

**From Hysteria to Posttraumatic Stress Disorder:  
Psychoanalysis and the Neurobiology of Traumatic Memories**

Yoram Yovell, M.D., Ph.D.

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Memories in general, and memories of trauma in particular, are of extreme importance to psychoanalysis, and have been for over a hundred years. To this day, traumatic memories are the focus of controversy regarding both theory and technique within the field of psychoanalysis. Over the past few years, they have also become a subject of heated, politically charged debate in the popular media and in the general psychological and psychiatric literature. Psychoanalytic formulations of traumatic memories have come under intense scrutiny, and are sometimes unfavorably contrasted with current neurobiological and psychological formulations.

Within the framework of a case example, I will review some of the original psychoanalytic approaches to traumatic memories. Then I will review a few recent neurobiological findings about the structure and function of the brain's memory systems. Last, I will attempt to use neurobiological insights into the nature of memory to update psychoanalytic theory regarding traumatic memories, and illustrate how they may be applied to our clinical work. In doing so, I take the approach that psychoanalytic theory can and should go beyond our current neurobiological understanding of the mind, but it must never contradict it. Therefore, psychoanalytic theory should be continuously re-examined and revised, to ensure its coherence with emerging neurobiological research findings.

This approach is in itself debatable, and the desired relationship between psychoanalytic theory and neurobiology is a focus of disagreement within psychoanalysis. Freud, however, when formulating his original models of the mind, based them on the neurobiology of his day, with which he was intimately familiar. He also acknowledged that his ideas should be revised and reformulated in light of emerging biological data:

The deficiencies in our description would probably vanish if we were already in a position to replace the psychological terms by physiological or chemical ones... Biology is truly a land of unlimited possibilities. We may expect it to give us the most surprising information and we cannot guess what answers it will return in a few dozen years to the questions we have put to it. They may be of a kind which will blow away the whole of our artificial structure of hypotheses (Freud, 1920, p. 60).

I believe that it is no less important for us to keep challenging and revising our hypotheses today.

**Tara's dream**

I would like to begin my discussion with a dream. This dream was reported to me by Tara, a corporate lawyer who was 29 years old when she first came to see me. Tara lived alone in an apartment she recently purchased in Manhattan. When she entered my office, my initial impression was that Tara was attractive, slim, upbeat, and energetic. She also appeared younger than her stated age. Despite her youth, Tara was already quite successful in her professional life, and was well on her way to becoming a partner in her law firm. But her personal life was, in her words, "a big mess." Once she began to describe her life to me, Tara no longer seemed upbeat. She wanted help overcoming her Bulimia Nervosa, as well as her self-hate, her feelings of emptiness and worthlessness, and her mild but persistent depression, which was associated with overeating and oversleeping. In addition, she had no friends, and

she felt that she was “stuck” in an unsatisfying six-year relationship with Harold, a man who was twenty years her senior. My impression of Harold, formed through Tara’s descriptions of him, was that he was a loner, that he was well-meaning but emotionally constricted, and that he felt very damaged himself. Tara said that she didn’t love Harold, but she was unable to leave him because she felt “safe” with him. She felt that way because Harold was not disgusted by her body or by her eating disorder. She wondered whether therapy might help her gather the courage to leave Harold, something she had wanted to do for years.

We began twice-a-week psychoanalytic psychotherapy, with some supportive elements. I also referred Tara to an outpatient eating disorders program, where she attended the weekly sessions faithfully for four months, and made several women friends. In addition, I started Tara on Zoloft, which I switched to Effexor three months later, because her depression did not remit fully on Zoloft.

Tara had the dream about a year into therapy. By that time, she was no longer depressed, and her eating disorder was in full remission despite ongoing urges to binge and purge. Tara had left Harold, although they still exchanged frequent e-mails. On the night of the dream, Tara had had her second date with Jeff, a man she met through her job and whom she liked a lot. Jeff was Tara’s age, and seemed genuinely interested in her. At the end of their evening together, Tara invited Jeff to her apartment, where they talked for a long time, and kissed passionately. Before he left, Jeff confessed that he was in love with Tara and that he wanted to be her boyfriend. When Tara arrived at my office the next day, she was depressed, pessimistic, and reported that she felt “numb.” She told me that after Jeff left, she binged and purged, something she hadn’t done in many months. She couldn’t say what made her feel and behave the way she did. My attempts to help Tara clarify her feelings led nowhere. Then she told me the following dream:

I am playing outside of our house, with a few other girls. I’m about four or five years old. We see a snake in the grass. It’s a huge python, not a boa constrictor. Someone says that it’s a boa constrictor, but it’s not. It’s bigger, and it’s not a pet. But it’s a friendly snake, and we all play together. It crawls under our dresses and does all this stuff that makes me scared. As we play with it, it curls around us and begins to squeeze us. At first it’s funny and it tickles and we all laugh, but then it squeezes hard. It begins to hurt. I can’t breathe. I try to scream but I can’t. And I can’t move. The snake is all around us. I feel that the other girls who were playing with the snake are already dead. They were crushed. I know that I am about to die. And everything is dark. And I’m suffocating. I think that that was the end of this dream. I’m not sure how it ended. But then there was a second part: I don’t know who killed it, but I guess it was my mother. I come back inside the house and open the door to the kitchen. I see my mother standing with her back to me. She’s cutting something big on the counter. There is blood everywhere: on the walls, on the floor, on my mother. She is cutting up the snake, and shoving the pieces down the disposal unit. There’s a lot of noise in the kitchen, and the smell of blood and raw meat. My mother doesn’t notice me at first, and I just stand behind her and watch her work. It’s disgusting, I can see its guts and everything. But I can’t look away. Then she turns around and sees me. I can tell how upset she is. She is very angry that I came in and saw what she was doing. I was not supposed to see it. It’s almost like she was trying to get rid of the snake before anyone noticed it, and clean up all the mess. But now I saw it, and I am in trouble. Then I woke up. It was still dark outside. I was afraid to go back to sleep so I just sat up in my bed and waited for it to be morning.

As I listened to Tara and wondered how to respond to her dream and the events that preceded it, I imagined what she might have felt as she waited for the morning to come. The clarity of her dream narrative, probably born of secondary revision during the sleepless pre-dawn hours, was in stark contrast to the confusion and numbness with which she usually experienced and described her inner world.

### **Freud's theories of hysteria**

In order to better explain my response to Tara's dream and put it in historical and neurobiological perspective, I would like to describe another difficult evening, which took place more than a century ago, the evening of April 21, 1896. On that evening, Sigmund Freud, a neurologist who was days away from his 40th birthday, and desperate for an academic promotion that was long overdue, gave a lecture to the distinguished members of the "Society for Psychiatry and Neurology" in Vienna. The ambitious title of his talk was "The Aetiology of Hysteria." His expectations for the evening were probably high. But, by Freud's own account, the evening turned out to be a disaster. In an emotional letter to his friend Wilhelm Fliess, written days after the lecture, Freud summed up the response to his talk succinctly: "...the donkeys gave it an icy reception."

Why didn't the distinguished Viennese physicians like what they heard on April 21? And why did Freud himself, in the years to follow, come full circle and change his own mind about the subject matter of his talk? It is perhaps because, in his lecture, Freud traced the origins of the hysterical symptoms that plagued many young women in Vienna to repressed memories of sexual abuse that occurred in their early childhood. In other words, he saw hysteria as a Posttraumatic Stress Disorder, which was accompanied by recovered memories of sexual abuse. This bold conclusion was foreshadowed by the famous statement in the opening chapter of *Studies on Hysteria*, which Freud had published with Josef Breuer three years before his poorly received lecture: "Hysterics suffer mainly from reminiscences."

But in his 1896 talk, Freud was even more specific in spelling out what he believed:

...at the bottom of every case of hysteria there are one or more occurrences of premature sexual experience, occurrences which belong to the earliest years of childhood but which can be reproduced through the work of psychoanalysis in spite of the intervening decades (Freud, 1896b, p. 203).

In his inimitable style, which probably did not help him at all on that fateful evening, Freud added, "I believe that this is an important finding, like the discovery of a source of the Nile in neuropathology."

Freud based his shocking conclusion on a case series of 18 patients he had treated, 12 women and six men. In two cases, he was able to obtain parallel history from a family member that confirmed his patients' memories of childhood sexual abuse, which were recovered and reported while they were in therapy with Freud. But his conviction in his own "seduction theory" did not last. Within a couple of years, his letters indicate, he came to feel that his patients' reports were derivatives of repressed, unconscious wishes rather than recovered memories.

In 1924, 28 years after his unpleasant evening, an older and disillusioned Freud, who was editing a new edition of his 1896 paper, added a footnote, after his statement about the link between hysterical symptoms and childhood sexual abuse: "All this is true; but it must be remembered that at the time I wrote it I had not yet freed myself from my overvaluation of reality and my low valuation of phantasy."

