assistance of caretakers in the co-construction of coherent narratives about the self, it may also result in damage to neural structures required to organize cohesive narratives and the story of the self that will persist into adult life.

12 The Impact of Trauma on the Brain

For each of us there is a point at which anxiety and fear cross the line into trauma. Trauma can cause severe disturbances in the integration of cognitive and emotional processing. The neurobiological reactions to these experiences appear to lie on a continuum of intensity. The earlier, more severe, and more prolonged the trauma, the more negative and far reaching the effects (De Bellis, Baum, Birnmaher, Keshavan, Eccard, Boring, et al., 1999; De Bellis, Keshavan, Clark, Casey, Giedd, Boring, et al., 1999). Unresolved and unintegrated trauma may result in a disorder known as *posttraumatic stress disorder* (PTSD). PTSD is comprised of a set of symptoms that reflect the physiological dysregulation and lack of integration of multiple networks of implicit and explicit memory (Siegel, 1999).

We have all heard the sayings "What doesn't kill you makes you stronger" and "Time heals all wounds." These bits of common wisdom conjure up pictures of difficult and traumatic experiences that, once overcome, result in greater levels of physical and emotional well-being. Although trials and tribulations can certainly build character, they can also create permanent biological, neurological, and

psychological compromise. Trauma produces a wide variety of homeostatic dysregulations that interfere with all realms of personal and interpersonal functioning (Perry, Pollard, Blakley, Baker, & Vigilante, 1995). Support for the negative impact of trauma comes from research that has shown that cumulative lifetime trauma increases the likelihood of developing PTSD (Yehuda et al., 1995). A history of previous assaults also increases the chances of developing PTSD following rape (Resnick, Yehuda, Pitman, & Foy, 1995). Likewise, childhood abuse increases the chances of developing PTSD after adult combat exposure (Bremner, Southwick, Johnson, Yehuda, & Charney, 1993). It has also been shown that severe stress reactions during combat make subsequent negative reactions to mild and moderate stress more likely (Solomon, 1990).

The effects of early and severe trauma are widespread, devastating, and difficult to treat. Because of the importance of a context of safety and bonding in the early construction of the brain, childhood trauma compromises core neural networks. It stands to reason that the most devastating types of trauma are those that occur at the hands of caretakers. Physical and sexual abuse by parents not only traumatizes children, but also deprives them of healing interactions. The wide range of effects involved in the adaptation to early unresolved trauma results in the phenomena of *complex posttraumatic stress disorder.*

Trauma is biochemically encoded in the brain in a variety of ways, including changes in the availability and effects of neurotransmitters and neuromodulators. Neuroanatomic encoding occurs through changes in structures like the hippocampus, and in the coordination and integration of neural network functioning. These changes in neurobiological mechanisms are reflected in the victim's physiological, psychological, and interpersonal experiences (see, e.g., Carroll, Rueger, Foy, & Donahoe, 1985). Deficits in psychological and interpersonal functioning then create additional stress which further compromises neurobiological structures. In this way, adaptation

to trauma, especially early in life, becomes a "state of mind, brain, and body" around which all subsequent experience organizes.

Expanding the Definition of Trauma

Trauma is not limited to surviving life-threatening experiences (as the standard diagnostic manual appears to suggest; American Psychiatric Association, 2000). For a young child, trauma may be experienced in the form of separation from parents, looking into the eyes of a depressed mother, or being in a household with a high level of marital tension (Cogill, Caplan, Alexandra, Robson, & Kumar, 1986). For an adolescent, chronic stress and trauma may come from the incessant teasing of peers or taking care of the needs of an alcoholic parent. For an adult, chronic loneliness, the loss of a pet, or a constant sense of shame or failure may have the same impact. Although it is impossible to understand the nature of prenatal and infant experience, we can assume that the infant is stressed well before the brain develops the capacity for consciousness. While we can't ask if it is stressed, we can assess whether an infant's body is experiencing biochemical changes indicative of a stress response (Gunnar, 1992, 1998).

There is a distinct possibility that stress is possible even before birth; an unborn child may become stressed as a result of the shared biological environment with its mother. Studies suggest that maternal stress is associated with their children's lower birthweight, irritability, hyperactivity, and learning disabilities (Zuckerman, Bauchner, Parker, & Cabral, 1990). Rats born to stressed mothers show more clinging to the mother, less locomotion, and decreased environmental exploration (Schneider, 1992). Prenatal stress may also result in permanent alterations in dopamine activity and cerebral lateralization, making offspring more susceptible to both anxiety and limiting their functioning into adulthood (Field et al., 1988). Children of Holocaust survivors have an increased prevalence of PTSD despite similar rates of exposure to traumatic events when

compared to children of non-Holocaust survivors. This suggests that they experienced a transferred vulnerability through interactions with their traumatized parents (Yehuda, 1999).

Maternal depression may actually serve as a highly stressful or traumatic experience for infants and children. Tiffany Field and her colleagues found that infants whose mothers were depressed during the infant's first year of life demonstrate biochemical, physiological, and behavioral dysregulation. These children show more neurophysiological and behavioral signs of stress and depression, including greater activation in their right frontal lobes, higher levels of norepinephrine, lower vagal tone, and higher heart rates and cortisol levels (Field, 1997; Field et al., 1988). These infants tend to develop behaviors and biological processes that mirror their depressed mothers. Just like their depressed mothers, such infants engage less in interactive behaviors (e.g., orienting toward and gazing at others) that are so vitally important for healthy development. Infants of depressed mothers behave this way even with other adults, making it more difficult for them to successfully interact with nondepressed others (Field et al., 1988).

