DOES PHARMACOLOGICALLY-ALTERED MEMORY CHANGE PERSONAL IDENTITY?

SISTER RENÉE MIRKES, OSF, PHD

The National Institutes of Health reports that some 7.7 million American adults are diagnosed with post-traumatic stress disorder (PTSD) annually.[1] The debilitating symptoms of this disease hinder daily living, diminish quality of life, and, sometimes in chronic cases, last the lifetime of the traumatized individual. Unfortunately, a large population of PTSD patients receives little or no relief from the conventional treatment of psychotherapy and/or antidepressants.[2] It is understandable, then, that these patients and their caregivers welcome the "potential lifeline"[3] of a reliable and cost-effective drug like propranolol that could attenuate the hyper-emotionality of traumatic memory.

However, despite encouraging results from preliminary clinical studies, some have raised ethical concerns about using propranolol to treat PTSD. Here I respond to what I consider the most substantive of these ethics objections, viz., that attenuating fear memory pharmacologically could alter the personal identity of the PTSD patient. I postulate that the personal identity objection to drug-induced memory alteration in the context of PTSD lacks medical, ethical, and philosophical ballast for several reasons. First, the emotional and physiological reactions, as well as the neurological profiles, of PTSD patients differ dramatically from those of healthy trauma survivors and are oftentimes resistant to traditional psychotherapy. Second, propranolol alters a solitary traumatic memory and its hyperemotional expression in a way that biochemically mimics the memory extinction process of healthy trauma survivors. And, third, propranolol-induced attenuation of a traumatic memory constitutes a positive accidental change to the personality of PTSD patients, enabling them to integrate their life-changing experience into their personal pursuit of happiness and fulfillment.

The Emotional and Behavioral Profile of a PTSD Patient

In 1980, the American Psychiatric Association recognized PTSD as a diagnosable disorder in its *Diagnostic and Statistical Manual of Mental Disorders* [3rd edition].[4] The first criterion for the diagnosis of PTSD is that the person has experienced, witnessed, or been confronted with a traumatic event—military combat,[5] rape, murder, mugging, suicide bombing, car accident, natural disaster,[6] abduction, or terrorism[7]—that involved "actual or threatened death or serious injury . . . to self or others."

Second, those who are emotionally traumatized respond to the original incident with "fear, helplessness or horror" and, subsequent to the initial disturbance, re-experience the episode and its emotional fear in one (or more) of the following ways:

> 1) recurrent and intrusive distressing recollections of the events, including images, thoughts, or perceptions; 2) recurrent distressing dreams of the event; 3) acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback

episodes, including those that occur on awakening or when intoxicated); 4) intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

Third, the behavioral pathology of the PTSD patient manifests itself in persistent avoidance of "thoughts, feelings or conversations" associated with the original trauma and in "numbing of general responsiveness" by three (or more) of the following:

1) efforts to avoid thoughts, feelings, or conversations associated with the trauma; 2) efforts to avoid activities, places, or people that arouse recollections of the trauma; 3) inability to recall an important aspect of the trauma; 4) markedly diminished interest or participation in significant activities; 5) feeling of detachment or estrangement from others; 6) restricted range of affect (e.g., unable to have loving feelings); 7) sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span).

Fourth, those diagnosed with PTSD also persistently manifest behavioral symptoms of increased arousal as indicated by two (or more) of the following: "difficulty falling asleep; irritability or outbursts of anger; difficulty concentrating, hyper-vigilance, or exaggerated startle response."

Fifth, trauma survivors are diagnosed with PTSD if the duration of the disturbance is more than one month and if "[t]he disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of interpersonal behavior." The personal and social impairment can take many forms: drug abuse, alcoholism, marital problems, unemployment[8], and suicide.[9]

Firemen and policemen, rescue workers[10], SWAT teams, members of the military, and individuals whose jobs directly involve them in traumatic incidents are most at risk for PTSD.[11] For anyone within this "high risk" profile, the three most important factors affecting the likelihood of being diagnosed with the disorder[12] are "the severity, duration, and proximity of [the] individual's exposure to the [emotionally-charged, life-threatening] event."[13] A trauma survivor has acute PTSD if the symptoms just described last less than three months, or chronic PTSD if the symptoms last three or more months. Sometimes, the onset of PTSD is delayed, in which case the symptomatic behavior outlined above occurs six or more months after the horrific event.

Various forms of cognitive therapy involving memory extinction training[14] are the traditional means of enabling PTSD patients "to build new [mental] associations and extinguish the bad memory link." Unfortunately, the traditional therapeutic approach does not help a third of PTSD patients and does not produce consistent results in the remaining two-thirds of stress disorder patients. Old, bad memories return and often are as virulent as when previously experienced.