Freud's original formulation on the etiology of hysteria was that it was due to the hidden memory of traumatic early experiences of sexual abuse, which were repressed and seemingly forgotten, only to crop up as symptoms years later. This view was in sharp contrast to the opinion of Freud's mentor, the influential French neurologist Jean-Martin Charcot. Charcot had a strict biological approach to mental illness, and had no interest in understanding the meaning of his patients' symptoms and communications. In the obituary Freud wrote for Charcot, he cited Charcot's theory regarding the etiology of hysteria:

...heredity was to be regarded as the sole cause. Accordingly, hysteria was a form of degeneracy, a member of the familial neuropathies. All other aetiological factors played the part of incidental causes... (Freud, 1893, p.21)

Although he admired Charcot, Freud was an independent thinker, and he believed that Charcot was wrong about the etiology of hysteria. In the aftermath of the first World War, this issue became a focus of urgent clinical interest because of the epidemic-like appearance of Shell Shock, an ailment which resembled hysteria in every aspect. The key features of hysteria as described by Charcot were dissociative spells, memory disturbances, crippling anxiety, vivid nightmares, and pseudoneurological symptoms such as paralysis and mutism. These symptoms waxed and waned, but the course of the illness tended to be chronic. All these features were also central to Shell Shock. Several clinicians who worked with traumatized soldiers, including W. H. R. Rivers, Abraham Kardiner, and Herbert Spiegel, felt that Shell Shock and hysteria were one and the same, and treated their patients with a modified version of the talking cure.

In her book *Trauma and Recovery*, Judith Lewis Herman (1992) traces the nosology of this cluster of symptoms from the end of the nineteenth century, when it was called hysteria, through its successive incarnations as Shell Shock, Traumatic Neurosis, and, finally, Posttraumatic Stress Disorder (PTSD), the name currently used in the general psychiatric and psychological literature.

The prevailing and perhaps politically correct approach today is to consider the traumatic experience as *the* etiology of PTSD, rather than just its trigger. Most of the neurobiological data I will present will support this notion. However, Charcot's hypothesis was not totally wrong. There is now also strong evidence for a genetic predisposition to PTSD, the most environmental of all mental disorders: In a well-conducted but virtually ignored study, William True and his colleagues (1993) analyzed over 4000 twin pairs who served in the military during the Vietnam era. In medicine, the gold standard for ascertaining whether an illness has a genetic component has been the assessment of its concordance in twin pairs. The method is based on the fact that identical twins share 100 percent of their genetic material, whereas fraternal twins, like ordinary siblings, share 50 percent of their genetic material. If identical twins are more likely than fraternal twins to either both have or both not have the illness in question (that is, to be *concordant* for it), then this is considered proof that there is a genetically-encoded predisposition to the illness. True and his colleagues discovered that identical twins were significantly more concordant for PTSD symptoms than fraternal twins. The concordance in identical twins was far from 100 percent, indicating that environment and exposure are crucial, but a genetic basis for PTSD does exist.

Part of what makes True's study so fascinating and so powerful is that no two twins served in the same unit in Vietnam, and as a result, no two twins were exposed to the same combat trauma. After the tragedy of the Sullivan family in World War II, which served as the basis for the movie *Saving Private Ryan*, the military adopted a policy of not allowing two siblings to serve in the same unit. What this means is that all the twins who experienced trauma and PTSD in Vietnam had their experiences independent of each other.

### **Neurobiology of traumatic memory**

To return from PTSD to hysteria, Freud's first theory of hysteria was that his patients' memories of traumatic abuse were real. His later view was that their memories were mostly the product of infantile fantasy. Either way, it appears that something is the matter with memory processes in people who report a history of trauma: "Hysterics suffer mainly from reminiscences." As we will see, current neurobiological findings support this notion. But the theories that psychoanalysts first put together about the nature of memories were formulated before the current wave of discoveries in neurobiology. In the last thirty years, there has been incredible progress in the neurobiological understanding of memory in general and traumatic memory in particular, and psychoanalytic theory, at least in part, appears to have fallen behind.

Over a decade ago, I had the good fortune to work as a graduate student in the laboratory of Eric Kandel and Jimmy Schwartz, at the Center for Neurobiology and Behavior, Columbia University. Dr. Kandel, my mentor, who was trained as a psychiatrist, wrote recently that psychoanalysis, which revolutionized our understanding of "mental life," has "not evolved scientifically," and that,

This is regrettable, since psychoanalysis still represents the most coherent and intellectually satisfying view of the mind. If psychoanalysis is to regain its intellectual power and influence... it will need to be engaged constructively by those who care for it and who care for a sophisticated and realistic theory of human motivation. (ref)

I will not go into the interesting debate within psychoanalysis, which Kandel alluded to in his paper, about whether or not psychoanalysis needs to make its theories congruent with current neurobiological knowledge. In my opinion, the dilemma is only how to do it, or, more accurately, how to do it without precipitating a breakdown of psychoanalysis' identity and integrity.

### **How the brain remembers**

Let us begin by looking at how the brain remembers. Human beings have two distinct memory systems that usually work seamlessly and in parallel: the declarative, or explicit memory system, and the procedural, or implicit memory system. Examples of declarative memories are your social security number, and the weather on the day of your wedding. Examples of procedural memories are how to ride a bicycle, and the way you react when you get really scared.