In another study, it was found that depressed mothers were angry at their infants more of the time and were more likely to poke at them, disengage from them, and spend less time in "matched states" (Field, Healy, Goldstein, & Guthertz, 1990). These results suggest that infants are modeling on their mother's behavior, exhibiting resonance with their depressed states, and reacting to the negative behaviors directed toward them. Based on biological, physiological, and behavioral data, having a depressed mother is a stressful and potentially traumatic experience for an infant. Fortunately, it has been shown that interventions with depressed mothers and their infants have had positive results. For example, remission of maternal depression and teaching mothers to massage their infants on a regular basis resulted in improvement of the infants' symptoms (Field, 1997).

We would not consider these infants traumatized in the usual sense, and often think of infants as extremely resilient. On a biological level, however, the experience of having a depressed mother may result in the same changes as would occur in an adult exposed to a life-threatening situation. After all, infants' lives—and the building of their brains—depend on positive interactions with their parents. The loss of maternal presence, engagement, and vitality may all be experienced (at a biological level) as life threatening to an infant.

Neurotransmitter and Hormonal Changes

As we saw in chapter 11, states of acute stress result in predictable patterns of biochemical changes. There are increases in the levels of norepinephrine, dopamine, endogenous opioids, and glucocorticoids, and a decrease in serotonin. When stress is prolonged or chronic, changes occur in the baseline production, availability, and homeostatic regulation of these neurochemicals. These changes result in long-term behavioral and psychological alterations. Each of these substances serves a different purpose in the stress response and contributes in different ways to the long-term impact of PTSD.

As we have seen, increased levels of norepinephrine (NE) prepare us for fight-or-flight readiness and reinforce the biological encoding of traumatic memory. Higher levels of NE correlate with an increase in the experience of anxiety, arousal, and irritability. Heightened long-term activation of NE results in an increase in the level of tension and a heightened or unmodulated startle response (Butler et al., 1990; Ornitz & Pynoos, 1989). Besides being stronger, the startle response is also more resistant to habituation in response to subsequent milder and novel stressors (Nisenbaum, Zigmond, Sved, & Abercrombie, 1991; Petty, Chae, Kramer, Jordan, & Wilson, 1994; van der Kolk, 1994). Consistent startle experiences also enhance the victim's impression of the world as a dangerous and unsettling place. This is a good example of a feedback loop between physiological and psychological processes. An increased level of dopamine (activating the frontal cortex) correlates with hypervigilance, paranoia, and perceptual distortions when under stress. Symptoms of social withdrawal and the

avoidance of new and potentially dangerous stimuli (neophobia) are shaped by the anxiety of these biochemical changes.

Lower levels of serotonin have been found in traumatized humans and in animals after being subjected to inescapable shock (Anisman, 1978; Usdin, Kvetnansky, & Kopin, 1976). Chronically low levels of serotonin are correlated with higher levels of irritability, depression, aggression, arousal, and violence (Coccaro, Siever, Klar, & Maurer, 1989). Prisoners convicted of violent crimes have lower serotonin levels when compared to criminals convicted of nonviolent crimes.

Endogenous opioids, which relieve pain in fight-or-flight situations, can have a profound effect on reality testing and memory processing when released in response to a variety of emotional situations unrelated to danger. Higher opioid levels result not only in analgesia, but also in emotional blunting and difficulties with reality testing. Most likely, they are also involved with dissociative reactions and the experience of depersonalization and derealization, both of which provide an experience of distance from the traumatized body (Shilony & Grossman, 1993). Opioids are also related to self-harm in adults abused as children (van der Kolk, 1994), a topic we will soon address.

As we have seen, high levels of glucocorticoids have a catabolic effect on the nervous system and are thought to be responsible for decreased volume of the hippocampus (Wantanabe, Gould, & McEwen, 1992) and related memory deficits (Bremner, Scott, Delaney, Southwick, Mason, Johnson, et al., 1993; Nelson & Carver, 1998). Patients with PTSD related to childhood physical and sexual abuse have been shown to have hippocampi that are 12% smaller than those of comparison subjects (Bremner et al., 1997). Another study showed that right hippocampi were 8% smaller in patients with combat-related PTSD. Glucocorticoids sacrifice long-term conservation and homeostasis for short-term survival. Chronically high levels have negative effects on brain structures and the immune system. Traumatized individuals thus have higher rates of physical illness, which enhances their experience of being fragile and vulnerable individuals.

These biochemical and neuroanatomical changes are paralleled by such symptomatology as emotional dyscontrol, social withdrawal, and lower levels of adaptive functioning. Together, these and other negative effects of trauma result in a person whose functioning is compromised in some or all areas of life. The impact of trauma depends on a complex interaction of the physical and psychological stages of development during which it occurs, the length and degree of the trauma, and the presence of vulnerabilities or past traumas. The impact of a wide variety of traumatic experiences is woven into the structure of personality, often making it difficult to identify and treat.

The Symptoms of Posttraumatic Stress Disorder

Trauma results in a variety of psychological and physiological processes reflecting the reaction of the mind and body to threat. The pattern of reaction to trauma is predictable and connected to a variety of well-understood biological processes. Reaction to trauma tends to gradually diminish within a context of resolving the traumatic situation, gathering support from others, and having the ability to talk through the experience; these allow for regaining a sense of psychological control and biological homeostasis. An awareness of the importance of these healing processes has led to the development and testing of interventions made at different intervals following traumatic situations (Mitchell & Everly, 1993).

Talking through the traumatic experience in the context of supportive others creates the neurobiological conditions for the reestablishment of neural coherence. The co-construction of narratives drives the integration of cognition, affect, sensation, and behaviors. These are the very channels that can stay dissociated when early trauma, such as child sexual abuse, is never discussed or processed. The suffering of Holocaust survivors and combat veterans is often exacerbated by the psychological and political dynamics that encouraged them to remain

silent about their horrifying experiences. Co-constructed narratives in an emotionally supportive environment can provide the necessary matrix for the psychological and neurobiological integration required to avoid dissociative reactions.