How the Brains of PTSD Survivors Process Life-threatening Events

A medieval custom provides an unusual bit of historical evidence for the direct way in which highly emotional experiences lead to long-term memory formation.[15] In lieu of keeping written records of important events—land grants, weddings, funerals, negotiations between landed gentry—the feudal lords of the 5th to the early 16th century selected a young child around seven years of age, instructed him to observe carefully

the important political/legal/social event at hand, and then threw the child into a river. With this rather grisly—and definitely traumatic—custom, "the memory of the event would be impressed on the child and the record of the event maintained for the child's lifetime."[16]

The focus of a significant portion of the last fifty years of neural and memory research is its investigation of the neurobiological processes that explain why this medieval memory-aid worked so effectively. (Or, apposite to us 21st century Americans: why the events of 9/11 are so indelibly imprinted on our individual and collective long-term memory.) In sum, a considerable segment of memory research[17] has sought to explain how and why highly emotional events make for strong experiential memories.

Roger Pitman, a psychiatrist at Harvard Medical School and a memory researcher, describes the "promiscuous [brain] system"[18] that encoded the lasting memory of the medieval child [19] and your enduring memory of 9/11.

> Stimuli from your sense organs are continuously entering your brain and converging on the thalamus, a clearing house for the senses. From there, the information is quickly dispatched along an express route to an almond-shaped region of the forebrain called the amygdala for a crude assessment of the 'emotional quality' of the stimuli. If the amygdala recognizes a potentially threatening component—such as the screeching brakes of a large vehicle or a curved shape on the ground that could be a snake—it triggers the body's stress responses: a typical "fight or flight" rush of adrenaline and noradrenaline. . . . The amygdala triggers a rapid fear response to allow the body to take evasive action. Simultaneously, . . . other paths take signals from the thalamus to higher areas of the brain for more considered analysis of whether the stimuli represent a threat. If, for example, the curve turns out to be a piece of hosepipe in the grass, then the prefrontal cortex reins in the amygdala response. But if the stimuli turn out to represent a genuine threat, adrenaline and noradrenaline trigger a cascade of reactions in the amygdala, which then instructs the hippocampus—the brain's memory centre—to process the memory of those fear-inducing stimuli in a special way, imprinting them deeper than usual.[20]

But what occurs in the brains of people prone to PTSD when they experience highly emotional events? For this at-risk population, memory research has helped to identify the direct causal links between (1) their malfunctioning neuromodulatory processes that *over*-imprint emotionally charged events into their long term memory, (2) their *hyper*-emotional/physiological arousal responses, and (3) their *pathological* behavioral profile that is consistent with the eventual diagnosis of PTSD.

After the research dust settles, we learn that at least two neural mechanisms are malfunctioning during and after a traumatic experience in the brain of a person predisposed to PTSD.

First, there is a problem with consolidation of the highly emotional memory—the process of strengthening the original memory trace and moving it from short-term to long-term storage.[21] As Pitman explains, the neural site for the psychiatric pathology of PTSD starts deep in the limbic system of the brain, in the almond-shaped region called the amygdala. For the person predisposed to a traumatic stress disorder, the life-threatening event *over*stimulates the brain's endogenous stress hormones; the excessive

release of adrenaline (epinephrine) from the adrenal gland triggers the excessive release of noradrenaline (norepinephrine) in the amygdala. As this "noradrenergic hyperactivity" [22] floods the basolateral amygdala[23], it produces *hyper*emotional arousal in the PTSD-prone victim. In other words, over-activation of the amygdala by stress hormones during highly emotional events causes a serious problem in the way the hippocampus of PTSD-prone trauma victims encodes the tragic event. The traumatic memory is *over*consolidated, that is, *over*-imbedded in the long-term memory of the PTSD subject.

As it turns out, the traumatic memory is not only persistent, but also self-reinforcing. Any number of internal or external stimuli may "trigger" the return of the memory. Research has shown that, during its reconsolidation phase, the memory assumes a labile or unstable state and, as such, is disposed to being altered, i.e., re-made or re-consolidated by the neurotransmitters epinephrine and norepinephrine. Unfortunately, every retrieval of the traumatic memory in response to a sound, a smell, a weather pattern, an anniversary of the event, or the place where the original trauma occurred, sets up a *vicious* "feedback loop" [24] in the brain of a PTSD patient. Remembering the life-changing event causes a *further* release of stress hormones that, in turn, causes *further* overconsolidation of the memory. Just as in the memory's original encoding, so in its reconsolidation phase: repeated stress hormone *hyper*activity in the brain of the person prone to PTSD orchestrates the memory's pathological imprinting. And, the accumulative effect of repeated intrusive flashbacks or debilitating nightmares—in short, *over*consolidated-fear-memory-upon-*over*consolidated-fear-memory—transmutes the survivor's subclinical stress disorder into clinical PTSD.