For the purpose of our discussion of traumatic memories, I will focus on the declarative memory system, and on a part of the procedural memory system, which mediates emotional memories of fear and anxiety. These two memory systems are anatomically and developmentally distinct: Declarative memory is mediated to a large extent by the hippocampus, and emotional memory is mediated, at least in part, by the amygdala. Jake Jacobs, Lynn Nadel, and their associates called the hippocampus-based memory the "cool" system, and the amygdala-based memory the "hot" system. This neurobiological distinction between declarative memories and emotional memories, or, schematically, between hippocampus and amygdala, has considerable explanatory power. It may illuminate several issues relevant to psychoanalysis, including, perhaps, my patient Tara's dream, and the elusive phenomenology of PTSD. I will now describe these two key elements in some detail.

The hippocampus is a slender, elongated cluster of neurons that is located in the inner side of the temporal lobes of the brain. Its shape reminded brain anatomists of a sea horse, and it was named after a Greek mythological horse-shaped sea monster. It is extensively connected to many other parts of the brain. We all have two hippocampi, one on each side of our brain,

and total destruction of one hippocampus does not cause a severe memory deficit. This is perhaps the reason why the critical role of the hippocampus in memory formation was not appreciated in full until the middle of this century, when a patient lost both his hippocampi due to a medical intervention.

In 1954, both medial temporal lobes of a young man known as H.M. were surgically removed, in an effort to control his severe, intractable epilepsy. The operation, as they say, was a success. The frequency of his seizures was much reduced. But as Brenda Milner discovered, H.M.'s life was devastated by his surgery: he had completely lost the ability to acquire and retain new conscious memories. From that point in his life, he could not hold on to the memory of anyone he met, or have a lasting conscious memory of anything he saw. Except for memories of people and events that were laid down before his operation, he was doomed to become a perpetual "clean slate." But as Milner discovered later, H.M. was still able to learn and retain new motor and perceptual skills, like playing certain games, without, of course, ever remembering that he had learned them.

The second structure relevant to this discussion is the amygdala, which is the Greek word for almond. It got its name because it resembles an almond in its shape and size. This group of neurons lies immediately in front of the hippocampus, at the tip of the medial part of the temporal lobe, and we have one on each side of the brain. The amygdala is also richly connected to many other parts of the brain, and is believed to mediate the emotional experience of fear. Joseph LeDoux and others have shown that the amygdala is a central coordinator of fear and anxiety in the brain, and that it is required in order to make an association between a frightening experience and a neutral one, a very important issue for the phenomenology of PTSD.

To follow an example given by Joe LeDoux, suppose that you are in a car accident in which you are injured and traumatized, and the car's horn gets stuck and doesn't turn off. You are trapped in the car, terrified and in pain, and at the same time you keep hearing the horn go on and on. Your amygdala will associate the sound of the horn with the traumatic experience of the accident. From that day on, whenever you hear a car's horn on the road, you will immediately re-experience the sensation of fear, and its physical manifestations: an increase in heart rate, blood pressure, perspiration, muscle tension, etc.

At this point, we should note two important issues. The first is that this association and re-experiencing of fear, which is mediated by the amygdala, is not dependent on the hippocampus: it acts through neural pathways from the sensory thalamus to the amygdala that bypass the hippocampus and the neocortex altogether. What this means is that for the sound of a horn to produce intense anxiety, you do not need to have a conscious recollection of the accident. You can of course still be reminded of the accident, through the declarative memory system mediated by the hippocampus. But the fear response to the sound of the horn does not require any conscious recollection of the accident, and may occur without it. This is a crucial point, and we will return to it later.

The second issue is that the amygdala is virtually incapable of forgetting. Once the neural connections that mediate a traumatic association are made in the brain, they remain intact for the lifetime of the organism. This is a unique feature of the amygdala. The "cool" hippocampus, which is exquisitely refined in its ability to remember details and nuances of events, is also terribly forgetful. The "hot" amygdala, in contrast, is very simple and unrefined in its repertoire, but it never forgets an association.

**The effect of traumatic stress on declarative memory**

Now I would like to return for a moment to Freud and the trouble he got into in 1896. One reason his theory on the etiology of hysteria was met with an “icy reception,” and which might have caused Freud to doubt himself in the years to come, was that his patients’ memories of their traumatic abuse were a bit strange, to say the least: they re-emerged, young and fresh, like Sleeping Beauty, after lying dormant for decades (as we will see, the same is true for Tara’s traumatic experience, which was awakened with a kiss). The memories were incoherent, patchy, vague, and often contradictory. In short, Freud’s patients were very unreliable witnesses. And this brings us to another piece of contemporary neurobiology which might be relevant to the psychoanalytic understanding of traumatic memories: the effect of traumatic stress on declarative memory.