When the trauma is severe, prolonged, or happens to a vulnerable individual, PTSD can develop. PTSD is caused by the loss of the regulation of the neurobiological processes dedicated to the appraisal and response to threat. There are three main symptom clusters in PTSD: hyperarousal, intrusion, and avoidance. These three groups of symptoms reflect the dysregulation of the central nervous system in response to unintegrated stress. Put another way, these symptoms demonstrate the loss of integration among neural networks controlling cognition, sensation, affect, and behavior.

Hyperarousal reflects a stress induced dysregulation of the amygdala and autonomic nervous system, resulting in an exaggerated startle reflex, agitation, anxiety, and irritability. That jumpy feeling we get when we drink too much caffeine gives us a taste for this experience. Chronic hyperarousal leads one to experience the world as a more dangerous and hostile place. Constant or uncontrollable agitation makes us less desirable as companions and thus, less able to benefit from the companionship of others.

Intrusions occur when traumatic experiences break into consciousness and are experienced as happening in the present. These may manifest in flashbacks resulting in a veteran hitting the ground in response to a car backfiring, or a rape victim having a panic attack while making love to her husband. These are activations of subcortical systems cued by stimuli reminiscent of the initial trauma. You may remember from the chapters on memory and fear that the amygdala both controls this activation and tends to generalize from the initial stimuli to a wide variety of cues. There is no sense of distance from the trauma in time or place, because the cortico–hippocampal networks have not been able to contextualize the somatic, sensory, and emotional memories within networks of autobiographical memory.

Avoidance is the attempt to defend against dangers by limiting contact with the world, withdrawing from others, and narrowing the range of thoughts and feelings. Avoidance can take the form of denial and repression, and, in more extreme instances, dissociation and amnesia. The power of avoidance was highlighted by the research of Williams (1994), who found that 38% of adult women who had suffered documented sexual abuse when they were children had no memory of the event. Compulsive activities can also aid in avoiding negative affect, as can alcohol and drug abuse, both so common in victims of trauma. Avoidance serves short-term anxiety reduction but perpetuates the lack of neural network integration. The passage of time does not cure trauma, nor does it diminish the intensity of flashbacks.

When experienced in combination, these symptoms result in a cycle of activation and numbing reflecting the body's memory of the trauma (van der Kolk, 1994). Instead of serving to mobilize the body to deal with new external threats, traumatic memories become the stimuli for continuing emotional responses. Someone suffering from PTSD is, in essence, in a continual loop of unconscious self-traumatization, coping, and exhaustion. When these symptoms are experienced on a chronic basis, they can devastate every aspect of the victim's life, from physical well-being to the quality of relationships to the victim's experience of the world.

Complex Posttraumatic Stress Disorder

Complex PTSD occurs in the context of prolonged inescapable stress and trauma. It is complex because of its extensive physiological effects and its impact on all areas of development and functioning (Herman, 1992). Enduring personality traits and coping strategies evolving from traumatic states tend to increase the individual's vulnerability to more trauma. This can manifest through engagement in abusive relationships, poor judgment, or a lack of adequate self-protection. Long-term PTSD has been shown to correlate with the presence of what are

called "neurological soft signs" pointing to subtle neurological impairments (Gurvits et al., 2000). These neurological signs could suggest a vulnerability to the development of PTSD, or they could reflect the impact of the long-term physiological dysregulation caused by PTSD (Green, 1981).

When confronted with threat under normal circumstances, the processes related to arousal and the fight-or-flight response become activated; the threat is dealt with and soon passes. Children are not well equipped to cope with threat in this way. Fighting and fleeing may actually decrease their chances for survival because their survival depends on dependency. When a child first cries for help but no help arrives, or when trauma is being inflicted by a caretaker, he or she may shift from hyperarousal to dissociation (Perry et al., 1995). Traumatized children who are agitated may be misdiagnosed as suffering from attention deficit disorder, while the numbing response in infants can be misinterpreted as a lack of sensitivity to pain. This may also be true for women who are often unable to outrun or outfight male attackers.

Until recently, surgery was performed on infants without anesthesia because their gradual lack of protest was mistakenly interpreted as insensitivity to pain as opposed to a traumatic reaction to it (Zeltzer, Anderson, & Schecter, 1990). Recent survey research suggests that less than 25% of physicians performing circumcision on newborns use any form of analgesia (Wellington & Rieder, 1993), despite physiological indications that neonates are experiencing stress and pain during and after the procedure (Hoyle et al., 1983). These practices appear to be a holdover of beliefs that newborns either don't experience or don't remember pain (Marshall, Stratton, Moore, & Boxerman, 1980). It makes sense that an appreciation for the possibility of PTSD reactions in neonates and young children has lagged behind other areas.

Research with rats has demonstrated that exposure to inescapable shock serves to sensitize their hippocampi to subsequent releases of norepinephrine under stress (Petty et al., 1994). This suggests that

after prolonged, inescapable shock, rats (and most likely humans) react more strongly to subsequent stress that is milder in nature. This neurobiological shift results in small stressors being experienced as more extreme. Petty and his colleagues suggest that this may help to explain the coping difficulties seen in victims of PTSD when confronted with mild to moderate stress (Petty et al., 1994). Think back to Sheldon, whom I described in chapter 5; he still suffered from anxiety 60 years after his childhood experiences during World War II.