Second, from observation of the abnormal behavior patterns of PTSD patients, we deduce that the natural memory extinction process—the neurologically-based mechanism that, over time, tempers the emotional impact of the traumatic memory and weakens the memory of the event itself—is either malfunctioning or arrested by the overstimulation from stress hormones. In short, the excessively strong fear memory of the PTSD patient, resistant as it is to the normal memory attenuation process, becomes "a 'black hole' in the mental life of the PTSD patient, attracting all associations to it."[25] Predictably, PTSD survivors, crippled by fear and anxiety and paralyzed by intrusive memories of the event, "become 'stuck' on their trauma, 'reliving it in thoughts, feelings, actions or images.'" Developing a sense of helplessness that can permanently change their ability to deal with stress and human relationships, PTSD patients undergo a negative alteration not only of their self-concept but also of "[their] view of the world as a manageable place."[26] The unhealthy trauma survivor, unable to get beyond the original disturbance and its concomitant mental pain, lacks the power to return to a normal personal and interpersonal life.

How the Brains of Healthy Trauma Survivors Process Life-threatening Events

Why do healthy trauma survivors not evince the abnormal behavioral profile of PTSD survivors? [27] The short answer is that they have learned to cope. That is to say, they have learned to manage the fear and a myriad of negative emotional reactions not only to memories of the original trauma but also to subsequent events of their life that are highly stressful.

Recent research in the neurochemistry of memory formation helps us understand the chain of neural-coping mechanisms that account for the emotional behavior and physiological reactions of persons who outlive their traumatic events. First, their basolateral amygdala is not over-stimulated by the cascading release of excessively strong stress hormones at the memory's consolidation or reconsolidation phase. Second—and as a direct result of the first— the healthy survivor does not evince the hyper-physiological reactions (greatly increased heart rate and blood pressure) and their psychic correlate (excessive emotional arousal), whenever the original event is recalled in response to trigger events. Third, with the passage of time, healthy survivors of trauma learn that the internal and external stimuli of trigger events are not a threat. Their normally functioning memory extinction process helps their brains "make new pathways that override the old one, though they don't erase it."[28] And their prefrontal cortex consistently reigns in the amygdala. Over time, then, with functional reconsolidation and memory extinction processes in place, healthy survivors tend to experience less emotional arousal when recalling the original trauma and to remember the event itself with less clarity and force.[29]

In other words, trauma survivors who are not prone to PTSD are able to conserve and to reinforce their traumatic memory at just the "right pitch":

> Neither *too much*, engulfing [them] in trivia or imprisoning [them] in the past, nor *too little,* losing track of life's defining moments or of knowledge needed for everyday life; neither with *too much emotion*, allowing past misfortunes to haunt or consume [them], nor with *too little emotion*, recalling what is joyful, or horrible, or inconsequential, all with the same monotone affect.[30]

Propranolol-Induced Alteration of Traumatic Memories in PTSD Patients

Neuroscientists have been investigating two critical questions regarding memory alteration in PTSD patients. The first question: Would a course of propranolol administered shortly after an acute traumatic event have the same secondary preventive effect as it did on the memories of rats,[31] viz., to reduce or prevent the physiological symptoms of PTSD?

Before we answer, we need to review relevant neurological facts. In the nucleus of the amygdala of the PTSD-prone brain, there is excessive noradrenergic activity during a highly emotional, life-threatening event. This abnormal neurochemistry is responsible for enhanced memory consolidation that begins shortly after the life-changing experience. It was precisely during the consolidation phase that researchers saw their first window of opportunity for the use of memory-blunting drugs such as propranolol.

A small,[32] but important, randomized, double blind human study[33] provides a positive answer to the first question. Propranolol (40 mg, four times daily for ten days) was randomly administered within six hours of the traumatic event to half of the forty-one initial trauma-survivor participants; a placebo was given to the remaining half. Afterward, each participant was taped while verbally describing his/her trauma. Three months later, as each completer-participant listened to the taped account and simultaneously imagined his/her traumatic event, investigators monitored the participant's heart rate and other physiological reactions. None of the eight propranolol

subjects, but six of the fourteen placebo subjects, "were physiologic responders during script-driven imagery of the traumatic event." It appears that, in the former group of trauma survivors, propranolol blocked over-consolidation of the traumatic memory (by blocking the noradrenergic hyperactivity in the amygdala) with the end result that the propranolol users (just like the rats in previous animal studies) did not present with the physiologic symptoms of increased heart rate and blood pressure typical of PTSD. This and other studies suggest that "acute, post-trauma propranolol may have a preventive effect on subsequent PTSD."

The second question: Would the use of propranolol to block the noradrenergic stimulus in PTSD patients following reactivation of a fear memory have the same result as it did in studies involving rats,[34] viz., to disrupt the reconsolidation of the fear memory? The idea of using beta-blockers to attenuate traumatic memories originated in animal studies that demonstrated the surprising pliability of consolidated memories when recalled.[35] This discovery prompted memory investigators to conduct human trials to test the hypothesis that memories assume a labile state (reconsolidation) when "recalled under emotive conditions."[36] Human test results suggest that when propranolol is administered while the participant is recalling the memory in response to internal or external stimuli the beta-blocker interferes with the memory's reconsolidation "such that an altered version is put back in storage. . . ." all the while blocking "the neurotransmitters [epinephrine and norepinephrine] involved in laying down memories."[37] In a Dutch trial,[38] propranolol not only attenuated the behavioral expression of the fear memory but also prevented its return. Another study[39] concluded that blocking "pre-synaptic norepinephrine release with a beta-adrenergic antagonist such as propranolol [administered after a PTSD patient recalls a consolidated memory] may be useful in attenuating traumatic memories, *even well-consolidated old memories* [emphasis added]."