Traumatic stress may have dual, contradictory effects on explicit memories: it can make them stronger, as in the phenomenon of “flashbulb memories,” or it can make them disappear, as in traumatic amnesia. The classic example of “flashbulb memories” is that everybody remembers where they were when they first heard that JFK was shot. I was too young at the time to remember much of anything, and we will discuss infantile amnesia later in this paper. But I still remember vividly the corridor in which I stood when I was told that my father died, although that took place over 25 years ago.

The work of James McGaugh and his colleagues revealed that the enhanced remembering of explicit events during emotionally stressful moments is mediated by adrenaline and noradrenaline, a stress hormone and a stress-related neurotransmitter which are indirectly activated by the amygdala during times of stress and danger. Adrenaline and noradrenaline then activate both the hippocampus and the amygdala.

There was previous evidence that adrenaline played this role in animals, and in order to test its effect in humans, McGaugh and his colleagues devised an ingenious experiment: they designed two slide shows that both told a short story, with matching narratives, using almost the same words. The narratives and the slides were as similar as possible, with one important difference: in one story, a boy watches a disaster drill, in which actors are dressed up to look as if they were severely wounded, and are evacuated to a hospital. In the hospital, a surgical team practices disaster drill procedures all day. In the other story, the boy gets caught in an accident, becomes critically injured, and is rushed to a hospital, where a surgical team struggles to save his life all day. Groups of healthy subjects were shown either one or the other story, and one week later, they were all interviewed to see how much of the story they still remembered. And here’s where adrenaline comes in: in each group of subjects who watched either story, half were given a placebo, and half were given Inderal, a drug used to treat hypertension and certain cardiac conditions by blocking the beta-receptors for adrenaline and noradrenaline. Inderal crosses the blood-brain barrier quickly, and it is used by psychiatrists to treat stage fright and other phobic conditions.

Among the subjects who received the placebo, those who viewed the traumatic accident story remembered better than those who viewed the disaster drill story, although the words and the images were almost identical. The enhanced explicit memory was the result of the different emotional meaning of the experience, something that should not come as a surprise to psychoanalysts. But among the subjects who received Inderal, there was no difference in explicit recall between those who viewed the traumatic story and those who viewed the drill story. This suggests that adrenaline and noradrenaline, released during trauma, fear, and danger, may be responsible for “flashbulb memories.”

But how are traumatic memories forgotten? How can something as vivid and as traumatic as sexual abuse, which is highly likely to generate an adrenaline rush, be forgotten, only to emerge years later as a recovered memory? And what is responsible for dissociation, the

disconnection between memory of facts and memory of feelings, which was so typical of hysterics a hundred years ago and is still typical of trauma victims today? These sneaky qualities of traumatic memories might have been what led the eminent neurologist Krafft-Ebing, who chaired Freud's unfortunate presentation, to call Freud's claim that hysterical women were sexually abused as children and then repressed their memories "a scientific fairy tale."

The concept of repression is a Freudian legacy that is central to psychoanalysis to this day. Even after Freud abandoned his traumatic theory of neurosis, he retained the notion that repression of unacceptable memories, wishes, and urges is responsible for neurotic symptoms. Most psychoanalysts probably still find this idea clinically useful. I certainly do. But repression of traumatic memories can no longer be maintained as a psychological concept independent of the brain. Knowledge of the relevant neurobiology may modify our understanding of the mechanism of repression. This, in turn, may modify our therapeutic technique.

In an influential paper, Jacobs and Nadel suggested a neurobiological mechanism for repression and dissociation of traumatic memories which is based on the action of another stress-related hormone: cortisol. Cortisol is secreted into the bloodstream by the adrenal glands, in response to a hormonal signal from the pituitary gland. This hormonal signal is triggered at times of stress by the amygdala, through the hypothalamus. What this means is that at times of fear and trauma, both adrenaline release and cortisol release are mediated by the amygdala. These stress hormones act in tandem to prepare the organism for a fight-or-flight response. But over the last twenty years it has become increasingly clear that excess cortisol may have far-reaching effects on memory. High levels of cortisol, like those present during traumatic stress, have opposite effects on the amygdala and the hippocampus: As LeDoux has shown, cortisol increases activity in the amygdala. In contrast, activity in the hippocampus first increases and then dramatically decreases as cortisol levels increase.

