and a sense of impending death (avoidance/numbing); sleep disturbance and exaggerated startle response (increased arousal); persistence of psychopathology (duration greater than 1 month); and social and occupational incapacity (disturbance in functioning).

From a purely cognitive point of view, he was considered a good candidate for resumption of many of his social and occupational activities, since the cognitive deficits generally were mild. However, effects of the PTSD rendered him socially and occupationally disabled. He was diagnosed with anoxic encephalopathy and posttraumatic stress disorder and was referred for psychiatric treatment.

Psychiatric Treatment. The patient entered psychotherapy 5 months after the accident and was seen in weekly and then twice-weekly psychotherapy for 1 year. This man—who had worked steadily for 31 years in construction, had managed two small properties that he owned, and was proud of his past ability to provide for his family—now experienced himself as markedly changed, disabled, and fearful. His waking moments were entirely consumed in ruminating about the accident and its effects. Among the most prominent symptoms were nightmares each night about being buried, near-constant fear of imminent death, and intrusive thoughts that the earth would open up and swallow him when he was outside his home. A trial on tricyclic antidepressant medication during the first year of treatment was not helpful.

He was transferred to a second, and then a third, psychotherapist for 3 additional years of weekly treatment. Additional therapeutic efforts included an amobarbital interview and a visit to the site of the accident as attempts to elicit recollections of the trauma. Despite these efforts, he never consciously recalled the traumatic event, and there was no change in the severity of his symptoms or in his functional state.

Throughout the 4 years of treatment, each of his psychotherapists conducted therapy on the assumption that the patient must have been repressing conscious memories of the terror of being buried alive. The patient consistently maintained that he did not remember the event and believed that there must have been some other, undiscovered physiological basis for his problems.

DISCUSSION

This case of PTSD with symptoms representing specific aspects of the trauma without conscious recall demon-

strates the role of nondeclarative memory in this disorder and supports the position that sensitization and conditioned emotional responses are sufficient to generate PTSD and maintain its debilitating symptoms. The case also illustrates the inadequacy of the commonsense view that PTSD and neurogenic amnesia are incompatible.

The contemporary understanding of the existence of independent memory systems provides a rational explanation for symptoms of PTSD that emerge as a result of an event that produces both overwhelming stress and cerebral insult causing amnesia for the traumatic event. Evidence from both animal and human research demonstrates the existence of at least two independent memory systems, each mediated by different brain structures.[10,11] The declarative system, comprising in part one's episodic or personal history, is mediated by medial temporal and diencephalic structures. Other, nondeclarative memory systems are mediated by various structures independent of the medial temporal–diencephalic system and are responsible for changes in behavior and psychological functioning, including those involved in habit and skill learning and in conditioned emotional learning. Classical conditioning and sensitization (augmentation of response subsequent to exposure to painful or otherwise intense stimulation) are examples of learning in which conscious recall either is not necessary or is not even involved as a component of the memory process. Thus, the observation of PTSD in the absence of declarative memory for the traumatic event might be considered an expected clinical corollary to the findings in preclinical studies of multiple memory systems.

In the present case study, the patient's declarative memory was rendered dysfunctional temporarily as a result of anoxia, reflected in the retrograde and anterograde memory disturbances. Accordingly, although the emotional trauma must have been experienced consciously (because it occurred before he succumbed to the anoxia), the neurophysiological effects of the anoxia rendered the experience inaccessible as an episodic memory. However, certain aspects of the event, in particular the excessive fear and associated sensory images, not only were registered, but also must have been consolidated and remained accessible to one or more nondeclarative systems. This mechanism is identical to that postulated for PTSD in cases of head injury.[2,3]

It has been argued that occurrence of PTSD is not possible in the absence of conscious or preconscious recollection of an emotionally traumatic event.[5,6] According to this position, this patient's PTSD in the context of failure to recall the trauma must be considered a consequence of repression. However, that argument would seem indefensible in this case. The nature of the cerebral insult (anoxia), the duration of coma, the occurrence of retrograde and anterograde memory disturbances, and the neuropsychological findings all are consistent with brain injury and its associated cognitive effects. These factors, in particular the retrograde and anterograde memory disturbances, would account for the patient's inability to recall the accident and are incompatible with the notion of repression as an active, albeit unconscious, masking of the episodic memory.

Alternatively, it might be argued that this patient's PTSD developed as a result of factors that occurred some time following the accident, coma, and initial confusional period and was not related to nondeclarative learning during the exposure. In this view, the PTSD would have developed secondarily on the basis of information the patient received from family and professionals about the accident and his injuries. It is difficult to disconfirm this notion directly, although such an account is not well supported by the history and has little explanatory power. Symptoms of PTSD were noted within 3 weeks of the event, at a time when the acute, postinjury confusion was still resolving. Appearance of symptoms in this time frame would support (but not prove) the notion that the event itself, rather than secondary traumatization, was the cause of PTSD. Furthermore, it is difficult to conceive how the hyperarousal symptoms and specific fear responses might be generated by purely declarative processes as opposed to implicit learning. At least one recent experimental attempt to induce symptoms of PTSD in normal volunteers by means of an analogue of secondary exposure to emotionally provocative events failed.[12]

In the present case, the period of exposure to the emotional trauma was on the order of minutes, whereas in cases of head injury, exposure is typically on the order of seconds. The differential in terms of the period of exposure to the traumatic event may explain the greater severity of symptoms in the present case as compared with that reported in cases of PTSD with traumatic head injury. Such covariation of extent of exposure with symptom severity appears to be consistent with other forms of PTSD in which duration as well as intensity of emotional trauma determine the severity of symptoms. Comparison of this case with cases of PTSD with traumatic brain injury supports the notion that PTSD in general is mediated by nondeclarative memory systems since, even in the absence of declarative memory for the traumatic event, symptom severity is related to extent of exposure.

Clinically, cognitive-cerebral deficits may exacerbate symptoms of PTSD and increase the resistance to treatment and to any potentially ameliorative experience. For example, in this case, cognitive impairment, particu-

larly the executive dysfunction involving reasoning difficulty and perseveration, almost certainly potentiated the rumination and the intractability of the patient's other symptoms.

This case and others involving PTSD with amnesia for the traumatic event provide insight into the etiology of all forms of PTSD, irrespective of co-occurrence of cerebral insult. That is, it is conceivable that the psychopathological mechanism in all cases of PTSD is essentially identical with that involved in cases of neurogenic amnesia. The significance of the co-occurrence of brain injury and PTSD for understanding the etiology of PTSD is that the disorder develops in spite of associated episodic memory disturbance, thereby unmasking the causal role of implicit memory. Accordingly, we suggest that in PTSD with or without neurogenic amnesia, nondeclarative memory constitutes the fundamental psychopathological mechanism.

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