Answering the second question in the positive, then, the results of memory alteration studies in humans have led researchers to postulate that PTSD patients could experience the same kind of propranolol-induced fear attenuation at the memory's reconsolidation phase as evidenced in animals.

Effects of Pharmacologic Memory Alteration on Personal Identity

The following citations [emphasis added] are representative examples of what I consider to be the most substantive moral objection to pharmacologically-induced memory attenuation, viz., it threatens the integrity of the traumatized person's identity.

> To some extent, these unchosen memories constrain us; though we may regret the shadows they cast over our pursuit of happiness, we cannot simply escape them while remaining *who we really are*.[40]

> The pattern of our personality is like a Persian rug. It is built one knot at a time, each woven into the others. There's a continuity to self, a sense that who we are is based upon solid, reliable experience. We build our whole interpretation and understanding of our world based upon that experience or on the accuracy of our memories. If you disrupt those memories, remove continuity, what you have is *an erosion of personhood*.[41]

> With altered memories we might feel better about ourselves, but it is not clear that *the better-feeling "we" remains the same as before*.[42]
>
> The capacity to alter or numb our remembrance of things past cuts to the heart of what it means to remember in a human way, and it is this biotechnical possibility that we focus on here. Deciding when or whether to use such biotechnical power will require that we think long and hard about what it means to remember truthfully, to live in time, and to seek happiness *without losing or abandoning our identity*.[43]
>
> But to construct the narrative of one's life, not through thought and conversation, struggle and prayer, but simply by erasing some of the materials of that life is to *risk losing what is essential to being human*.[44]
>
> Removing bad memories is not like removing a wart or a mole. It will *change our personal identity* since who we are is linked to our memories.[45]
>
> In modulating a person's memories, we are talking about nothing less than *altering the central part of what it means to be a human being*.[46]

In neglecting to state what they mean by personal identity or essential humanness, these citations have the unfortunate effect of conflating two very distinct realities: a person's essential identity (that which characterizes his personhood) and a person's accidental identity (that which characterizes his personality). Strictly speaking, the etymology of the word 'identity' dictates that the term 'personal identity' should only be used to designate the essential nature of the human person. At the root of the English word 'identity' is *idem*, a Latin word meaning "the same," as in the "state or fact of being the same" person. This etymological background suggests that one's identity—one's being, substance, or essential features—remains the same[47] despite undergoing many changes or alterations to one's accidental features, including those to one's memories, that occur over a lifetime. A person's identity, then, refers to his or her unchanging substance—a composite being who is at once *embodied*, *intelligent*, and *free*—that endures throughout a lifetime of accidental changes to his or her personality.

To avoid confusion between essential and accidental changes in the remainder of my analysis, whenever I refer to Mr. Y, a chronic PTSD sufferer and propranolol trial participant, I will use the term "personal identity" to denote his essential identity and the term "personality" to describe his accidental characteristics.

Mr. Y has undergone only one substantial or identity "change" to date, and that was at his conception, when his substance and identity came into being. And Mr. Y has only one essential identity change yet to undergo—at his death[48]—when his body will cease to be part of him, and only his spiritual soul will survive. Aside from his conception and death, all other changes, including that of propranolol-induced memory attenuation, alter Mr.Y accidentally, i.e., alter his personality.

Keeping this in mind, we can say, to the extent that any of the opening citations imply that drug-induced memory alteration is unethical because it changes Mr. Y's essential identity, the opening citations are completely in error. No drug—or, more accurately, no effect of a drug, including memory alteration—could substantially change the person taking the medication. However, if any of the opening statements object to memory

alteration because it threatens the integrity of Mr. Y's non-essential characteristics, or personality, then the validity of the claim deserves further adjudication.

Just because accidental characteristics, such as memories, are not a part of Mr. Y's personal identity does not mean they are unimportant. Nor does it mean that we need not be concerned about their deliberate and selective alteration through, for example, pharmacological means. First, Mr. Y's experiences and memories are metaphysically significant in the sense that they set him apart—individualize him—from every other person who has ever lived, who is now living, or who will ever live. It is safe to say that Mr. Y's experiences and memories of them contribute more decisively to his individuation than do physical accidents such as his hair color and skin color. Second, Mr. Y's ability to recall his experiences truthfully also has ethical implications. When Mr. Y remembers his unique experiences at just the "right pitch," he puts flesh on the bones of his individual quest to do good and avoid evil. Mr. Y strengthens his habit of prudence, for instance, by remembering his past actions that were too hasty and resulted in increased difficulties and, thus, learns to exercise more foresight in the future. A clear recall of an experience where he failed to respond appropriately to basic human drives for food, drink, or sex could contribute to Mr. Y's virtue of temperance by preparing him to respond more temperately in the future. When Mr. Y remembers that he lied to someone and realizes that, as a direct result, he has lost the person's trust, Mr. Y would be inclined not to lie again. In fact, Mr Y has acquired all the natural virtues, those habits that make him uniquely able to do good consistently in the various aspects of his life, as a direct result of reflection on his experiences recorded in his long-term memory.