What this means is that a high level of traumatic stress can cause a partial or complete shut-down of the hippocampus, and with it, a shut-down of declarative memory formation. As a consequence, very traumatic events may be remembered in a patchy, incomplete way, or not at all. But keep in mind that the amygdala is not shut down by traumatic stress. On the contrary, its activity is enhanced. This may result in situations in which explicit memory of a traumatic event is hazy or absent, but its emotional memory, with all the fear and terror associated with it, may be present for a lifetime, waiting to re-emerge on cue. When the traumatized person encounters a traumatic stimulus, like a car horn going on and on in LeDoux's example, he might experience a surge of intense anxiety, without any conscious recall of what happened to him. In this way, neurobiology may account for the clinical phenomenon of dissociation: a pathological disconnection between facts and feelings. Furthermore, under conditions where the declarative memory of a traumatic experience is vague and incomplete, it might be recalled as an explicit memory only under circumstances of greater emotional intensity, which may occur during the course of psychotherapy, thus leading to a recovered memory which emerged during therapy.

### **Re-evaluating repressed memories**

Until this point, we have reviewed the anatomy of explicit and emotional memory, and explored a neurobiological mechanism for "flashbulb memories" and for repressed, recovered memories. We have also seen how the neuroanatomy of memory and the physiology of stress may lead to a chronic dissociation between facts and feelings in the wake of a traumatic experience. Thus, we are on our way toward elucidating a neurobiology of PTSD. But does that mean that everyone who reports having been abused as a child is telling the truth? What about false memories? Do they exist? And if so, how do we distinguish them from real memories? Freud ended up feeling that he couldn't. He did not doubt the veracity of those

childhood abuse memories for which he obtained parallel history from an adult family member, but he came to disbelieve many of the others. In his “Autobiographical Study”, Freud looked back and reflected on his own career. In describing the unhappy events of the spring of 1896, he said:

...I was at last obliged to recognize that these scenes of seduction have never taken place, and that they were only phantasies which my patients have made up or which I myself had perhaps forced on them... (Freud, 1925, p. 34)

This is exactly the claim made by the “False Memory Syndrome Foundation”, and it is made all the more poignant because, as I can confirm from my own experience, and as countless clinicians have observed over the last hundred years, patients who present with or recover memories of childhood abuse are often extremely suggestible. In addition, as we saw earlier, their testimonies are often patchy, self-contradictory, and vague. Freud and his first patients invented psychoanalysis as a non-manipulative alternative to the prevailing treatment for hysteria: suggestion. But suggestion continues to haunt the analytic encounter. Suggestion in psychoanalysis can be subtle, it can occur without intent, and the therapist himself may be unaware of what he might be inducing.

An honest and startling example of this is a case report by Person and Klar. Another report by Michael Good (1994) describes a documented case of autosuggestion. In this case, memory of brutal and abusive gynecological surgery during childhood, which unfolded as a key trauma during the course of adult psychoanalysis, turned out to be false, thus changing the course and perhaps the outcome of the analysis. It seems as if people who report traumatic memories of abuse often confabulate, that is, they “fill in the blanks” in their declarative memory, in the same way that patients with dementia do. Therefore, in the case of recovered memories, the accuracy of a specific recovered incident may be low. Jacobs and his colleagues presented a model which may account for the variable veracity of recovered memories.

A recent neurobiological discovery, which is relevant to the psychoanalytic understanding of false memory, is that functional brain imaging can tease apart true from false memories, because they are associated with different patterns of cerebral activity, at least in some instances. Daniel Schacter and his colleagues gave their subjects lists of words to study, while imaging their cerebral blood flow with <sup>15</sup>O water in a positron emission tomography (PET) scanner. Cerebral blood flow, as imaged in PET, is a time-sensitive correlate of regional brain activity. The subjects in Schacter’s study have heard some but not other words on the list during a training session that preceded the scan, and were asked to indicate whether the words they now saw on the list had been heard before. Schacter and his colleagues tricked their subjects into false memories in the following way: the list of words that the subjects heard during the training session was rigged with words that were semantically related to another word, which was put on the test list without being presented earlier. For example, the training session would include the words “chocolate,” “eat,” “taste,” “sugar,” and “candy.” The test list would then include the word “sweet”, which was not presented before. Most subjects would then confidently and falsely “remember” that they had heard “sweet” before.

When comparing regional brain activity during true recall versus false recall, the investigators found in both cases an increase in activity in the region of the left hippocampus, indicating that it may participate in false as well as in true memory. This may account for the fact that false memories seem perfectly real to the people who have them: they are “recalled” by the hippocampus, just like real memories. But only true memories were associated with increased activity in the left temporoparietal cortex, where verbal auditory information is processed. Thus, true and false memories appear to be associated with differential but partially overlapping neuronal activity patterns, in a way that might account for an individual’s subjective conviction in the veracity of his false memories. It is tempting to envision that we

will soon be able to apply this technique to memories of sexual abuse, but Schacter himself cautions that this goal is probably far away. In reviewing his data, Schacter adds that despite the fact that the memories he elicited were false, they related to true occurrences: for example, the subjects did indeed hear words with a theme of food and taste, just not the word “sweet.” In analogy, Schacter suggests that false recovered memories may still capture something important about the past, and should not be dismissed, even though they may not accurately reflect historical reality.