Dissociation allows the traumatized individual to escape the trauma via a number of biological and psychological processes. Increased levels of endogenous opioids create a sense of well-being and a decrease in explicit processing of overwhelming traumatic situations. Psychological processes such as derealization and depersonalization allow the victim to either avoid the reality of his or her situation or watch it as an observer. These processes provide the experience of leaving the body, traveling to other worlds, or immersing oneself into other objects in the environment. Hyperarousal and dissociation in childhood establish an inner biopsychological environment primed to establish boundaries between different emotional states and experiences for a lifetime. If it is too painful to experience the world from inside one's body, self-identity can become organized outside the physical self.

Early traumatic experiences determine biochemical levels and neuroanatomical networking, thus impacting experience and adaptation throughout development. The tendency to dissociate and disconnect various tracks of processing creates a bias toward unintegrated information processing across conscious awareness, sensation, affect, and behavior. General dissociative defenses resulting in an aberrant organization of networks of memory, fear, and the social brain contribute to deficits of affect regulation, attachment, and executive functioning (van der Kolk et al., 1996). The malformation of these interdependent systems results in many disorders that spring from extreme early stress. Compulsive disorders related to eating or gambling, and somatization disorders in which emotions

are converted into physical symptoms, can all be understood in this way. PTSD, borderline personality disorder, and self-harm can all reflect complex adaptation to early trauma (Saxe et al., 1994; van der Kolk et al., 1996).

I Am Not Crazy!

Jesse was referred to me by her neurologist after months of extensive medical and neurodiagnostic testing. Her team of doctors could find no physical causes for the debilitating pain she experienced in her head and throughout her upper body. Alternative forms of treatment, such as chiropractic and acupuncture, were also tried without symptomatic relief. Jesse came to see me on the insistence of her husband, and she was not the least bit happy about it. Sitting opposite me with her arms crossed and her jaw set, she glared at me and said, "I am not crazy!"

Life had been going well for Jesse. She had a solid marriage and a happy and healthy four-year-old daughter. She found her work as an executive in a small computer firm interesting, and she liked her colleagues who told her that they appreciated her contributions to the business. Approximately a year earlier, she had started to develop pain in her head, hands, arms, and back, and began a fruitless search for a medical explanation. The pain became the center of her attention as her interest and ability in being an executive, friend, wife, and mother gradually diminished. By the time she came for therapy, she had been spending most of her days taking medication, sneaking away for naps, and withdrawing to her room whenever she could find an excuse. There was no longer any fun or relaxation in her life, and her husband had become seriously concerned.

We were slow in developing a therapeutic relationship, given her resistance and fear of being seen as "crazy." She reluctantly shared about her troubled childhood. Jesse felt she had obviously gotten over her traumatic past based on her later success at work and in her marriage. As she told me of her mother's physical abuse at the hands

of her father, she remained confident that there was no connection between her present physical pain and the emotional pain of her youth. Unfortunately, a common occurrence in her childhood was to be locked in her room by her father before he would begin to beat her mother. She would lie in bed feeling horrified by their screams, her mother's cries to Jesse to help her, and the long ominous silences that always followed.

Jesse remembered that she would pound on the door and yell to get her father's attention. As she grew older, however, she gave up her outward protests and instead lay in bed crying and clutching her head. Jesse said that she found herself driving her nails into her head and shoulders, drawing blood, and eventually scarring herself. She showed me some of the scars she still carried. In listening to these terrible reminiscences, I felt that her pain symptoms might well be implicit somatic memories of these experiences. The stresses in her present life, including the fact that her own daughter was reaching the age she had been when she first became aware of the beatings, could all serve as triggers for these memories. From a psychological perspective, her pain could be seen as a form of loyalty or connection to her mother through suffering.

I decided not to share these interpretations because of Jesse's resistance to the possible psychological origins of her pain. Instead, I continued to encourage her to talk about her childhood in as much detail as she could tolerate. She also told me of her mother's prolonged battle with cancer when Jesse was a teenager, and how she nursed her through the final months. In my work with Jesse, I avoided any talk of her physical pain and continued to refocus her on sharing childhood experiences with me.

In the process of repeatedly sharing stories from her childhood, her memories became increasing more detailed and her emotions more available and better matched to the situations she described. Jesse expressed her rage at her father for his violent behavior and she was able to realize that she was also angry at her mother for not leaving him when Jesse was young. As she went through these memories

and put them into the perspective of her current life, Jesse was gradually able to feel that she could connect with her mother through happiness instead of joining her in suffering.

We both came to notice that the intensity of her pain and the time she spent focusing on it gradually diminished. Toward the end of our last session, she thanked me for helping her and said that although she didn't understand how or why, her physical and emotional pain did seem to be connected. Our unspoken agreement was that I wasn't allowed to be the one to suggest this possibility. Jesse winked at me and said, "You are a tricky fellow."

Traumatic Memory

It has long been recognized that moderate amounts of stress enhance learning and memory by increasing vigilance and heightening attention, whereas high levels impair learning and memory (Yerkes & Dodson, 1908). Trauma is a state of high arousal that impairs integration across many domains of learning and memory. The neural networks in the limbic system and cortex involved in memory are influenced by several systems of ascending fibers that modulate arousal (Squire, 1987). In this way, stressful, threatening, and traumatic memories are emphasized based on instinctual and learned appraisal of their dangerousness. Each of these systems has its own neurotransmitters, which have different effects on the encoding and storage of memory. We have already discussed NE, serotonin, dopamine, glucocorticoids, and endogenous opioids, all of which impact memory processes in different ways.

When NE is administered to rats after an aversive event, low doses enhance retention whereas high doses impair retention (Introini-Collison & McGaugh, 1987); this supports Yerkes and Dodson's theory that moderate levels of arousal enhance memory whereas high levels impair memory. In a study by Cahill and his colleagues (Cahill, Prins, Weber, & McGaugh, 1994), subjects were read emotionally evocative and neutral stories and shown related slides.