As a matter of principle, then, every effort should be made to preserve Mr. Y's memories, good and bad, pleasant and unpleasant, because from and through them Mr. Y learns to advance in his quest for happiness or self-fulfillment. But the issue at the heart of the opening citations is not whether some kind of wholesale alteration of memory is ethical, nor whether drug-induced memory alteration should be used by a healthy person, nor whether it should be used frivolously. No responsible ethicist, psychiatrist, or memory researcher is suggesting that propranolol be used now (or in the future) to alter the whole fund of Mr. Y's long-term memories. Nor do medical personnel envision propranolol for healthy trauma survivors or for persons wanting to alter memories for superficial reasons. Rather, clinicians are looking to use propranolol only in chronic cases, like that of Mr. Y, in which the fear memory is harmful and maladaptive, and the psychological weight and intensity of the emotional component of a *single* traumatic memory is excessive and obsessive, barring him from remembering the event at the "right pitch" and, consequently, from personality maturation and reentry into a normal personal and social life.

So, proposing the question one more time: Does propranolol-induced alteration of a traumatic memory threaten the integrity of the personality of Mr. Y, who is diagnosed with chronic PTSD? As we have already shown, the change involved in the blunting of the pathological fear and anxiety that formerly accompanied his every recall of the traumatic experience has no effect whatsoever on Mr. Y's essential identity. Mr. Y is the same person after his participation in the clinical trial that resulted in memory alteration as he was before. And, with respect to the accidental change to Mr. Y's personality through propranolol-induced memory modulation, I have demonstrated that it is consistent both with that occurring in any healthy trauma survivor and with that occurring in PTSD

patients who are helped by traditional psychotherapy. The truth of the matter is that, freed from the shackles of excessive fear and angst, Mr. Y's personality may actually be changed for the better. Memory attenuation could restore his former healthy self-concept,[49] enabling Mr. Y to recall the traumatic incident more realistically, to assess its moral meaning more astutely, and to apply its lessons more effectively. Pharmacological alteration of his traumatic memory is no more a threat to Mr. Y's personality or character development than the natural neuromodulatory processes are to the personality of a healthy trauma survivor. Thus, whether achieved naturally, pharmacologically, or psychotherapeutically, one could argue that memory alteration is supportive of, rather than a threat to, the health and integrity of the individual's personality.

Finally, what a difference it could make if Mr. Y, as a prospective participant in a clinical trial, was exposed to the important distinction between essential and accidental identity and, then, to the reality that the accidental change that might result from his participation mimics that which occurs naturally in the brains of healthy trauma survivors. Only when Mr. Y understands that drug-induced memory attenuation does not pose a threat to the developmental integrity of his character, and might actually advance it, can he give a truly informed consent to his participation in propranolol trials.

Conclusion

This article highlights conclusions and insights that should properly frame the debate about the question of whether memory alteration changes the identity of a PTSD patient. First, the emotional and physiological reactions, as well as the neurological profiles, of PTSD patients differ dramatically from those of healthy trauma survivors, and are oftentimes resistant to traditional cognitive therapy. Second, propranolol alters a solitary traumatic memory and its hyperemotional expression in a way that biochemically mimics the normal consolidation and memory extinction processes of healthy trauma survivors. And, third, propranolol-induced attenuation of a traumatic memory, because it constitutes an accidental change enabling PTSD patients to integrate the life-threatening experience into their pursuit of happiness, has the potential to improve rather than detract from the healthy maturation of their personality. Thus, when we apply the personal identity objection to propranolol-induced memory attenuation within the context of PTSD and the correct understanding of accidental change, it does not stand.

Endnotes

1. National Institutes of Health, Fact Sheet on Post-Traumatic Stress Disorder, July 2007.
2 Typically, cognitive therapy or exposure therapy (helping people change unproductive or harmful thought patterns) or cognitive behavioral therapy (helping patients to desensitize upsetting reactions to traumatic memories) and antidepressants (selective serotonin reuptake inhibitors) are the traditional treatment options for PTSD. The spectrum of cure varies: "More than half of patients experience some improvement; few achieve a complete cure and for a large proportion nothing works" [Vince Gaia, "Rewriting your past," *New Scientist* 188(2005):3].
3 Ibid.
4 NIH Fact Sheet.
5 A Vietnam veteran eventually diagnosed with PTSD describes the initial and the immediate physiological effects from hyperemotional arousal that precipitated onset of the emotional disorder: "You have just received a signal for a hasty ambush. You sit in the elephant grass

trying to figure out your field of fire. Then you hear them coming, talking and laughing and making jokes. You hold your breath, and your heart stops. You freeze, like you can't move. These voices get louder and louder. When they get right in front of you, you can see them from the waist down, with their AKs slung. You count them as you pass. When you get to four, all shit breaks loose. You pull your trigger and hold it down. The next thing you know, you're staring at a dead Gook's feet. Your teammates are yelling, 'Get up. We gotta go!' Now your heart is pounding and you feel jittery all over, like you want to run, but there's no place to go. You stand up and see the top of the Gook's head blown off, his brains glaring in the sun. You've never seen blood and guts before. You feel sick to your stomach and in a state of shock" [cited in James L McGaugh, *Memory and Emotion* (New York: Columbia University Press) 2003:122].