### **Amnesia and the hippocampus**

Before we return to my patient Tara and conclude this discussion, I would like to make two observations about amnesia and its relation to repression and to the hippocampus. First, I would like to use our understanding of the brain’s memory systems in order to discuss how we might re-establish a basic tenet of psychoanalysis on a more current biological foundation. Freud pointed our attention to the phenomenon of infantile amnesia: the fact that most people do not have consistent explicit memories from the time before they were three or four years old. Freud attributed this, of course, to a massive repression which accompanies the resolution of the Oedipus complex. Jacobs and Nadel proposed that the reason for this amnesia is the delayed maturation of the hippocampus, the recorder of declarative memory in older children and adults, which does not become fully functional until age three or four. Thus, infantile amnesia is largely driven by brain development, and not by conflict. But during the course of normal development, the amygdala becomes functional before the hippocampus, in rats and probably in humans too. What this means is that traumatic experiences early in life may be remembered as emotional memories only, without ever being recalled as explicit memories. As we have seen, people with such experiences early in life are at risk for developing false memories, in an attempt to fill in the gaps that stretch between what they feel and what they know about their own past.

A second neurobiological observation about amnesia and PTSD is that the stress of repeated or ongoing psychogenic trauma actually shrinks the hippocampus, injures or kills its neurons, and may disrupt its function chronically. We have already remarked on the fact that victims of severe psychogenic trauma make unreliable witnesses, because their memories are often patchy and chaotic. Douglas Bremner and his colleagues imaged the brains of patients with combat-related and childhood abuse-related PTSD with magnetic resonance imaging (MRI), measured the volume of their brain regions, and tested their declarative memory. They found that patients with PTSD have an 8 percent reduction in the volume of their right hippocampus, and have a worse declarative memory overall. They also found that in combat veterans, the severity of hippocampal size deficit correlated with the length of combat exposure: the longer the exposure to battle trauma, the worse the damage to the hippocampus. This finding agrees well with the clinical observation that patients with PTSD have a hard time remembering, both in therapy and in the rest of their lives. Classical psychoanalytic theory has ascribed this to conflict-driven repression. Contemporary neurobiology seems to suggest that this may be the result of chronic stress-driven neuronal injury.

### **Deciphering Tara's dream**

We will now return to Tara and her dream, in which a snake tickled her, suffocated her, and killed her, and was then chopped up and discarded by her mother, in an attempt to conceal what had happened.

Tara has a history of childhood sexual abuse that was “forgotten” and then recovered in her adulthood. She remembered the incidents, which occurred when she was about four years old, while she was in law school, a few years before she began therapy with me. Her recollections were confirmed by her brother, who was three years older than Tara, and was with her when these incidents took place. At the time, they were both staying with friends of the family for

the summer, while their parents were away on an extended vacation. Tara was raped repeatedly during the summer by the friends' adolescent son. Like Freud's patients, Tara did not want to talk about her history of abuse with me, and only reported it in response to a direct question during our initial consultation. Like Freud's patients, she was terribly ashamed of it, and her memories of it were patchy and disjointed. Whenever the subject would come up in therapy, Tara would feel "numb" and dissociate in my office. She would then often skip the next session. She is unwilling and probably unable to work overtly in the transference at this point in the treatment.

It is not clear to Tara or to me whether her mother, or the mother of that family, whom Tara loved dearly, tried to cover up the rape, as her dream might imply. It is also not clear to me whether the dream represents her unconscious concern that I, her transference mother, might try to cover up the fact that something terrible happened to her on the night of the dream, when she and Jeff kissed in her apartment, thus triggering in her a response of panic and depression. At the same time, I am aware of Tara's aggressive, destructive wishes that might have found their way into her dream. But we are now back to the familiar ground of psychoanalytic therapy.

In conclusion, I would like to discuss how all that we have reviewed might be relevant to patients like Tara, and to describe how I used it in her treatment. Neurobiology informed and modified my treatment of Tara in four ways: first, in psychoanalytic parlance, it helped me shift from a conflict model to a model that allowed for both conflict and deficit. Specifically, I allowed for the possibility that many or all of Tara's difficulties in remembering her past were due to chronic hippocampal damage, and hence would not be reversed by even the most illuminating interpretation. This prevented me from making unhelpful and incorrect interpretations that would have led only to frustration and anger.