Half of the subjects were given propranolol (a drug that decreases the effects of the NE) and the others were not. Results demonstrated that subjects who received propranolol had significantly impaired memory for the emotion-arousing stories but not for the neutral stories.

Activation of the amygdala (and the related physiological and biological changes) is at the heart of the modulation of emotional and traumatic memory (Cahill & McGaugh, 1998). The release of norepinephrine during the stress response serves to heighten the activation of the amygdala, thus reinforcing and intensifying memories for traumatic events (McGaugh, 1990). Individuals with PTSD have had their amygdaloid memory systems imprinted with trauma at such an extreme level that their memories are resistant to cortical integration (van der Kolk et al., 1996). Extreme trauma results in the inhibition of neural networks (cortico–hippocampal) that could contextualize and attenuate them. When we think of trauma overwhelming the defenses, we can also think in terms of an intense activation of subcortical networks serving to inhibit the participation of the hippocampus and cortex in the memory process.

Traumatic experience can disrupt the storage (encoding) of information and the integration of the various systems of attention and memory (Vasterling, Brailey, Constans, & Sutker, 1998; Yehuda et al., 1995; Zeitlin & McNally, 1991). *Memory encoding* for conscious explicit memory can be disrupted when the hippocampus is blocked or damaged by glucocorticoids or is inhibited by heightened amygdala functioning. This could lead to a lack of conscious memory for traumatic and highly emotional events (Adamec, 1991; Schacter, 1986; Squire & Zola-Morgan, 1991). *Memory integration* can be impaired by disruption of the cortico–hippocampal tracks dedicated to the integration of new memories into existing memory networks. Remember that these systems also provide contextualization in time and space, and integration of sensory, affective, and behavioral memory with conscious awareness.

Thus, although we may have very accurate physiological and emotional memories for a traumatic event, the factual information

may be quite inaccurate given the inhibition of cortico–hippocampal involvement during the trauma. Add to this the tendency of the left-hemisphere interpreter to confabulate a story in the absence of accurate information, and we may have what represents the underlying mechanisms of the malleability of memory.

Traumatic Flashbacks and Speechless Terror

Flashbacks are commonly reported by individuals who have experienced trauma. They are described as full-body experiences of aspects of the traumatic event, including physiological arousal and sensory stimulation. In a sense, the victim of a flashback is transported back in time to the traumatic experience. Flashbacks are so intense that they overwhelm the reality constraints of the contemporary situation and send the victim into an all-too-familiar and recurrent nightmare.

The power of traumatic flashbacks was driven home for me one day in a therapy session with a professional football player who stood nearly twice my size. When recalling his early abuse, he began to cry softly as he spoke of one particularly painful experience from childhood. He described in agonizing detail his small body growing limp after repeated blows from his father's fists. This explicit memory cued an implicit memory, a flashback; suddenly, he was standing over me and breathing heavily. Despite my alarm, I managed to sit quietly, eventually asking him what he was feeling. While looking into my eyes he asked me in a child's voice to please not hurt him anymore. His fear of me in contrast to our relative sizes was a stark demonstration of primitive memory systems overriding normal conscious processing.

Traumatic flashbacks are memories of a quite different nature than are those of nontraumatic events. To begin with, they are stored in more primitive circuits with less cortical and left-hemisphere involvement. Because of this, they are strongly somatic, sensory, and emotional, as well as inherently nonverbal (Krystal, Bremner, Southwick, & Charney, 1998). The lack of cortical–hippocampal involvement results in an absence of the localization of the memory in time, so when it is triggered it is experienced as occurring in the present (Siegel, 1995). Flashbacks are also repetitive and stereotypic, often seeming to proceed at the pace in which the events originally occurred. This suggests that although the cortex may condense and abbreviate memories in narrative and symbolic form, these subcortical networks may store memories in more concrete, stimulus-response chains of sensations, behaviors, and emotions.

In flashbacks, the amygdala-mediated fear networks (primarily in the right hemisphere) are activated. The amygdala's dense connectivity with the visual system most likely accounts for the presence of visual hallucinations during flashbacks. This is compared with the hallucinations in schizophrenia that involve the temporal lobes and are usually auditory in nature. Bereaved individuals often report seeing their loved ones sitting in their favorite chair or walking across the room in some familiar way. Those who have been attacked will sometimes think they see their attacker out of the corner of their eye. These emotionally charged visual hallucinations and illusions most likely reside in these amygdala-mediated systems.

Rauch and colleagues (1996) explored the neurobiology of intense fear using patients with PTSD. They took eight patients suffering from PTSD and exposed them to two audiotapes: One was emotionally neutral and the other was a script of a traumatic experience. While they were listening to these tapes, measures of patents' heart rate and regional cerebral blood flow (RCBF) were measured via PET scans. RCBF was greater during traumatic audiotapes in right-sided structures including the amygdala, orbitofrontal cortex, insular, anterior and medial temporal lobe, and the anterior cingulate cortex. These are the areas thought to be involved with intense emotion.

An extremely interesting and potentially important clinical finding was a decrease in RCBF in Broca's area (left inferior frontal and middle temporal cortex; Rauch et al., 1996). These findings suggest active inhibition of language centers during trauma. Based on

these results, *speechless terror*—often reported by victims of trauma—may have neurobiological correlates consistent with what we know about brain architecture and brain–behavior relationships. This inhibitory effect on Broca's area will impair the encoding of conscious memory for traumatic events at the time they occur. It will then naturally interfere with the development of narratives that serve to process the experience and lead to neural network integration and psychological healing. Activating Broca's area and left cortical networks of explicit episodic memory may be essential in psychotherapy with patients suffering from PTSD and other anxiety-based disorders.