6 The Asian tsunami of 2004 and Hurricane Katrina of 2007 are recent examples of traumatic natural disasters.

7 Perhaps the best perspective for understanding PTSD is through the lens of 9/11. Chances are most of us remember exactly where we were, who we were with, and from which room we viewed the first TV reports of the event. On the other hand, few of us have distinct memories about our life experiences on September 10, 2001. In the first case, the emotional fear and anxiety linked to the event of a terrorist attack on U.S. soil guaranteed that the 9/11 memory was indelibly recorded in our long-term memory bank. In the second case, our experiential memories of 9/10 were written in pencil (as short-term memory) and subsequently erased. Although feelings of sorrow, fear, and anger made it difficult for most Americans to carry out their life-as-usual activities post-9/11, most of us learned to get beyond it, learned to integrate its lessons into our long-term memory bank and to return to life's duties and responsibilities. But persons with a propensity for PTSD, or those diagnosed with PTSD—and this is the tragedy of their disease—cannot get over or beyond a seriously traumatic event such as a terrorist attack, rape, or murder.

8 One study estimates that "a person with PTSD will endure 20 years of active symptoms and will experience almost 1 day a week of work impairment, perhaps resulting in a $3 billion annual productivity loss in the U.S." [Adam J. Kolber, "Therapeutic Forgetting: The Legal and Ethical Implications of Memory Dampening," 59 *Vand L Rev* 1561(2006):4]. The Department of Veterans Affairs has raised similar concerns about the cost of treating PTSD in soldiers. It reports that veterans received PTSD benefit payments "totaling 4.3 billion in 2004, up from 1.7 billion in 1999" [last accessed on 30 July 2009 at www.ncptsd.va.gov/ptsd101/modules/Friedman%20 PTSD%20Transcript.pdf].

9 DSM-IV: 309.81, PTSD.

10 Perhaps one of the most tragic incidents leading to severe cases of PTSD involved desk people and baggage handlers who were sent to clean up body parts after the 1978 PSA plane crash. Having no formal training in rescue work and lacking the necessary coping skills to deal with a trauma of that magnitude, a large percentage of these airline personnel were subsequently diagnosed with such severe cases of PTSD that many of them were unable to work for the rest of their lives [Scott LaFee, "Blanks for the memories," *San Diego Tribune* Feb. 11, 2004 last accessed on 2 June 2009 at http://www.cognitiveliberty.org/neuro/memory_drugs_sd.html].

11 DSM-IV: 309.81, PTSD.

12 There are other factors predisposing an individual to PTSD: the neurological pathologies—first, the abnormal release of the adrenal stress hormone, adrenaline, that, in turn, triggers the over-release of noradrenaline within the amygdala both at the time of consolidation of the traumatic memory and at its reconsolidation when the memory is retrieved and, second, the memory extinction process that fails to function normally. Then there are personal and sociological factors suggesting that persons who are female, are younger to middle-aged adults, are poor and have a history of depression, drug addiction, or alcoholism are more apt to experience PTSD [DSM-IV-TR 309:81, PTSD].

13 Ibid.

14 Memory extinction training teaches PTSD patients how to replace fear memories with fearless memories. They learn to link a noxious stimulus [desert-like heat—which formerly was associated with human destruction in the Iraq conflict caused, say, by a roadside bombing] to a pleasant stimulus [flowering cactus, peaceful sunset]. This kind of training is similar to traditional treatment of human phobias where the subject is presented with the feared object, but without its associated danger ["Brain cells Related To Fear Identified, Paving The

Way For More Effective Treatment Of Post-Traumatic Stress And Other Anxiety Disorders," *Science Daily* July, 2008 last accessed on 23 June 2009 at http://www.sciencedaily.com/releases/2008/07/080710173007.htm].

15 Any information that we glean from our experiences "that may help us in the future (for instance, the downwind smell of a saber-toothed tiger) goes into long-term memory where it can last a lifetime. Long-term memory involves three processes: encoding, storage and retrieval. First, we break new concepts into their composite parts to establish meaning. Furthermore, we include the context around us as we learn a new concept, or experience another episode in our life. For example, I might encode the phrase 'delicious apple' with key descriptive ideas—red color, sweet taste, round shape, the crisp sound of a bite—and then such contextual items as 'I'm feeling good because it's a happy fall day and I'm picking apples.' Second, as we store the memory, we attach it to other related memories, like 'similar to Granny Smith apples but sweeter,' and thus, consolidate the new concept with older memories. Third, we retrieve the concept, by following some of the pointers that trace the various meaning codes and decoding the stored information to regain meaning. If I can't remember just what 'delicious apple' means, I might activate any of the pointer-hints, such as 'red' or 'picking apples.' Pointers connect with other pointers so one hint may allow me to recover the whole meaning" [April Holladay, "How does human memory work?" *USA Today* May 15, 2007 last accessed on 4 June 2009 at http://www.usatoday.com/tech/columnist/aprilholladay/2007-03-12-memory-first_N.htm].