Second, neurobiological insight into the effects of stress on memory formation in patients with a history of trauma dictated a shift in the timing of my therapeutic interventions with Tara: like generations of analysts before me, I was trained to "go for the affect" and to "strike while the iron is hot" during therapy with neurotic patients. In other words, analysts attempt to deliver their interpretations during or immediately after moments of increased emotional tone in the session. I believe that this is generally useful, especially in patients with obsessive character traits. It may prevent intellectualization and lead to meaningful, mutative "moments of meeting" in therapy. However, patients with PTSD present to therapy with hippocampi that are already compromised, and with hypersensitive amygdala. In this setting, an exaggerated cortisol response to emotional stress, triggered by their amygdala, may overwhelm their hippocampi, and render them incapable of retaining even the best interpretation. Furthermore, excessive negative affect during a session may lead to nothing more than a traumatic repetition of the original insult in a patient suffering from PTSD. Thus, with patients like Tara, it may actually be better to "strike while the iron is cold," and deliver interpretations during moments of emotional calm, when they can be processed and remembered. As Richard Brockman pointed out in his thoughtful book *A Map of the Mind*, a therapist can and should control the level of affect during a session to suit the patient's diagnosis and current clinical status. Brockman also suggests that in doing so, the therapist should take into account the interplay between hippocampus and amygdala.

Third, the medications I used to treat Tara, Zoloft and Effexor, are both serotonin reuptake inhibitors (SRI's). Serotonin is important for the integrity of hippocampal neurons. Preliminary research suggests that SRI's may protect hippocampal neurons against damaging morphological changes induced by stress and mediated by cortisol. Gould and her colleagues also found that neurons continue to proliferate in the adult hippocampus, and that social stress inhibits their growth. What this implies is that the adult hippocampus may be capable of regeneration and healing under the right conditions, and that continued stress may

interfere with this process. It is tempting to speculate that psychotherapy and antidepressants may both facilitate psychological healing by making neuronal regeneration possible.

Fourth, contemporary neurobiology allowed me to first understand and then explain to Tara some of what she was going through, and to help her make sense of her chaotic inner world. I was able to tell her that the physical experience of kissing Jeff and feeling close to him may have triggered her emotional memory of the abuse she suffered as a four-year-old girl, without a corresponding explicit recollection. In other words, her amygdala remembered vividly what her hippocampus barely recalled. She therefore felt terrified, guilty, and ashamed after Jeff left, and had no idea why. Like a survivor of a car accident who is terrified every time he hears a car horn, Tara had a response of anxiety, shame, and self-loathing to her experience of physical and emotional closeness with Jeff. Her dream, still the royal road to her unconscious, revived the memory of that trauma and its aftermath in a symbolic form. Telling her all that relieved some of Tara's anxiety, and gave her an intellectualized model that she could hold on to. In addition, it helped her become aware of her unconscious wish to retaliate, detach, and withdraw from Jeff, which related to her original trauma, and which destroyed some of her previous relationships with men.

I was aware, throughout my work with her, that Tara's difficulties with reconstructing her own past might be viewed from two distinct vantage points. Psychologically, they may be the result of conflict-driven repression of emotionally painful memories. Neurobiologically, they may be the result of a damaged hippocampus coupled with a hypersensitive amygdala. These two vantage points are not necessarily mutually exclusive. Rather, they might, in certain circumstances, describe the same phenomenon in two different languages.

Whether one or the other model is more applicable in any clinical situation might be a matter of degrees: Denial and repression of emotionally painful or unacceptable facts is a universal human tendency, quantified and documented in the general psychological literature. One example is "positivity bias," the normal tendency to attribute mostly positive qualities to oneself and deny one's own negative qualities, even in the face of objective evidence to the contrary. Strong positivity bias has been found in normal populations of toddlers, adolescents, and adults, and in diverse cultures. It is unlikely that most instances of such repression of unacceptable facts in everyday life are the result of hippocampal damage. By definition, this "normal" repression also has a biological explanation, but it probably involves complex neocortical interactions, and we are still unaware of it. Our current stress-based neurobiological model might not increase our understanding or enhance our treatment of such "normal" cases of repression. The psychoanalytic conflict model is applicable to this form of repression, however, and may lead to clinically useful predictions and treatment options.

In contrast, the pervasive memory disturbances found in PTSD are probably better accounted for by the neurobiological model, which also suggests treatment modalities other than interpretation of unconscious conflict. Blind application of the psychoanalytic model to people like Tara may be at best unhelpful and at worst damaging. Most cases of repression seen in everyday practice probably fall somewhere in between Tara's forgotten trauma and "normal" repression. The clinician must therefore decide which model to apply at any given time. Flexibility, empathy, and knowledge of basic neurobiology are as essential to us today as they were when Freud first listened to his patients' troubled accounts of their past.

Tara and patients like her will continue to consult analysts and therapists, rather than neurobiologists, to help them find their way to better lives. But psychoanalysts are obliged to listen carefully to what neurobiology has to say, and adjust their theories accordingly. The house of psychoanalysis is still, as Eric Kandel asserted, "the most coherent and intellectually

satisfying view of the mind.” But this house will prosper only if it rests on solid foundations, rooted in current biological research.