Activating Broca's Area During a Flashback

Jan, seeing me for a one-time consultation, reported that she had suffered from severe physical and sexual abuse from early childhood into her late teens. She told me over the phone that she was having flashbacks of increasing frequency in recent years; it had gotten to the point where she was having three or four a day. Although her therapist had encouraged her to express them as much as possible, Jan felt like she was getting worse instead of better. Expressing her feelings only triggered more frequent and intense flashbacks. She reported becoming less and less functional, which made her decide that she needed a different approach to therapy.

Jan arrived at my office with a stack of diaries and *The Wall Street Journal* under her arm. It was hard to believe that this was the same person I had spoken to over the phone. My first thought was that dissociation is an amazing defense. Jan was a well-dressed woman in her mid-40s who was obviously bright and had a good deal of self-insight. The childhood experiences she recounted in my office were horrendous, and I marveled at her very survival. Her intelligence and sheer will to live were remarkable. It seemed obvious, however, that her repeated reexperiencing of these memories was not helping. The nature of these memories was not changing over time, nor were the

emotions evoked by her memories diminishing. In this case, they seemed to retraumatize her each time she experienced them.

She began by talking about her work, and then described the psychotherapy and other forms of treatment in which she had engaged. Approximately 10 minutes into the session, as she was discussing the family members who had abused her, she began to have one of the flashbacks she had described over the phone. Jan reported pain in various parts of her body and contorted as if what she was describing was happening to her at this very moment. After 20 seconds, she began to gag as a part of the memory of the sexual abuse she experienced decades earlier. She was reexperiencing these painful episodes not only as pictures in her mind, but as somatic memories throughout her body.

As she curled into the fetal position on the couch and gasped for breath, my mind raced trying to think of some way to help. Remembering the research done by Rauch and his colleagues, I thought that I should somehow try and activate Broca's area. I began to speak to Jan in a firm but gentle voice, loud enough to reach her in the midst of her traumatic reenactment but not so loud as to frighten her and add to her trauma. I wondered if it mattered which ear I spoke into, wondering which ear has a more direct connection to the left hemisphere language centers. I moved closer to her (careful not to get too close) and repeated over and over, "This is a memory, it isn't happening now. You are remembering something that happened to you many years ago. It was a terrible experience but it is over. It is a memory, it is not happening now."

As I repeated these and similar statements, I was concerned that Jan would be unable to breathe or that my presence might cause her more fear. The words of one of my supervisors flashed through my mind: "Whatever you do, don't panic." I was also encouraged by the fact that she had survived this many times. After 10 minutes (which seemed to me like 10 hours), she appeared to calm down and return to the present. Jan reported that she heard me speaking as if I were far away, but focused on my voice and words as best she could. It was as

if I were there in the past with her, calling to her from a safe future where she would be away from all these people who hurt her.

At the end of the session she thanked me and left; I didn't hear from her for a number of months. When she called one afternoon, she reported that since her visit with me, the nature of these flashbacks had changed. She said she had wanted to wait before she called me because she didn't expect that the change she experienced after our session would last. Given her dozen-plus years in a variety of unhelpful treatments, it was easy to be sympathetic to her negative expectations. Jan described that since our session, the flashbacks had changed in a number of ways. She began by saying that they were less physically intense, that the bodily sensations were not as strong as before. Along with this, they were also less frequent; on a few occasions she had even been able to stop one that was coming on by thinking of her version of my words during the session: "This is just a memory, you are safe now, no one can hurt you."

Perhaps most interesting was the fact that during these flashbacks she was now able to remember that she was not a child, that she was not to blame, and it was those who were hurting her who were bad. These thoughts were the sorts of things her other therapists had told her in the past, but only recently could she process them during her flashbacks. I told her that I felt these were signs that the experiences were beginning to be connected to her conscious adult self, and that now she was able to fight and care for herself even in the face of her past. I encouraged her to keep talking throughout the flashback experiences and bring with her as much assertiveness, anger, and power as she could muster. After a few minutes, we ended our conversation and I sat back thinking that neuroscience *could* actually be applied to psychotherapy.

It is impossible for me to know with any certainty whether what I had done with Jan during our one meeting had anything to do with the changes in her flashbacks. If it did, perhaps the active ingredient was the simultaneous activation of the left-hemisphere verbal areas along with the emotional centers of the right hemisphere and limbic

structures that stored the flashbacks. Being simultaneously aware of inner and outer worlds may support a higher level of cortical functioning, resulting in increased network integration. In other words, this process results in a memory configuration that is no longer "implicit only" but instead becomes integrated with the contextualizing properties of explicit systems of memory (Siegel, 1995).

The speechless terror, which has been recognized as part of posttraumatic reactions since ancient times, now has a neural correlate consistent with what is known about brain functions. Why does Broca's area become inhibited during trauma? Why would evolution select silence in times of crisis? Perhaps when one is threatened it is better to either run or fight or simply keep quiet and hope to stay undetected. In other words, evolution has taught the brain to "Shut up and do something!" when in danger. The freezing reaction of animals (being still and quiet when they sense a predator) allows them to be less visible (because a still and silent target is more difficult to spot). Spoken language is fundamentally "sound" that primitive fear circuitry has selected to silence. Perhaps those early prehumans who hung around for conversation and negotiation with predators didn't fare well enough to pass down as many genes as did those who either kept quiet, fought, or ran away.

The Addiction to Stress and Self-Harm

Another phenomenon with a possible biochemical mechanism is an addiction to stress experienced by some patients with PTSD. They report that they feel calm and competent in life-threatening situations but find it difficult to cope with normal day-to-day life. A large portion of the initial work with these patients is designed to help them both decrease the creation of stress and, paradoxically, tolerate the anxiety related to the absence of stress. They need to learn how to function in a nontraumatic state. This can usually be accomplished through some combination of stress-reduction techniques, medication, and psychotherapy.