16 McGaugh, *Memory and Emotion*, ix.

17 In his book, *Memory and Emotion*, Dr. James McGaugh explains the strides that have been made in the century-long history of memory research: "… much has been learned about the workings of memory and the brain processes that enable them. First, it was important to learn, despite centuries of skepticism, that memory can be studied objectively, using the general methods and techniques appropriate for any scientific inquiry. Next, it was essential to develop the specific methods required for investigating animal and human memory. It was also essential to discover the critical lessons provided by disorders of human memory. Finally, the development of many kinds of research techniques has enabled investigations of the brain systems and neurobiological machinery that coordinate and create fleeting or lasting representations of our experiences" [p. 138].

18 "Promiscuous system" is a term coined by neuroscientist McGaugh to explain the fact that "we do not have a single general system in our brains that is responsible for our learning and memory but many systems" [*Memory and Emotion*, x]. Cf. also JL McGaugh, "Memory consolidation and the amygdala: a systems perspective," *TRENDS in Neurosciences* 25(2002): 456-461.

19 McGaugh, *Memory and Emotion*, ix.

20 Gaia, "Rewriting your past," 4.

21 Another way of appreciating the "promiscuous" nature of the brain's capacity to encode, store, and retrieve memories is to examine the complex orchestration between various parts of the brain involved in the process of memory consolidation. "How does our brain consolidate a new short-term memory like 'delicious apple' and place it into long-term memory? We use the hippocampus, an ancient part of the cortex, to consolidate new memories. An event creates temporary links among cortex neurons. For example, 'red' gets stored in the visual area of the cortex, and the sound of a bitten apple gets stored in the auditory area. When I remember the new fact, 'delicious apple,' the new memory data converges on the hippocampus, which sends them along a path several times to strengthen the links. The information follows a path (called the Papez circuit), starting at the hippocampus, circulating through more of the limbic system (to pick up any emotional associations like 'happy fall day,' and spatial associations like 'apple orchard'), then on to various parts of the cortex, and back to the hippocampus. Making the information flow around the circuit many times strengthens the links enough that they 'stabilize,' and no longer need the hippocampus to bring the data together . . . . The strengthened memory paths, enhanced with environmental connections, become a part of long-term memory" [Holladay, "How does human memory work?" 1-2]. Memories are certainly not made instantly but are consolidated slowly over time. Moreover, "our long term memories vary in detail and in length" [McGaugh, *Memory and Emotion*, 48].

22 Noradrenergic is the adjectival designate for noradrenaline or norepinephrine. The latter is a catecholamine that has the dual role of a hormone and a neurotransmitter.

23 J Debiec and JE LeDoux, "Noradrenergic signaling in the amygdala contributes to the reconsolidation of fear memory: treatment implications for PTSD" *Ann N Y Acad Sci* 1071(2006):521-4.

24 McGaugh, *Memory and Emotion*, 122.

25 Ibid.

26 Kolber, "Therapeutic Forgetting," 4.

27 I am using the term "healthy trauma survivor" to denote the person whose neuromodulatory processes of consolidation and memory extinction are functioning normally. And, conversely, I would define PTSD survivors as unhealthy insofar as their neuromodulatory processes are operating abnormally or subnormally. PTSD patients are also physiologically and emotionally unhealthy, but these last conditions are directly caused by the neurological pathology.

28 Gaia, "Rewriting your past," 4.

29 In addition to the natural memory extinction process, all of us, including healthy survivors, selectively reconstruct our remembered experiences, in the interest of creating "a coherent life story." "Nevertheless," Adam Kolber points out, "we do not worry whether our better-feeling naturally reconstructed selves remain the same as before. It is, thus, not at all clear why we ought to revere the selective rewriting of our lives that we do without pharmaceuticals, yet be so skeptical of pharmaceutically-assisted rewriting" ["Therapeutic Forgetting," 18].

30 President's Council on Bioethics, "Beyond Therapy: Biotechnology and the Pursuit of Happiness," Washington, D.C., October, 2003:10 last accessed 12 May 2009 at http://bioethics.gov/reports/beyondtherapy/chapter5.html.

31 The idea of intervening directly in the consolidation of a memory originated in the 1990s when researchers discovered that enhancement of fear memory consolidation could be reduced by beta-blockers. Cf. L Cahill, B Prins, M Weber, JL McGaugh, "β-adrenergic activation and memory for emotional events," *Nature* 371(1994):702-704 and L Cahill, CA Pham, B Setlow, "Impaired memory consolidation in rats produced with β-adrenergic blockade," *Neurobiol Learning Memory*. 74(2000):259-266.