These phenomena point to the possibility that extreme and prolonged stress—significant enough to result in a chronic PTSD reaction—may motivate the creation of new trauma. The new trauma would, in turn, stimulate the production of endogenous opioids that would lead to an increased sense of well-being. A so-called "normal" life leaves traumatized persons a blank screen onto which their dysregulated psyches can project fearful experiences, leaving patients such as these in a state of constant vigilance, arousal, and fear (Fish-Murry, Koby, & van der Kolk, 1987). Trauma and stress as coping strategies provide them with a shift from being anxious and wary to being calm and competent. Because these individuals are so physically worn down by this lifestyle, they often present with depression, exhaustion, and a variety of medical conditions. It is as if they have a drug addiction, except that it is completely unconscious and they are their own pharmacy.

At a biochemical level, endogenous opioids (e.g., the endorphins discussed in the neurochemistry of bonding and trauma) also appear to be involved in severe cases of self-harm and suicide (van der Kolk, 1988). This same chemical system mediates the distress calls of baby primates and mothers' response to these calls. Infants become distressed when their mothers are absent, and are soothed and calmed upon their mother's return. The return of the mother is correlated with the release of endorphins creating the sense of well-being. Endorphins are also released after injury to provide analgesia for pain, allowing us to continue fighting or escaping (Pitman et al., 1990). This system, originally used to cope with pain, was adapted by later-evolving networks of attachment and bonding.

The addiction to stress has a related but more severe variant: self-mutilation and other forms of self-harm. Adults who engage in repeated self-harm almost always describe childhoods that included abuse, neglect, or a deep sense of shame. This correlation has led many theorists to explore the psychodynamic significance of self-harm as a continued involvement with destructive parents. Suicide has been described as the final act of compliance with the parents' unconscious wish for the death of the child (Green, 1978). The association between self-harm and disorders of attachment has been noted and primarily explained through psychological models.

Self-injurious behaviors in humans are often responses to real or imagined abandonment and loss. Research has demonstrated that the frequency of self-harm decreases when people who engage in this behavior are given a drug to block the effects of these endogenous opioids (Pitman et al., 1990; van der Kolk, 1988). Abstracting from the animal model, this would suggest that the state of distress activated by the experience of abandonment is reversed via the release of endorphins caused by the injury. The analgesic effects of these morphine-like substances may account for the reports of reduced anxiety. People who engage in self-harm report a sense of calm and relief after cutting, burning, or hurting themselves. These self-injurious behaviors may be a form of implicit memory that is reinforced, in part, by the endogenous opioid system.

Repeated suicide attempts are often reinforced by the rapid attention of health care professionals, family, and friends. When woven into the personality as a means of affect regulation, this attention-getting behavior results in a kind of characterological suicidality (Schwartz, 1979). This behavior parallels the distress calls of primates whose endorphin levels drop in the absence of the mother. The reappearance of the mother results in a raising of these endorphin levels and the infant discontinues its cry. Characterological suicidality can serve a similar biochemical regulatory purpose if this system was inadequately formed during childhood. Although there are many sound psychological explanations for the relationship of childhood abuse with self-harm and suicidality in adulthood, the process may have a biochemical mechanism that could benefit from pharmacological interventions designed to block the impact of endogenous endorphins.

The Brain and Borderline Personality Disorder

According to Freud, participation in analysis requires sufficient ego strength to withstand the stress of therapy while simultaneously maintaining contact with reality. Based on this assumption, Freud did

his best to make sure that his prospective clients were not psychotic. Psychotic individuals are characterized by severe distortions of reality, disorders in their thinking processes, and decompensation under stress. They are also unable to differentiate their transference and other projective processes from reality. These are all reflections of low ego strength. Despite Freud's best efforts to filter out these people, every so often he got a surprise! People who appeared to be average neurotics seemed to become psychotic in the context of the therapeutic relationship. Freud came to refer to these people as having psychic structures on the *borderline* between neurotic and psychotic.

Over the years, the conception of a borderline psychic structure evolved into what is now called a *borderline personality disorder* (BPD). As we have already seen, BPD may represent one variant of complex PTSD. The strongest evidence for this concept is the frequent occurrence of early abuse, trauma, and the use of dissociative symptoms in these individuals. Patients who carry this diagnosis are characterized by:

1. Hypersensitivity to real or imagined abandonment.
2. Disturbances of self-identity.
3. Intense and unstable relationships.
4. Alternating idealization and devaluation of themselves and others (black-and-white thinking).
5. Compulsive, risky, and sometimes self-damaging behaviors.

Although there are a number of theories concerning its cause, many feel that the etiology of BPD stems from problems in early life related to bonding, attachment, and a sense of safety. Research also suggests that affective disorders in these patients and their parents occurs above chance levels. Overall, both their reported history and their symptoms suggest that early attachment was experienced as highly traumatic, emotionally dysregulating, and possibly life threatening.

In my work with these patients, I have always felt that they may provide us with a window to the intense and chaotic experience of infancy. As we have seen (and this is where our neuroscientific knowledge comes in handy), the amygdala is highly functional at birth. Remember that the amygdala is at the center of neural networks involving both fear and attachment. The hippocampal and cortical networks that eventually organize and inhibit the amygdala grow gradually through childhood. Because of this developmental timetable and the prolonged dependence on others for survival, relationships must sometimes be as overwhelmingly frightening to infants as we see they are to patients with BPD.

The symptoms that emerge in this disorder cause patients to create problematic and chaotic relationships that can lead them through a lifetime of serial abandonments. It is even common for therapists to abandon these patients because of their intense criticism and hostility. I find that remembering that such patients are essentially frightened children helps me to maintain a therapeutic posture. Their primitive fear, rage, and shame are a form of implicit posttraumatic memory from a very early stage of development that are activated by real or imagined criticism or abandonment. When these memory networks become activated in treatment, they are so primitive and powerful that the patient is unable to maintain contact with reality. We also see the same phenomena in PTSD flashbacks, most likely stored in the same implicit memory systems. This confused Freud, because he believed that everyone was either neurotic or psychotic. Here was a horse of a different color: primitive and highly complex PTSD.

Examining BPD in light of the neuroscience we have reviewed in previous chapters, here are a few of the neurobiological processes that may be involved in how these symptoms become encoded within neural networks:

1. Amygdaloid memory systems are traumatically primed during early attachment experiences to react to any possible indication of abandonment by triggering sympathetic fight-or-flight

reaction and raising baseline levels of norepinephrine and stress hormones.

2. Orbitofrontal systems are inadequately developed during attachment to engage in healthy self-soothing and the inhibition of fear activation by circuits of the amygdala.
3. Orbitofrontal systems develop separate tracks of positive and negative experiences that are never integrated.
4. Orbitofrontal dissociation may result in disconnection between right- and left-hemisphere and top-down processing, partly accounting for rapid and radical shifts between positive and negative appraisals of relationships.
5. The networks of the social brain are unable to internalize images from early interactions with caretakers to provide self-soothing and affect regulation.
6. Rapid fluctuations between sympathetic and parasympathetic states result in baseline irritability and sympathetic survival responses to real or imagined abandonment.
7. Chronic high levels of stress hormones compromise hippocampal functioning, decreasing the brain's ability to control amygdala functioning and exacerbating emotional dyscontrol.
8. Amygdaloid dyscontrol heightens the impact of early memory on adult functioning, increasing the contemporary impact of early bonding failures.
9. Hippocampal compromise decreases reality testing and memory functioning, contributing to the inability to maintain positive or soothing memories during states of high arousal.
10. Early bonding failures lead to lower levels of serotonin, resulting in greater risk of depression, irritability, and decreased positive reinforcement from interpersonal interactions.
11. Self-harm during dysregulated states results in endorphin release and a sense of calm, putting these individuals at risk for repeated self-abusive behavior.

These are just some of the factors that may be involved in the neurobiology of BPD. Because this diagnosis has so far been outside the purview of neurology, little brain research has been done with BPD patients. Neuropsychological findings with these patients, however, does suggests dysfunction in the frontal and temporal lobes (Paris, Zelkowitz, Guzder, Joseph, & Feldman, 1999; Swirsky-Sacchetti et al., 1993). Executive and memory functions within these brain networks do not provide adequate organization for these patients. We have learned that these functions are built and sculpted in the context of early relationships; it makes sense that they are impaired in BPD patients. The central concept in the treatment of these individuals is structure and limit setting, combined with flexibility and patience (just as it is with raising children). The therapist must provide an external scaffolding within which the client can rebuild these brain networks of memory, self-organization, and affect regulation. On another level, the therapist serves as an external neural circuit to aid in the integration of networks left unintegrated during development.

Neural Network Integration

Unresolved trauma results in information-processing deficits that disrupt integrated neural processing. In fact, the experience of dissociative symptoms immediately after a trauma is predictive of the later development of PTSD (Koopman et al., 1994; McFarlane & Yehuda, 1996). Conscious awareness is split from emotional, and physiological processing. A lack of integration of right- and left-hemisphere functions subsequent to stress may also disrupt processes of interpersonal bonding and bodily regulation (Henry, Satz, & Saslow, 1984). Children victimized by psychological, physical, and sexual abuse have been shown to have a significantly greater probability of demonstrating brainwave abnormalities in the left frontal and temporal regions

(Ito et al., 1993). Brainwave dyscoherence may put individuals at higher risk for the development of all forms of psychiatric disorders (Teicher et al., 1997).

The biochemical changes that occur secondary to trauma enhance primitive (subcortical) stimulus–response pairing of conditioned responses related to sensation, emotion, and behavior. These same changes undermine cortical systems dedicated to the integration of learning across systems of memory into a coherent and conscious narrative (Siegel, 1996). As we understand more about the neurobiological processes underlying PTSD, we will better learn how to treat and possibly prevent this debilitating yet curable mental illness.

Therapies of all kinds, especially those within the cognitive schools, have proven successful in the reintegration of neural processing subsequent to trauma. Systematic desensitization, exposure, and response prevention can all enhance these integrative processes. Recognition of the risks to neural network integration posed by overwhelming stress and trauma has resulted in the development of some newer treatments such as critical incident stress debriefing (CISD; Mitchell & Everly, 1993). Using our present model, CISD may help to prevent PTSD by enhancing the interconnections among neural networks—at risk for dissociation—soon after trauma.

CISD moves through phases of processing cognitive and emotional aspects of experience, using both psychoeducation to enhance an understanding of the body's reaction to stress and group process to provide a context for reality testing and normalization of posttraumatic reactions. The sequential activation of networks of cognition, emotion, sensation, and behavior are encouraged in the context of support and conceptual understanding of the entire process. This is a new treatment modality for PTSD, and conclusive research concerning the efficacy of its timing and various components remains to be done.

Summary

The brain's reaction to trauma provides us with a window to the general processes of learning and plasticity. The brain does indeed change in reaction to environmental events. This helps us to understand why the safe emergency of psychotherapy is able to alter neural networks organized in a manner not conducive to mental health. From the first moments of life, stress shapes our brains in ways that lead us to remember experiences most important for survival. Most of our learning experiences are not traumatic but rather subtle, nondramatic, and unconscious. The interactions between parent and child, the politics of the schoolyard, and experiences of small victories and losses all contribute to shaping who we will become.

In the final chapter, we will review some of the basic principles that have emerged from our exploration of neuroscience and psychotherapy, especially as they apply to the future of both fields.