32 Neuroscientists engaged in memory research are unanimous in calling for more and larger propranolol studies with PTSD patients.

33 R Pitman et al., "Pilot Study of Secondary Prevention of Posttraumatic Stress Disorder with Propranolol," *Biol Psychiatry* 51(2002):189.

34 JL McGaugh, "The amygdala modulates the consolidation of memories of emotionally arousing experiences," *Annu Rev. Neurosci* 27(2004):1-28; J Debiec & JE LeDoux, "Disruption of reconsolidation but not consolidation of auditory fear conditioning by noradrenergic blockade in the amygdala," *Neuroscience* 129(2004):267-272; CE Canal & PE Gold, "Different temporal profiles of amnesia after intra-hippocampus and intra-amygdala infusions of anisomycin," *Behav Neurosci* 121(2007):732-741.

35 K Nader, GE Schafe, JE LeDoux, "Fear memories require protein synthesis in the amygdala for reconsolidation after retrieval," *Nature* 406(2000):722-726; Y Dudai, "Reconsolidation: the advantage of being refocused," *Curr Opin Neurobiol* 16(2006):174-178; NC Tronson & JR Taylor, "Molecular mechanisms of memory reconsolidation," *Nat Rev Neurosci* 8(2007):262-275; Valerie Doyere, Jacek Debiec, Marie-H Monfils, Glenn E. Schafe & Joseph E. LeDoux, "Synapse-specific reconsolidation of distinct fear memoires in the lateral amygdala," *Nature Neuroscience* published online 11 March 2007;doi:10.1038/nn1871.

36 Gaia, "Rewriting your past," 3.

37 "β-blockers tackle memories of horror," *Nature*.436(2005); JR Strawn & TD Geracioti, "Noradrenergic dysfunction and the psychopharmacology of posttraumatic stress disorder," *Depress Anxiety* 25(2008):260-71.

38 Merel Kindt, Marieke Soeter & Bram Vervliet, "Beyond extinction: erasing human fear responses and preventing the return of fear," *Nature Neuroscience*, published online 15 February 2009; doi:10.1038/nn.2271.

39 J. Debiec, JE LeDoux, "Noradrenergic signaling in the amygdala contributes to the reconsolidation of fear memory: treatment implications for PTSD," *Ann N Y Sci* 1071(2004):521-4.

40 PCOB, "Beyond therapy," 9.

41 LaFee, "Blanks for the memories," 5.

42 PCOB, "Beyond Therapy," 6.

43 Ibid.

44 Gilbert Meilaender, "Why Remember?" *First Things* August/September(2003): 24.

45 David Derbyshire, "Pill to erase bad memories: Ethical furore over drugs 'that threaten human identity'," *Mail Online*, 16 Feb 2009 [last accessed 17 March 2009 at http://www.dailymail.co.uk/news/article-1145777/Pill-erase-bad-memories-Ethical-furore-over-drugs.htm].

46 Gaia, "Rewriting your past," 6.

47 The same perdurability of personal (essential) identity applies to persons who have been diagnosed with Alzheimer's or other kinds of dementia, especially in its final stages. Many caregivers, not realizing what they are saying, will declare something to the effect that "This is not my mother." Or, "my parent is not the same person he/she was before the devastation of the disease." It is very important to avoid language that implies, wittingly or unwittingly, that the cognitively impaired person has lost his or her personhood. The parent, spouse, or sibling with Alzheimer's is the same person after and during the disease, even in its end stages, that he or she was before. However, their personalities have changed, unfortunately, in ways that prevent them from consciously giving and receiving love. The test of caregivers, then, is to show genuine respect to those who, because they can no longer recall their life's experiences, are barred from normal social and familial relationships, and to continue to love them with every fiber of their being. And—this is important—they must do so not only because they intellectually understand the difference between essential and accidental identity, but also because they realize, despite the ravages of their loved ones' disease and the changes it has caused to their character, that dementia patients retain their personhood, and its correlative dignity, to the end.

48 Since the roots of Mr. Y's substantial nature are in his soul, not his body, he will not lose his personal identity even at death. Moreover, the separated human soul of Mr. Y, retaining an essential relation to his individual body, will be reunited to it at the final resurrection.

49 Leslie Stahl (CBS, 60 Minutes) interviewed the 52 year-old Louise O'Donnell-Jasmin who participated in a propranolol trial after having struggled with PTSD symptoms ever since she was raped at age 12. Her response to Stahl's question of whether the trial helped her was "Yes, the link, what held the emotions to the memories, it's like the umbilical cord has been cut. . . . And every day it gets better. . . . I have regained my identity. What was broken when I was 12 was fixed. They have given me back myself" [Daniel Shorn, "A Pill To Forget?" CBS News, 11 November 2006 last accessed on 17 June 2009 at http://www.cbsnews.com/stories/2006/11/22/60minutes/main2205629.shtml].

Sister Renée Mirkes, OSF, PhD, is director of the Center for NaProEthics, the ethics division of the Pope Paul VI Institute, Omaha, NE, USA.